KURU IN CONTEXTS

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A thesis
submitted to the University of Western Sydney
in fulfilment of the requirements
for the degree of
Doctor of Philosophy

March 2001
PLEASE NOTE

The greatest amount of care has been taken while scanning this thesis,

and the best possible result has been obtained.
Acknowledgments

This study has been greatly assisted by help from staff at a number of libraries. I would especially like to thank Di Roberts and Sue Venables at the University of Western Sydney, Hawkesbury library, Elizabeth Agostino at the University of Melbourne archives and Gavan McCarthy, Chief Archivist at the Australian Science Archives Project, now known as AUSTEHC, for their patience, skill, interest and support. Thanks also to staff at the Adolf Basser Library, Canberra, the University of Melbourne, the University of Sydney, the Australian National University, the John Curtin Medical Library and the Australian National Film and Sound Archive.

I am also grateful to faculty and staff at the Division of Pacific and Asian History at the Australian National University, who made me welcome in May/June 1999 while on a National Visiting Scholarship. Professors Hank Nelson, Donald Denoon, Bryant Allen, Michael Bourke and Gavin McCormack, each helped to enrich my understanding of Pacific and world history and were later willing to help me with questions about the history of New Guinea. Special thanks to Hank Nelson who provided access to some of the administrative papers relating to kuru research.

An Australian Postgraduate Award for three and a half years of the study provided by the UWS Postgraduate Committee is also greatly appreciated, for it allowed me to concentrate on the project without having to divide my time between work and study.

Many other people have taken time to talk with me. I particularly thank Jennifer Cooke who has written about kuru and William Tomasetti who has worked with the administration in New Guinea. Colleagues at UWS, particularly Professors Mike Clear, Ruth Barcan and John McDonald have also given moral support and helpful advice. Special thanks to Dr Catharina Landström, who read many drafts and provided valuable criticism as well as lending moral support.

The most constant source of encouragement has come though, from my principal supervisor, Associate Professor Jane Goodall, whose humanity has helped to sustain me, and whose understanding of the difficulties that can be encountered when working with knowledge across a number of disciplines has made the task so much easier.

Finally, I would like to thank my friend Carolyn Britten for the many hours spent in conversation and for being a great sounding board. And to Will and the rest of my family who have endured my preoccupation for so long yet continued to love and support me.

While the work has been enriched by these many sources, obviously, the interpretation is my own and for it I take responsibility.
Statement of Authentication

The work presented in this thesis is, to the best of my knowledge and belief, original except as acknowledged in the text. I hereby declare that I have not submitted this material, either whole or in part, for a degree at this or any other institution.

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Synopsis

That cannibalism was the principal means of transmission of the disease called “Kuru” is a widely accepted belief in scientific and public discourse at the end of the twentieth century. This thesis argues that other explanations might have been excluded from consideration, in particular, iatrogenic transmission. Through analysis of archival and published material, the work examines the circumstantial evidence in support of this proposition, by looking at the research conducted on the disease within its historical and institutional context.

The work begins with an examination of the relationship between a number of diseases including, X disease, poliomyelitis, louping ill, scrapie and kuru through the first half of the twentieth century. It then examines the relationship between kuru, iatrogenic Creutzfeldt-Jakob disease and Bovine Spongiform encephalopathy during the second half of the century, with particular reference to the safety of medicines.

Major themes of the work revolve around the boundary between research on animal and human disease, the complexities of research in this area, and the different messages that exist simultaneously in three domains: scientific research and publications, government and institutional archives, and the public domain.

The thesis argues that the circumstantial evidence presented needs to be considered seriously and that further research in the area is required before we can come to a reliable understanding of the factors involved in the transmission of kuru.
Introduction

"Kuru is a chronic, progressive, fatal, viral infection of the central nervous system. ... [that] exists only in the mountainous interior of eastern New Guinea in an area of about 1,000 square miles. Only the Fore linguistic group and neighboring tribes with Fore ancestry (a total of about 35,000 people in 160 villages) are affected. Kuru ("trembling" in Fore language) was thought originally to be a genetically determined disease, with clinical expression in children of both sexes and in adult women. It is now known to be a viral infection, which is transmitted by cannibalistic rites. Since the abolition of mourning rites, during which women and children ate the brains of dead kinsmen, the incidence of kuru has declined. The disease is now rare in pre-adolescents and will soon become a footnote in medical history (Heffner & Strano 1976:49).

The above quotation comes from the text Pathology of Tropical and Extraordinary Diseases, published by the Armed Forces Institute of Pathology, Washington, D.C. and has been chosen to introduce the subject of kuru because it serves to highlight a number of points. Not only is it a fine description of the kuru story, along with its emphasis on cannibalism, but it also depicts the remoteness of place and those affected by the disease. The title of the book is an indication of how kuru has been classified and the publishing institution highlights but one of the directions from where interest came in 1957 when the investigation of the disease took place. It is worth reviewing what has been said about this disease before conceding that a footnote is to be its final resting-place.

About the study

This enquiry differs from many other studies on kuru in that most of the narratives written about the disease highlight anthropological notions of ‘primitive’ people living in a ‘stone-age’ culture. In these stories, cannibalism takes centre stage in the discourse for it is a widely held belief that this was how the disease was transmitted as evidenced by the above quotation. Another quotation from the scientific literature will
serve to reinforce the point and simultaneously provide a little more detail about the disease, its investigators, and its impact:

Kuru, a fatal disease affecting the stone age tribes of the Fore people indigenous to the New Guinea highlands, was first described in 1957 by Carleton Gajdusek and Vincent Zigas. Initially, victims lost coordination of the limbs and trunk, eventually, motor function became severely impaired. Death typically followed within a year after onset of clinical disease. In some tribes, 50% of deaths beyond infancy were caused by Kuru, a progressive degenerative disease of the central nervous system transmitted by ritualistic cannibalism (Bessen 1996:12).

One of the puzzling questions about kuru is, why is it that this disease has not been assessed from the point of view of an iatrogenic (medically acquired) disorder when diseases similar to kuru have been transmitted by this means? A few examples are the transmission of scrapie to sheep and of Creutzfeldt-Jakob disease to humans. While cannibalism might be responsible for some of the cases of kuru that occurred this does not mean that it caused every case. In medicine there are often multi causal factors involved in any given phenomenon. Yet, in the case of kuru it seems that it has been sufficient to rely solely on cannibalism as vector. One of the aims of this study is to highlight the iatrogenic means of transmission of these other diseases, known variously as prion diseases, transmissible spongiform encephalopathies (TSEs) and infectious amyloidoses1. A related aim is to provide circumstantial evidence to assess the possible relevance of iatrogenesis as a factor in the transmission of kuru. To show this requires a consideration of relevant historical and institutional factors prevalent around the time of the investigation. When these factors are brought into the foreground it is possible to provide a more nuanced account of kuru.

Of particular relevance is the study of a group of diseases known as Group B encephalitides, which includes yellow fever, Japanese B encephalitis and Murray Valley encephalitis (MVE). The relevance of this group for kuru lies in the fact that the kuru investigation in 1957 immediately followed research conducted throughout the nineteen fifties on Murray Valley encephalitis and involved the same scientists and institutions. MVE can be traced back to a mysterious disease that occurred in

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1 That so many different fields of researchers are engaged in research on prion diseases highlights complexity in this area and indicates that the problem is being tackled from many angles.
1917 which was later named X disease. X disease occurred again in the nineteen thirties, and again in the nineteen fifties when it was renamed MVE. Because of the close temporal relationship between MVE and kuru the thesis is concerned with where individuals and institutions involved in the study of the Group B diseases intersect over time with the study of what for the sake of simplicity will be referred to as prion diseases. Included within this latter group are scrapie, kuru, CJD, BSE and variant CJD, all of which have been studied over successive decades of the twentieth century with kuru lying in the middle of the time frame. The purpose then, is to juxtapose kuru and associated prion diseases with Group B encephalitides and to position the investigation of kuru and the study of all these diseases within an institutional and historical frame rather than relying on anthropological notions to support the narrative. This will broaden the debate about kuru, which is usually confined to discussion of events post 1957.

Given that the major kuru investigation occurred in the late nineteen fifties, it is relevant to tell this story with an assessment of post-war history and research as a backdrop. Against this backdrop, the thesis examines the investigation of the outbreak of the disease in one isolated region of the Eastern Highlands of New Guinea. It is therefore as much, if not more, about the times when kuru occurred and the institutions involved than it is about the medical or anthropological aspects of the disease, although, these last two elements, which have already received much attention, will be discussed when necessary.

Kuru is a topic that has fired the imagination of people in many fields of endeavour. By 1964 about 140 scientific articles had been published (Bibliography in Gajdusek, Gibbs & Alpers 1965: 403-409). Articles have been written in fields as disparate as neurology (Hornabrook in Subirana & Espadaler 1974), eugenics (Gajdusek 1962), medical science (Zigas 1975), psychiatry (Jervis 1968) and anthropology (Berndt 1958, Lindenbaum 1976, Arens 1979, 1990, 1997). Following a survey of much, though not all, of the published literature, at least four things are apparent.

Firstly, the cannibalism hypothesis with respect to kuru has been challenged within the field of anthropology by William Arens in his book *The Man Eating Myth* (1979).
While Lindenbaum or Berndt describe second hand accounts of cannibalism in great detail, Arens is much more critical of taking these accounts at face value. I will go into this later, but for now it is enough to point out that the cannibalism hypothesis has been questioned. Another feature of the published literature is the attention given to the pathology of the disease. This study differs in that it does not focus on the pathology of kuru alone, but instead draws attention to the similarities between the pathology of kuru and the pathology of X disease and MVE. This is not to argue that kuru and MVE are the same disease, but rather to highlight in which ways they are similar. The third thing about the literature is the confusion present. This has been explained in part because each piece of published work is invariably written within a disciplinary or professional boundary (Zigas 1975). This thesis attempts to obtain a broader perspective, and this is something that can not be achieved by staying within the designated boundaries, which are, after all, arbitrarily constructed and restrictive. This is not to say that a total view is ever possible but when we are respectful of, but at the same time cross, the borders of disciplinary knowledge, vision can be enhanced.

The fourth element of the literature, which is conspicuous by its absence, relates to the institutional context. One noticeable exception to this a-contextual approach is a paper presented by Michael Alpers at a meeting held in Washington in 1964 (in Gajdusek, Gibbs & Alpers 1965: 65), which provides a list of some of the events that could have been associated with the outbreak. The lack of attention to the context is an omission all the more surprising considering that kuru was a matter of international significance during the late nineteen fifties. This situation is little different from the way that iatrogenic Creutzfeldt-Jakob disease became internationally significant during the nineteen eighties, and BSE and new variant CJD became during the nineteen nineties. Because of the later outbreaks of similar diseases it is relevant and timely to examine the kuru story once again with the benefit of hindsight.

Using a retrospective approach extending back to the beginning of the twentieth century, I aim to show that concern was expressed in the community with respect to acute disease like X disease long before a similar concern was expressed with respect to human prion diseases. A tendency to concern the public, scientist, doctor and politician alike was a common feature of both disease groups. To observe this, it is necessary to look outside of the current field of prion research and to look back over time. This is not to deny that these two groups of diseases can be distinguished by
their incubation periods; the prion group generally have a long incubation period
while this period is much shorter for Group B diseases, although the boundary
between short and long is a moot point. But apart from this demarcation problem
between the periods, the intention here is to highlight similarities rather than
differences between the two groups.

The work draws on a wide range of published literature written about kuru for specific
scientific disciplines as well as on archival material relative to history, international
relations and kuru more specifically. Three bodies of work of particular relevance are
Series 10 of the Sir MacFarlane Burnet papers\(^2\), Sir John Gunther’s ‘Kuru File’\(^3\), and
the journals and letters on kuru published by the American National Institute of
Health\(^4\). These three sets of material provide different points of reference pertaining
respectively to the Australian medical history of both of the groups of diseases in
question, the administrative history of kuru in New Guinea, and an American
institutional perspective. All three sets of material are excellent source documents that
tell about the effect that kuru had on both Australian and American administrations
and the efforts made to control the disease.

Burnet, at the time of the investigation, was the Director of the Walter and Eliza Hall
Institute in Melbourne. Series 10 of the Burnet Papers entitled ‘X disease, kuru and
Gajdusek files’ consist of 38 files, a list of which is attached as Appendix 1. The first
two folios relate to work conducted in 1933 and 1956, respectively, when research
was carried out on X disease and Murray Valley encephalitis. The significance of this
pre-kuru period is that preliminary negotiations for the Kuru Project were held in
1956 yet these seem to be glossed over in many of the published works. Taking
account of the Burnet Papers helps to situate the investigation of kuru within its

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\(^2\) The Burnet Papers are held at the Archives of the University of Melbourne, and were collected by
Gavan McCarthy as part of the Australian Science Archives Project (now AUSTEHC) beginning in
1985. Hereafter the papers will be referred to as BP. I am grateful to Gavin McCarthy for the time he
made available to answer questions about the collection.

\(^3\) The Kuru File is a collection of administrative documents collected by John Gunther, which is held at
the Australian National University by Professor Hank Nelson. I am grateful to Professor Nelson for
making the papers available.

\(^4\) This last set includes Correspondence on the Discovery and Original Investigations on Kuru: Smadel
- Gajdusek Correspondence 1955-1958 (Gajdusek 1975), Journal 1955-1957 Australia and New
Guinea, Virology to autoimmunity, ethnopediatrics to kuru, enchantment by melaniscans, politics to
science (Gajdusek 1996), and Kuru Early Letters and Field-Notes from the Collection of D. Carleton
Gajdusek, edited by Farquhar & Gajdusek in 1981.
historical context. Gunther's papers portray well the issues faced by the New Guinea administration throughout the period preceding, during and after the investigation. Gunther became the newly appointed Assistant Administrator of the Territory of New Guinea in February/March 1957 just as the investigation of kuru was beginning. Whereas Burnet's and Gunther's Papers focus our attention on the Australian perspective, the publications by the National Institute of Health revolve more around the American institutions that supported Dr D. C. Gajdusek in his investigation of the disease.

In addition, tales of kuru that have been woven into the novel and told in the popular press are sometimes referred to in this thesis. Some might argue that such accounts are not worth attention in terms of their reliability. It is usually the case that scientific and administrative material can be assumed to be a more reliable source. The differences between rationality and imagination and between science and art are far more blurred and probably always have been than many would like to believe. I argue that a critical stance needs to be taken when reading all accounts of kuru, including those deemed 'scientific', as the earlier quotations show.

Of particular interest is the reference made to "stone age" tribes in Bessen's article and the certainty with which he and Heffner and Strano speak about the transmission route of the disease. Both speak with the same authoritative voice, yet it is worrying from a scientific point of view that the vector relationship is not qualified. As Arens has pointed out, it is not possible scientifically, or otherwise, to prove that the route of transmission was cannibalism. Through a detailed examination of the texts written about kuru, Arens has been able to show that there is uncertainty about when the practice of cannibalism ceased in the kuru region of New Guinea (1990:153). Independently of Arens's work, I too, found many inconsistencies in the literature with respect to cannibalism as well as other aspects of the study. While it would be acceptable then, to write about the vector relationship as being associative, it is not acceptable to write about it as though it were proved beyond doubt, yet this is what occurs in scientific reporting. A critical attitude does therefore seem warranted as questions about assessing other plausible relationships between the disease, its cause and transmission do not seem to have been canvassed in any great detail.
Before beginning the current study, I explored the ethics and institutional histories of the Human Pituitary Hormone Program (HPHP) as part of an Honours program within a School of Humanities. The HPHP came to a halt in Australia, the United States and England in 1985 following the realisation that the hormones could result in death for some recipients from iatrogenic CJD. The pituitary program - perhaps coincidentally, perhaps not – began in 1958 (Allars 1994), immediately following the first year of the kuru investigation. This previous study has been helpful in understanding some of the complexity of prion research.

At the beginning of the earlier work in 1995 I was not even aware that a disease called kuru existed, although a background in health science enabled me to locate and explore the medical literature once I embarked on the study of kuru in 1997. It did not take long to realise that kuru was similar to scrapie in sheep, which, I later realised, had been problematic scientifically since the beginning of the twentieth century. Nor had I ever heard about a group of biological agents known as slow, latent, or temperate viruses that could be transmitted by various routes. It did not take long to learn that the degree of risk associated with different means of transmission had been identified. The most effective means is through an intracerebral route. Then, the degree of risk becomes less with peripheral injections administered through an intraperitoneal, intramuscular and subcutaneous route, with the oral route being the least risky. Thus, the means of transmission is an important element in the story and gains a prominent position in the current enquiry.

As part of the Honours thesis, I also began reading literature on colonial exploitation, imperialism and its connections with the development of so-called “primitive” nations, which included works exploring colonialist medicine and the use of terms like ‘tropical medicine’ and ‘exotic’ disease in medical history. At this time, I read William Arens’s book *The Man Eating Myth* (1979), which talks about how the notion of cannibalism is put to work as a marker of denigration in some anthropological texts, and how this notion has been put to work in relation to kuru in particular. This left me wondering whether a Eurocentric discourse about the “other” and their so-called exotic diseases, had clouded the issues regarding kuru. Had the discourse distanced a western readership to such an extent that the disease seemed irrelevant to western medicine? Had effective research into such life threatening
diseases as CJD, BSE and nvCJD been hindered because the kuru story was enmeshed in tales of “primitive cannibals” and “exotic others”? And if so, how had this occurred? Was it the case that these terms had become so firmly intertwined in the kuru story that a serious attempt to explore the issues relating to prion diseases from another perspective was overshadowed? I wondered whether this had inhibited the dissemination of information about prion diseases. And if this was the case I wondered whether anything could have been learned had information been more freely exchanged? All of these questions led to the current study.

Later, I realised how much has been written about the notion of cannibalism, its use as a marker of the savage, of the uncivilised, and its potential effects. One of which as Arens (1990) suggests is that cannibalism seems to have acted to prevent further conversation about kuru. David Richards in *Masks of Difference* (1994) and Gustav Jahoda in *Images of Savages* (1999), while not specifically speaking about kuru, also remark on how the notion of cannibalism features in the construction of the primitive other. While Arens suggests that the notion can serve as a rhetorical device to denigrate others and Richards shows how cannibalism was assumed to be part and parcel of the life style of the “primitive savage”5, Jahoda has shown how

Images of the Other, that strange, exotic, incomprehensible creature, feared, abhorred, and yet in some ways also envied, have run as a constant thread through the European past (Jahoda 1999:1).

The kuru story in toto is a fine example of what Jahoda means. Images of the exotic native - of cannibals, and of diseased, feared, others - run rampant not only in scientific and popular accounts but also in press reports of the disease. Constant trivialisation of kuru as ‘laughing death’ in these media in the years following the initial investigation in 1957 has helped to reinforce the idea in the public’s mind, at

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5 Richards points out that when the anthropologist James Frazer collected data on ‘exotic’ people at the end of the nineteenth century, he did so by devising a series of questions based on his assumptions of what constitutes such people. The *Questions on the Customs, Beliefs and Languages of Savages* published in 1910 related mainly to bizarre sexual practices, cannibalism, and religious ritual. Richards also shows how a painting by Jacques Le Moyne de Morgues, ‘They Sacrifice First-Born Sons to the Chief in Solemn Rituals’ (1591), was significantly altered when it appeared in Frazer’s work *The Golden Bough* in 1890. Richards suggests that the image of the ‘savage’ portrayed to the populace was a reflection of savagery that was in fact a part of French life which seems to have been projected onto the life of the primitive. The alteration made to the painting consists of a reversal of the image accompanied by a number of other alterations which alter the meaning of the image of the original work to correspond more with the sensibilities of the late nineteenth century as opposed to the late sixteenth century (Richards 1994:147).
least, that kuru is only a disease of the “other”. Hysterical and extravagant behaviour was expected of those designated “savage” or “primitive”. For example, a press headline which appeared on October 6th 1957 in the Sydney Morning Herald reads ‘LAUGHING DEATH (Grim fate of natives) Strange disease hits N G’. Another example appeared two years later on September 30th 1959, when Gavin Souter wrote a piece in the Herald with the heading: “‘LAUGHING DEATH” HAUNTS TRIBE’. On October 11th that year the press reported ‘MORE DIE LAUGHING: Fresh Survey’. In 1990, Vincent Zigas named his book on the subject Laughing Death: The Untold Story of Kuru. Because the disease has been referred to in this way and because of the complex ways in which the notion of cannibalism has formed constructions of the “other” historically, it is not difficult to see how understanding of the disease might have been influenced. It might be argued that such widely circulating images have not affected researchers involved in the study of the disease, but researchers and non-researchers alike are influenced in the same way even if they do not recognise this. This is not to criticise but rather to argue, along with Arens (1998), that no one is a tabula rasa. Arens points out that ‘the theme of the “other” as cannibal had, and has, an existence beyond the control of those employing it at any particular moment’. He suggests that ‘the cannibal notion has the ability to manipulate those who harbour it rather than the other way around’. Such is the nature of cultural myths (1998:54). Arens highlights the fact that when Gajdusek first wrote about kuru in his journals, the people of the area are referred to as cannibals. At this early stage of the investigation there was no suggestion that this was linked to the disease. That linkage was not made until the nineteen sixties through the work of anthropologists. The point to make here, is that images and ideas that circulate in culture have unknown and sometimes un-recognised effects.

The critical writings that emanate from the fields of anthropology (Arens), literature (Richards) and psychology (Jahoda) introduce a major problem for an analysis of kuru, which has the notion of cannibalism so intertwined within its narratives. They cannot be ignored easily as once the ideas proposed in these works were published many disciplines including anthropology began to question their premises with regard to the construction of the ‘other’. Richards suggests that anthropology has begun to scrutinise its own discourse and assumptions regarding cultural representations of others, but that this is a long process (1994:290). The revision has not happened
without controversy. The idea proposed in The Man Eating Myth (1979) by Arens -
that cannibalism might not have been practised as a sanctioned norm in the past with
the exception perhaps of cannibalism for survival purposes – was challenged by
Riviere, who in a review in 1980 denounced Arens’s general proposition as
dangerous. Vincent Zigas (1990), a prominent figure in the kuru investigation,
suggested that such ideas were ridiculous. These opposing views indicate the
contested nature of debates about cannibalism and by association kuru. After spending
four years reading these critical works about the subtle effects that the notion of
cannibalism can have, I am still left with doubts about the relationship between kuru
and cannibalism. At the beginning of the study, my aim was to make some meaning
of apparently conflicting discourses regarding kuru and cannibalism, which
independently arrived at contradictory conclusions about the same event. Later, the
idea of multi-causality with respect to disease causation and transmission became far
more relevant as did the blind-spot that existed in the published work on kuru written
in scientific and popular discourse, which seems to have missed the insights gained in
art, literature and anthropology.

More reason for caution when analysing the investigation of kuru is given in the
recently published book entitled Darkness in Eldorado: How scientists and
journalists devastated the Amazon (2000) by Patrick Tierney, who questions the work
of anthropology, journalism and science during the nineteen sixties. Tierney starkly
reminds readers about research conducted in so-called “primitive” cultures in
collaboration with the Atomic Energy Commission at this time. By today’s standard
of ethics this work might be judged as unacceptable. But at the time it might also have
been seen as imperative. Because of the timing of the kuru study and the remoteness
of the location in New Guinea where the investigation took place, this work serves to
highlight the difficulty of analysing past events.

There are a couple of things that can be said with a little more certainty. There is a
marked contrast between the image of primitive people in a “stone age” world evident
in the discourse surrounding the ‘discovery’ of kuru, and the more clinical, scientific
discourse surrounding other prion diseases\(^6\). Another certainty is that the area where kuru occurred acted as a ground for scientific research in many fields including neurology, virology, immunology and molecular biology.

**Transdisciplinarity and method**

The aim now is to provide a brief survey of some of the literature that has informed this transdisciplinary study, with particular reference to materials and conventions.

The major influence on the project comes from the literature of Cultural studies, specifically how this is applied to scientific knowledge. Cultural studies of scientific knowledge has been broadly, defined by Joseph Rouse, who states that it includes ‘various investigations of the practices through which scientific knowledge is articulated and maintained in specific cultural contexts, and translated and extended into new contexts’ (1992:2). This includes paying attention to material culture, social practices, linguistic traditions and the constitution of identities and communities. As well, attention is paid to connotations of meaning. Cultural studies projects are conducted by researchers across a broad number of academic disciplines including historians, philosophers of science, sociologists and anthropologists. Generally, these scholars refuse ‘to require distinctive methods or categories to understand scientific knowledge (Rouse p.3) thereby de-emphasising the traditionally thought of boundaries between science and non-science, or between science and society. In opposition to the belief that scientific knowledge is separate from other knowledges, scientific knowledge is taken ‘to be a cultural formation that has to be understood through a detailed examination of the resources its articulation draws upon, the situations to which it responds, and the ways in which it transforms those situations and has an impact upon others’ (p. 4). One of the influences on cultural studies is the tradition of postcolonial anthropology, which, as Rouse argues, is suspicious of attempts to impose categories upon the Other’ (p. 10).

\(^6\) Arens (1990) remarks on this difference with respect to kuru and CJD, and points out how caution seems to be applied when discussing the cause and transmission of other prion diseases but the same does not apply to discussion about kuru.
I have paid attention to the significance of meaning and to the etymology of terms like 'kuru' and 'prion'. While writing about these words in chapter one, I have not been so interested in cause and effect relationships, as with where and when these terms are used in culture, the meanings that are ascribed to them at different times, and their effects. I have also been interested in exploring the roles played by metaphor and images, and the way these help shape understanding within culture. Taking these elements of cultural life into account means that a topic can be approached from a different angle than might be the case were it approached from within a scientific discipline. I have also paid attention to differing voices and highlighted inconsistencies rather than ignore them, as being sensitive to difference and contested meanings is part of a cultural studies project. All of these ways of approaching the study differentiate it from a scientific or traditional historical study.

Etymologically, the word *method* stems from the Greek term *methodos*, which literally means 'a going after' from *meta* = after + *hodos* = way (Collins English Dictionary 1992:718). But going after kuru and its stories is no simple matter as the stories have been spread far and wide, not only across a number of academic disciplines but also through press reports, novels and administrative, archival and popular texts. The study has also been informed by all of these works. As mentioned previously, Zigas (1975) suggested this dispersal, at least in part, was one of the reasons for the confusion found in the literature as each discipline comes to the disease from a different angle. It is vital that this thesis adds to our understanding of kuru, rather than adds to the confusion. How to do this in a way that is understandable to readers across a number of disciplines is one of the challenges I have tried to meet.

As with many scientific studies, the current one began with a broad research question: where, and how, does the discourse of cannibalism intersect with the discourse on kuru in particular, and prion diseases in general? Quickly discovering that it was hardly possible to find an article or book on kuru that did not mention cannibalism, I began to read about kuru research and about institutions that supported the investigation and to build up an overview of medical research conducted within a Post World War Two context. Attention was also devoted to tracing references in the published literature to gain a sense of the times. The issue then was how to interpret the material and how to write about it clearly.
The way I have structured the work can be characterised as *Bricolage*, a French term coined by Levi-Strauss. The term has been described as a ‘sort of construction of any sort made out of available pieces’ (Figlio 1976:31). Figlio points out however that while this might give the impression that factors are thrown together without reason or association, the pieces are put together with both purpose and design. A bricolage therefore is not something that is haphazard, but rather a complex method of putting together a narrative differently from the way a scientific work is arranged. The differences in style and structure of a work using bricolage and one using a scientific format are marked.

In his own work, Figlio suggests that a debate or series of debates about a certain topic in scientific history often have an organising principle. By looking for this organiser, it is possible to show that even when controversy surrounds an issue the same organising principle is often used and it is around this organiser that the debate spins. In his case, he shows how the notion of “organisation” itself was a concept flexible enough to be put to use by both sides of the vitalist/materialist debate in England and France in early nineteenth century scientific life. What he also shows is how this concept carried meaning in the social and political spheres. The meanings thus circulated across social, political and scientific domains. As Rouse has argued, the boundary between science and non-science is fluid and moves in two directions (1992:13).

In the kuru story specifically, and in the stories written about prion diseases generally, I would argue that the organising principle is the notion of cannibalism.

An example of the reliance on the notion of cannibalism can be found in the titles of the chapters of Richard Rhodes’ book on the subject of prion diseases, *Deadly Feasts: Tracking the secrets of a terrifying new plague* (1997). Part one of the book entitled ‘Among the Cannibals’ keys the reader into the subject. The first chapter ‘I eat you’ is devoted to a description of the way bodies were ostensibly, sectioned prior to being eaten in New Guinea. The last chapter of Part One, ‘The cannibal connection’, outlines the way in which the anthropologists Robert and Shirley Glasse developed their ideas about how cannibalism and kuru might be associated. This involved an article in *Time* magazine that talked about a particular flatworm, *planaria* that had
been chopped up and fed to other planaria. The cannibal planaria were said to be able to remember a maze that the initial planaria had been made to travel through. Rhodes points out that the planaria theory was later discredited (1997:101). Nevertheless, the Glasses were intrigued by the idea and began to think about the ways that cannibalism might be involved in the transmission of kuru. It acted therefore as a model. Part Two of Rhodes’s book moves onto the subject of the transmission of Creutzfeldt-Jakob disease through ‘High-Tech Neocannibalism’, which is the title of chapter eight in his book. Part Three, chapter eleven, entitled ‘Meat bites back’ moves onto discussion about BSE. This example demonstrates how the notion of cannibalism is used by Rhodes to unify and organise the discussion about prion diseases.

Jennifer Cooke in her book *Cannibals, Cows and the CJD Catastrophe: Tracking the shocking legacy of a 20th century disease* (1998), also uses the notion to organise. The opening sentence in the Preface of her book reads:

> This is a story about cannibalism and its legacies. It’s about the cannibalism recognised for centuries – humans eating other humans. And it’s about new-age cannibalism – feeding an animal species back to itself for recycling, for added protein, for profit. It’s also about high-tech cannibalism – transplanting of organs, of tissue, of blood or other bodily parts into members of the same species or another species’ (Cooke 1998).

The final chapter, ‘The shocking legacy of cannibalism’ sums up the various ways cannibalism is involved in all of the prion stories.

One further example can be found in the book *Fatal Protein: The Story Of CJD, BSE And Other Prion Diseases* (1998) by Ridley and Baker. The separation between the various prion stories is common in all three examples cited. Chapter two, ‘Concerning sheep’, is followed by ‘Kuru, a story of cannibalism’, which is then followed by chapter four, ‘Creutzfeldt-Jakob disease, the emergence of a disease entity’, and so on through the other prion diseases. This work, from the Department of Experimental Psychology at Cambridge is also written for a popular audience, and as with the other two books mentioned, cannibalism, if not organising the whole work, certainly organises the chapter on kuru.
Cannibalism is an emotive subject and a fascinating one, but by framing the stories
told about kuru, CJD and BSE in this way other parts of the story are bypassed. This
is not meant to detract from the serious way in which all of these authors treat relevant
issues related to high tech medicine and agricultural practices, but none of them focus
solely on the iatrogenic aspects of the discourse.

Because the current study used a particular approach, one where questions were raised
rather than one where closure was sought, it has been possible to look for other
organising principles of interpretation that might also work in the case of kuru. In
relation to at least two other prion diseases - scrapie, and iatrogenic CJD - iatrogenesis
is clearly implicated. The intention of this enquiry is to broaden the debate about kuru
and to provide circumstantial evidence that serious consideration should be given to
the possibility of iatrogenic transmission in the case of kuru. This requires attention
being paid to the historical and institutional context of kuru research.

As I progressed with this study, the utility of disciplinary boundaries became obvious,
as their construction enables a common language to be spoken in shorthand by a
group of people with similar interests. There is a down side to writing within any one
discipline though, in that language has a tendency to become ever more arcane with
the result that it is difficult for those outside of the boundary to understand a given
subject. The following paragraph is one example:

The most likely explanation for the successful transmission of SHa(Sc237)
prions to Tg(ShaPrP) mice expressing SHAPrP and the failure of transmission
of HuCJD prions to Tg(HuPrP) mice expressing HuPrP is the relative degree
of homology of SHAPrP and HuPrP with MoPrP: SHAPrP differs from MoPrP
at 16 amino acids whereas HuPrP differs at 28 (DeArmond & Prusiner

Another prominent researcher in the area, Paul Brown, has remarked that ‘current
experiments combining transgenic and “knock-out” animals involve a dizzying range

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While I am arguing against the use of this organising principle it needs to be said that I have also used
it in a paper I wrote in 1998: ‘Modern Day Cannibals and the Commodification of the Pituitary Gland’
which was presented at the COMMODIFICATION: theories, practices, histories and representations
Conference, Institute of Social Change and Critical Inquiry, University of Wollongong, NSW. The use
of the term here was related to coming to terms with feeling as though I had unknowingly been turned
into a modern day cannibal having received human pituitary hormone for the treatment of infertility.
of crossed and back-crossed mouse strains and gene copy numbers that threaten to become almost as difficult to interpret as the classical scrapie agent-mouse strain experiments of an earlier era’ (Brown 1994:85). Brown suggests that ‘in the fullness of time [these] should yield a coherent explanation of what is presently a very complicated story’. I hope he is right. In the meantime, left with a discourse so arcane, it is perhaps a good time to reflect back on the knowledge that has accumulated over the twentieth century.

**Arrangement of the work**

Chapter One revolves around meaning and definition and aims to show the different meanings that come with the terms, prion and kuru within different disciplinary settings, and how meanings outside of the biomedical field also have associations with either the kuru story or the history of New Guinea more generally. For example, in ornithology, prion is the name of a bird, which was written about in 1943 in a rare book co-authored by Edward Hallstrom. Hallstrom, from the early nineteen fifties, was influential in the development of New Guinea’s cattle and sheep industry. Likewise, the term kuru has meaning within different disciplinary contexts. Here, I explore the meaning of kuru as disease, kuru as sorcery, and nakurutitis disease of sheep and cattle. Through an exploration of the different meanings, the reader will be introduced to some of the complexity that confounds prion research.

Chapter Two begins with discussion about the arrival of Dr D. Carleton Gajdusek in New Guinea in early 1957 and questions the serendipity narrative that is dominant in many of the accounts written about the disease. Having challenged this narrative, the chapter focuses on the period at the end of World War I at which time a mysterious disease was reported in medical journals in Australia. The aim is to track the research on this disease, which was renamed X disease, through to the early nineteen fifties with the outbreak of Murray Valley encephalitis (MVE). This will enable me to show the overlap between some of the scientists who were involved in these studies as well as with the kuru investigation in 1957. An examination of this crossover through the decades will help to shift focus away from the idea that kuru happened out of nowhere in 1957, an idea that is not difficult to obtain when reading many of the more popular
accounts of kuru. Kuru may have been a unique disease, but nothing occurs in isolation. It, like any other line of research, has a history. The first file in Series 10 of the Burnet Papers provides a possible clue to the relevant background as it contains lengthy discussions about post vaccinal encephalitis, X disease and louping ill virus. As louping ill vaccine has great relevance for the transmission of scrapie it is difficult to ignore the arrangement of the Series.

With the help of the second file in Series 10 of Burnet’s Papers, Chapter Three examines the negotiations that took place during 1955 and 1956, just prior to the kuru investigation. With the help of this file and supplementary material, I aim to show the collaboration that existed throughout the nineteen fifties between Australian and American researchers who were involved in the kuru investigation. Much of this earlier work focussed on a group of diseases known as Group B encephalitides. Diseases included in this category are yellow fever, dengue fever, Japanese B encephalitis and Murray Valley encephalitis. All were thought to be mosquito borne diseases in nature. Louping ill is also a member of Group B but it was believed, in distinction from the other members of the group, that ticks were responsible for its transmission. By paying attention to the collaborative efforts of researchers, relating to the study of Group B diseases - particularly MVE - and kuru, it is possible to highlight the nexus between research on MVE and research on kuru and the scientific and institutional context within which the investigation of kuru was negotiated. In addition, the chapter provides a brief sketch of aspects of pre and post World War II scientific history that need to be considered when analysing prion diseases from a cultural perspective.

Chapter Four changes focus to discuss some of the problems faced by the institutions involved in the kuru investigation. This includes the work carried out at the Commonwealth Serum Laboratory and the Walter and Eliza Hall Institute. The major problem for these institutions between 1955 and 1964 relates to the production of vaccines and sera free from unwanted side effects. Polio vaccine was particularly problematic during this period and so it is worthwhile diving below the surface gloss of heroic attempts to wipe out this scourge to find that tensions ran high as scientists and institutions grappled with the complexities involved in vaccine production. Parallels are drawn, and overlaps explored between the study of kuru and the study of
polio. Through an examination of the institutional context, circumstantial evidence is provided in support of the idea that kuru might have been the unwitting result of a vaccine.

The theme of unwanted effects from vaccines is followed through in Chapter Five, with an emphasis on some of the meetings and conferences held on the subject. The chapter in general is primarily concerned to show that some of the scientists involved in kuru research were aware of the hazards of immunisation and contributed to the discussions that took place over the years at meetings and conferences on the subject. Another aim is to show how kuru was categorised at the beginning of the investigation in 1957, that is, within the same context as multiple sclerosis and Parkinson's disease, both of which at the time were considered to be diseases of unknown origin. The information provided does not go as far as to prove a relationship between kuru and vaccines but it does add to the circumstantial evidence in support of the idea that kuru might have a much closer relationship with a medically acquired disease than has hitherto been countenanced.

Chapter Six changes focus again by concentrating on events during the second half of the twentieth century. It begins with a brief examination of how information is disseminated about vaccine-associated disease and highlights the fact that this has been lacking in many respects. A comparison is then made between the lack of dissemination of information about vaccine research and a similar situation with respect to prion research generally, and its administration. The chapter focuses on the inquiries held into two prion diseases (iatrogenic CJD, and BSE) in order to learn about the way information about the safety of medicines is managed and to highlight the complexity of prion research.

Chapter seven, through an analysis of evidence presented at the BSE Inquiry, begins with an examination of vaccination policy in England during the nineteen eighties. One of my aims is to highlight factors that influenced policy decisions and to assess whether the policy decisions taken were appropriate in light of knowledge that was available about the efficacy of various routes of transmission of prion diseases at this time. Kuru and its perceived transmission route, is an important part of this debate, and I show how this disease was spoken about in relation to analysis of risk. A
comparison of the British and Australian situation is then made to show the similarity between policy in the two countries. The thesis concludes with a comparison of the situation that existed in America at the beginning of the twentieth century to show how little has changed with respect to the way information about the potential risks involved in the use of vaccine is managed.
Chapter 1

Prion and Kuru: Definition and Meaning

The terms ‘prion’ and ‘kuru’ have a variety of meanings dependant on where they are used. The intention here is to survey a number of these meanings and highlight their relevance to our understanding of kuru. Paul Brown has drawn attention to Prion Bay in Tasmania and recounts the following traveller’s tale to express the complexity involved in research on prion diseases.

As I left the heights and began my descent toward the long beach of Prion Bay, every thing changed. The bare, wind-tossed tops gave way to some of the thickest, most tangled and tortured rain forest it has ever been my misfortune to encounter. Out of the bright light and into a gray-green gloom of a nefarious netherworld, I attempted to follow a trail that had the remarkable ability of vanishing in the difficult places, leaving me scrambling through mud and slime and decaying mossbeds without any sense of direction – except down (Brown 1994:81 quoting Yeadon 1992).

The tale is a beautiful metaphor in that it provides an image of the difficult terrain on which prion researchers, governments, and administrators stand while working to prevent and control diseases like scrapie and BSE in animals, or kuru, iatrogenic CJD and the new variant form of CJD in humans. Brown also draws attention to another use of the term in ornithology where Prion is the name given to a group of birds that proved particularly difficult to categorise, not unlike prion diseases. In yet another field, insect virology, Neodiprion is the name of an insect.

The term Kuru on the other hand refers simultaneously to a particular prion disease, a type of sorcery in New Guinea and the name in local language for trembling with fear or cold. In the field of animal research the word nakuruitis refers to a disease of sheep and cattle that occurred in Kenya during the late nineteen forties and early fifties. The survey will help to highlight meanings attached to the terms, prion and kuru.

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1 The tale is referenced to a piece written for the Travel Section of the Washington Post on October 18th, 1992 page E1.
Prion as disease

The term *prion* is used in the context of disease research as if it was a novel term and yet it carries other meanings dating back to an earlier era. For two decades the term has been used within the realms of biomedicine and molecular biology for the name of a protein that is found in the body which somehow becomes corrupted or altered. The prion protein comes in two forms, cellular and scrapie. These forms are designated by PrP$c$ and PrP$sc$ respectively, and indicate the close association between human and animal disease in the discourse. There has been conflicting opinion over whether the build up of altered protein in the body is the cause of the disease or the result of another process. However, in 1991 one commentator wrote that ‘it was previously felt that the prions were the causative agent itself, ... now we understand them to be the manifestation of the disease process, rather than the cause’ (Ford 1991)$^2$. In other words, prion proteins are probably not the cause of prion disease but rather the end of a process of unknown origin.

The first use of the term in the context of molecular biology is attributed to Stanley Prusiner who received the Nobel Prize for Physiology and Medicine in 1997 for his work on prion disease. For an account of Prusiner’s ideas I draw on Richard Rhodes’s *Deadly Feasts* (1997), as it gives a sense of the complexity of the issues involved without being highly technical, and also portrays how the disease is spoken about for a popular readership$^3$. Prusiner first coined the term *prion* in 1982 when his paper ‘Novel Proteinaceous Infectious Particles Cause Scrapie’ was published in the American journal *Science* (Rhodes 1997:162).

Prusiner’s prion theory has been controversial as it proposes that “six separate and distinct lines of evidence” all show how “the scrapie agent contains a protein that is required for infectivity”. This means that rather than a virus or bacteria being the

$^2$ Brian J Ford was the Chairman of a Meeting held in London on April 29th 1991 at the Linnean Society, Burlington House, Piccadilly. At the time Ford was Chair of the History of Biology, Institute of Biology, London. The name of the meeting was ‘Bovine Spongiform Encephalopathy’.

$^3$ It is relevant to note that Richard Rhodes’s previous two books written before *Deadly Feasts* relate to weapons research. The titles are *Dark Sun: The Making of the Hydrogen Bomb* (1995), and *The Making of the Atom Bomb* (1986), the latter winning the Pulitzer Prize. *Deadly Feasts* is written in a more popular vein and lacks the scholarly approach taken in the other two books in that it does not cite references in the text nor does it contain a bibliography.
agent of disease - both of which are known to have genetic material as part of their constituents - the agents are “small pro-teinaceous in-feccious particles which are resistant to inactivation by most procedures that modify nucleic acids” and this “underscores the requirement of a protein for infection....” (Rhodes 1997:162 quoting Prusiner 1995). The term, then, derives from an emphasis on the pro part of protein and the in part of infectious, although it remains a mystery why the ‘o’ in pro and the ‘i’ in in have been transposed to read pri/on pronounced pree on.

Prusiner suggested that the term should replace the use of “unconventional virus” and “unusual slow virus-like agent” as it gives a more accurate sense of the protein particle involved. According to Rhodes, Prusiner’s prion theory is ‘essentially a variant of [Carleton] Gajdusek’s theory of protein crystallization’ and notes that Prusiner and Gajdusek agree that the particle does not consist of nucleic acids (Rhodes 1997:206). Other scientists however have been sceptical about the protein only hypothesis and believe that a ‘naked plant DNA’, or something similar could be responsible. Dr A.G. Dickenson in England speaks about a virion rather than a prion (Rhodes 1997:164). This slight alteration in name signifies the inclusion of genetic material, however small the fragment, and highlights the uncertainty associated with this area of research in terms of understanding exactly what it is that causes, or is the effect of, the disease. Laura Manuelidis (2000:2083), another researcher in the field, suggests that Prusiner has overemphasised the protein aspects of the discourse and in a review of Prusiner’s latest book Prion biology and diseases (2000) says that ‘it leads one to re-examine the objectivity of science and whether it is a myth vanished’. Manuelidis argues that too much emphasis has been placed on the use of the declarative sentence. Whether one agrees with this conclusion or not is not as important as understanding the contested nature of this field of research.

It is not surprising that different theories have been proposed for naming the agent of disease in light of the uncertainty involved in the field. Different views in a new field of research can be seen as the sign of a healthy community, but this field of research was not totally new in that there were at least two historical points of reference. Rhodes suggests one of these when he says that similar ideas to the ones proposed by those who believe in the protein only theory were suggested by the British mathematician J. F. Griffith in 1967 (1997:206). Another, which might also have
relevance for the protein only idea, is the research carried out by W. M. Stanley on plant viruses. In 1943 during a visit to America, Burnet noted that Stanley was the "man who first isolated a virus as a pure molecular unit of protein" (Sexton 1991: 102-106). This means that the time frame that marks the point at which it was thought possible that a virus could consist only of protein needs to be moved back to the nineteen forties, rather than the 'sixties with Griffith's work or Prusiner's in 1982.

**Neodiprions**

Within the field of insect virology, the term *Neodiprion* is used to refer to a particular group of insects, which succumb to a viral disease. Three examples are *Neodiprion americans banksianae*, *Neodiprion pratti banksianae*, and *Neodiprion sertifer*. The possible relevance of research in this area to the investigation of kuru is twofold. At the end of 1958, the year following the initial kuru investigation, MacFarlane Burnet and W. M. Stanley edited a three-volume edition entitled *The Viruses: Biochemical, Biological and Biophysical Properties* (1959), in which *neodiprion* is used with reference to polyhedral diseases^4^. At this time Burnet was involved in the investigation of kuru.

Polyhedra are protein crystals that seem to have virus particle occluded in their constituents (Smith in Burnet & Stanley 1959:371)^5^. During the 1940s, it was found that when the virus particles were released from the polyhedra through chemical means the polyhedra were not in themselves infectious. Rather it was the virus that was infectious when occluded within the polyhedra or protein crystal. From this we can see that it was not only during the nineteen seventies, eighties and nineties that scientists pondered over the matter of how proteins could be involved as agents of

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^4^ The early work on insect viruses focussed on the polyhedral viruses because of their easy recognition under the electron microscope. They could be found in either the nucleus or the cytoplasm of the various cells in the body of the insect, and were given the names nuclear or cytoplasmic polyhedral disease. Under the microscope, the polyhedra were seen to consist of 'many-sided crystalline inclusions associated with this type of virus' (Smith in Burnet & Stanley 1959:369). By 1959, two distinct groups of polyhedral virus diseases had been identified and named respectively, the *polyhedroses* and the *granuloses*. Smith also discussed a third group in which no intracellular inclusions were found. Whilst not wanting to draw too close an analogy, it is interesting that kuru as a prion disease shares this feature. Kuru pathology does not show signs of intracellular inclusions.

^5^ K. M. Smith worked at the Agricultural Research Council, Virus Research Unit, Cambridge, England
disease. Similar problems had been identified much earlier within the field of insect virology and Burnet was involved in the publication of Smith’s work in this area. Given Burnet’s involvement in the investigation of kuru during the late nineteen fifties, it is an interesting coincidence that he edited Smith’s work on neodiprions and more than twenty years later kuru and diseases like it became known as prion diseases because of Prusiner’s transposition of pro and in.

The second way that research on neodiprions might have relevance for research on the prion disease kuru, is that one of the insects that succumbs to polyhedral disease is *Neodiprion sertifer*. This is the name of the European pine saw fly (Smith in Burnet & Stanley 1959:372). At the time of the kuru investigation in 1957, pine plantations existed at Okasa on the south-eastern border of the kuru region. There are four elements here: Kuru, a strange prion disease in humans; a specific geographical location; a European pine plantation nearby; and a strangely named insect *neodiprion sertifer* that might have lived in the same habitat. Without further research it is not clear if this association is significant in more than name only. It is however an indirect example of how the meaning attached to neodiprion in the field of insect virology intersects with the meaning ascribed to the term in molecular biology - where prion is the name of a disease and a protein rather than the name of an insect.

It is as if this earlier line of investigation in the field of insect virology provided a prelude for what was to come. And yet references to this work are absent from the discussion of modern prion research. Maybe it has simply passed by unrecognised because it was carried out in the field of insect virology and not medical science. Whatever the case, its relevance might be more than in name only because just like recent research on prion disease, inquiry into neodiprions was equally embedded within a discussion about crystal-like fractions, some of which were difficult to detect under certain conditions.

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6 The third volume refers to neodiprions on pages 372, 373, 385 and 490. *Neodiprion americanus banksianae, Neodiprion pratti banksianae, and Neodiprion sertifer* can all succumb to polyhedral diseases.

7 *Neodiprion sertifer* can acquire a disease that affects the nucleus of cells, thus the classification of nuclear polyhedrosis.
Prion disease and the Ice 9 metaphor

Judging by the number of scientists (Byron Caughey & Peter Lansbury 1995, Caughey 1997, Carleton Gajdusek, Gajdusek 1994) who have written in this area, research on crystalline structures in relation to disease continues to be of relevance in the field of prion research. These scientists use the metaphor of 'Ice 9' employed by Kurt Vonnegut in his work of science fiction *Cats Cradle* (1963) to explain the mechanism of prion disease pathology. Here, the lines between non-fiction and science fiction become blurred. While there is nothing unusual about scientists using a metaphor to explain how something works, in this instance it is difficult to distinguish which came first, scientific experiments in the laboratory or ideas expressed in works of science fiction. Sometimes, works of science fiction are considered to be predictive, for example Huxley’s *Brave New World* or Shelley’s *Frankenstein*. Science in the so-called real world follows the worlds imagined in this schema. In the prion story, things did not proceed in this direction.

Caughey (1995:1) explains that Felix Hoenikker, the main character in *Cat’s Cradle*, created the substance called *Ice 9*, which was ‘the most terrible weapon’ the world had ever known. The substance was known to be more stable than normal ice, with a melting temperature of 114.4 °F, and was also kinetically inaccessible under standard conditions. In the book, Felix’s son Newt demonstrated that ‘ice 9 crystals seed their own replication’ and in the process he wiped out the world’s water supply. It is known that whatever prions are, when they shift into the altered form they are highly stable, and resistant to most known chemicals. Caughey suggests that Vonnegut’s *Ice 9* metaphor is useful in understanding how the normal form of prion protein in the body might change into the pathological, mutated form. He suggests that ‘the notion that a kinetically inaccessible, but thermodynamically stable’ form of a natural material could be very dangerous and might be relevant to prion disease.

As an insight into the potential danger, Rhodes describes a section in *Cat’s Cradle* where Dr. Breed speculates on the idea that there may be other forms of normal ice. In the book, normal ice is called Ice-one. Vonnegut writes,

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8 Gajdusek in interview with Rhodes (1997)
“Suppose water always froze as ice-one on Earth because it never had a seed to teach it how to form ice-two, ice-three, ice-four ...? And suppose there were one form, which we will call ice-nine – a crystal as hard as this disk – with a melting point of, let us say ... one-hundred-and-thirty degrees... When [the rain] fell, it would freeze into hard little hobnails of ice-nine – and that would be the end of the world!” (Rhodes 1997:196 quoting Vonnegut).

In the book, a nucleant of *Ice 9* is accidentally dropped into the ocean and all the water on earth freezes solid, thereby making it impossible for biological processes dependant on liquid water to take place (see Rhodes 1997: 196). According to Rhodes, Gajdusek ‘visualized a similar infective process at work’ in the transmissible spongiform encephalopathies (TSEs) (Rhodes 1997:197), which are also referred to as prion diseases or infectious amyloidoses.

In kuru the plaques in the brain consist of ‘great crystalline knots’ known as scrapie associated fibrils (SAFs), which accumulate. According to Rhodes, Gajdusek is said to have proposed that the infectious agent was a nucleant crystal of abnormal prion protein (PrP<sup>Sc</sup>). When the abnormal form makes its way to the site in the body where the normal protein (PrP<sup>Sc</sup>) is made, that is, in the membranes of nerve cells, the abnormal form displaces the normal protein and teaches the forming protein to ‘pattern itself’ as abnormal PrP (Rhodes 1997:197). With this idea in mind, Gajdusek is said to have changed his views about the agent away from the idea that a slow virus was at work, towards the idea of an infectious amyloid. That amyloid can also be infectious is a worrying prospect in that it can be transmitted experimentally from species to species but it is not an idea confined to the realms of fiction.

A similar process was at work in an experiment conducted on EDT, which was a compound widely used in industrial processes during the nineteen forties (Rhodes 1997:196). In a paper written in 1994, Gajdusek described how during World War Two an industrial “infection” occurred in which ‘the precipitation of ethylene diamine tartrate in a new crystalline state’ was formed (Gajdusek 1994:173). Gajdusek points out that the new form of EDT was studied as if it was a ‘transmissible replicating microbe’. It soon became apparent that the “infected” form of EDT did not work like the normal form, thus highlighting the similarity between prions and this phenomenon. Rhodes describes the infected form as ‘junk’. Whether or not this is the correct explanation, the industry was crippled because the process could not be
reversed⁹. Rhodes follows his account of the factory accident with his account of Kurt Vonnegut’s book *Cat’s Cradle*, which he says is fortunately only an apocalyptic novel. Nevertheless, some of the elements of this story have some uncanny resonances with prion research, as the work of Caughey and Gajdusek attests. For it is the Ice 9 metaphor from *Cat’s Cradle* that modern scientists use to explore and possibly explain the process of prion replication.

Gajdusek (1994) suggested that Kurt Vonnegut was one of the dreamers who has ‘priority on many of the ideas of today’s amyloidology’ (Gajdusek 1994). Rhodes’s work shows however that it was Vonnegut’s brother who had priority over the ideas that were then expressed by Vonnegut in *Cat’s Cradle* through the Ice 9 metaphor. Rhodes was keen to know how Vonnegut ‘happened upon the idea of an end-to-the-world variant form of ice’ (Rhodes 1997:208). He notes in his book that he had known Kurt Vonnegut since 1965, when he interviewed him for an article in the *Paris Review*. Having heard Gajdusek speak about the crystallisation theory of the transmissible spongiform diseases (TSEs) and having read Caughey and Lansbury’s papers, he re-read *Cat’s Cradle* before again contacting Vonnegut. It transpired that Vonnegut’s ‘brother had been a physical chemist at MIT and had done pioneer work on cloud seeding’. When Rhodes reminded Vonnegut of the EDT factory accident, he is reported to have replied, “That too”. Rhodes then told Vonnegut about the transmissible diseases we now call prions and the ‘possibility that they infected like ice-nine by crystal nucleation’. Apparently, Vonnegut replied, “Wouldn’t you know” (Rhodes 1997:208). This shows that despite what Gajdusek says about Vonnegut’s priority, the idea for the Ice Nine metaphor came from Vonnegut’s brother’s first hand experience in scientific experiments. The question is what relevance might this research have for the emergence of human prion diseases like kuru as opposed to the metaphor simply being useful to understand the agents of disease?

When considering the term *prion* - what it stands for, and how it works - the broader field of research which has become enmeshed in the prion story consists of industrial

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⁹ Ethylene diamine is thought to be the chemical responsible for the Mobil AVGAS problems that were experienced in January 2000 in the Australian airline industry, which brought much of the light aircraft to a standstill.
accidents, weapons research (when the ideas in *Cat’s Cradle* are also taken into account), and experiments on cloud seeding.

Cloud seeding is a process whereby rain can be made by ‘pumping crystals of silver iodide into clouds to supply nucleant around which raindrops could coalesce’ (Rhodes 1997: 208). Similar experiments took place in Australia during the nineteen forties. In 1947, Kraus and Squires published an article called ‘Experiments on the stimulation of clouds to produce rain’, which drew on earlier work conducted by Schaeffer and Langmuir, who had suggested a ‘possible method of planting ice particles in natural clouds’ (Kraus & Squires 1947). The Radiophysics Division of the Council for Scientific and Industrial Research (CSIR), Commonwealth of Australia, was also involved in this work.

Whether these experiments coincided with those in which Vonnegut’s brother was engaged is not clear without further research, but as well as being useful for producing rain, cloud seeding might have been a way to assist in the deployment of biological agents. Alexander Langmuir was known for his work in this area (Etheridge 1992:36). Burnet was another leading scientist who, during the nineteen forties and fifties, was actively involved in war research and called upon for his expertise in biological and nuclear science (Burnet 1968. Sexton 1991). Whether this line of research has any relevance for the emergence of prion diseases like kuru is difficult to say, but whatever its purpose, cloud seeding was a phenomenon that was associated with the Ice 9 metaphor. And Ice 9 is used to explain how prions alter their form and build up to produce disease.

Moving away from the discipline of biomedical science and looking instead to the discipline of ornithology where *Prion turtur* is the name of a bird, we can draw on another metaphor that is useful to explain prion research.

*Prion turtur: another metaphor for thinking about prion disease*

This meaning of *prion* is rarely mentioned within biomedical and biomolecular discourse on prion diseases, with the exception of Paul Brown’s article, ‘The “Brave
New World” of Transmissible Spongiform Encephalopathy (Infectious Cerebral Amyloidosis)’ (1994). Brown uses the image of the bird Prion turtur as another metaphor, this time to highlight the elastic-like quality of mind that is required when researchers grapple with new information on prion diseases.

Prion turtur belong to the genus Pachyptila, which is a ‘family of southern hemisphere seabirds’, and according to Brown, is the “original” prion10. The ‘earlier ornithologic usage proved to be remarkably prescient, given its wings to accommodate evolving knowledge about its nature’ (Brown 1994:81). The difficulty involved in classifying this group of birds was described by Gregory, M. Mathews and E. J. L. Hallstrom11 in a rare book Notes On the Order Procellariiformes, written in 1943. Notes records the names given to this Order since 1934, and includes a chapter with the title, ‘Discussion of the Major Changes of Nomenclature’, which is followed by a chapter on ‘Prions’12. The controversy over naming the birds then is little different from the controversy over naming the agent/s of disease now. Mathews and Hallstrom purposely aimed to avoid controversy in their work in the hope that the ‘facts portrayed’ would ‘be considered as a contribution to the stable nomenclature of the [tube-nosed] petrels’. No doubt a similar hope is in operation in relation to prion disease research. If only it was as easy as portraying the ‘facts’ to stabilise any field of research, let alone this one.

One example of where beliefs about kuru have changed over the years in light of new evidence relates to the incubation period of the disease. Exactly how long was it? When the investigation took place in 1957, because the youngest person seen by the

10 The print in Brown’s article comes from Plate No. 54 of John Gould’s 1848 publication The Birds of Australia (vol. 7) (see Brown 1994:82).
11 Hallstrom also helped in the preparation and publication of the book commemorating the First Inauguration of the Council of New Guinea (1951). J. K. Murray, the then Administrator of the Territory thanked him in his opening address at the meeting for ‘a gift’ of £40,000, which ‘greatly helped and expedited’ the development of a cattle and sheep industry in the Territory. Hallstrom also ‘donated £10,000 to build the Hallstrom Pacific Library, associated with the Australian School of Pacific Administration’. In a later work, Murray said that Hallstrom ‘did a great deal for the territory and its interests’ because of his interest in the territory’s fauna and flora (Murray 1968:336). Speaking about the development potential in his opening address Murray noted ‘the high cost per head of beasts landed in the Territory’. Because of this potential impediment to development the Administration established ‘a number of breeding stations in key locations in the main climatic environments’, the intention being to ‘breed good quality stock for sale to private buyers’. As a measure of Hallstrom’s influence in the affairs of New Guinea at this time, J. K. Murray’s remarks are useful.
12 Pages 22 to 27 in Mathews & Hallstrom 1943.
investigators was four years old it was presumed that the incubation period could not have been more than four years. This was predicated on the idea that vertical transmission does not occur. But as the disease kept appearing, by 1995 the incubation period was believed to be as long as 36 years\textsuperscript{13}. Tied up with the length of the incubation period is the idea that exposure to the agent of kuru occurred at mortuary rituals involving some form of cannibalism and that cannibalism stopped when the New Guinea Administration gained control of the restricted area, which occurred by the late nineteen fifties. Jared Diamond captures this idea: ‘Kuru was on its way to exterminating New Guinea’s Foré tribe of 20,000 people, until the establishment of Australian government control around 1959 ended cannibalism and thereby the transmission of kuru’ (1997:208). Based on this belief it has been assumed that no one contracted kuru after about 1960.

At the International Workshop on Creutzfeldt-Jakob Disease (CJD) held in Melbourne in 1995, there was a degree of curiosity from the participants in relation to the cannibalism hypothesis. Specifically referring to eight recent cases of kuru that existed in people from New Guinea who were in their fourth, fifth and sixth decade of life, the presenter of the section on kuru noted that ‘Every case that is seen can be traced to a cannibalistic act’ (Masters 1995:34). In 1998, Robert Klitzman (1998) wrote a popular account of his attempt to verify that each recent kuru victim was involved in a mortuary ritual involving cannibalism and his work is taken as virtual proof that the disease was acquired through this route. What is important to highlight at this point is the elasticity required when accumulating knowledge about prion disease and the utility of the Prion turtur metaphor to remind us of this. The need to accommodate knowledge as it developed about the bird applies equally to the way knowledge is developed about prion diseases. In the case of kuru though, new information does not seem to be allowed to suggest new frameworks of interpretation.

The idea that there might have been more than one point of exposure to the agent over time, which would account for the cases that have occurred long after the area was ‘controlled’ by the Administration, and cannibalism presumably ceased, does not

\textsuperscript{13} This information comes from the meeting, Summary of the International Workshop on Creutzfeldt-Jakob Disease (CJD), held in Melbourne, Australia in May 1995.
seem to have been canvassed. Nor the idea that something other than cannibalism might account for the disease in some cases. Given that iatrogenic causes have been implicated in other prion disease like CJD in humans, and scrapie in sheep (Brown et al 2000, Brown in Baker & Ridley 1996: 139-154, Brown 1990, Rappaport 1987), it seems worthwhile remaining open to other possible causes of kuru. In the case of scrapie outbreaks in Australia and New Zealand during the early nineteen fifties, an association has been made with the unanticipated effect of loping ill vaccine (Zlotnik in Gajdusek, Gibbs and Alpers 1965:275).

It is time to move on to explore how the term kuru has been used in various settings. This will help to introduce the influence of anthropology in the study of the disease and the narratives written about it.

**Kuru: sorcery and disease**

The influence of anthropology in the literature on kuru throughout the nineteen fifties (Berndt 1958), sixties (Mathews, Glasse & Lindenbaum 1968) and seventies (Lindenbaum 1979. Glasse and Lindenbaum 1971) is fundamental and notions of cannibalism and sorcery feature prominently. *Kuru Sorcery: Disease and Danger in the New Guinea Highlands* (1979), the title of Shirley Lindenbaum’s text, highlights this point. What this means is that as well as being embedded within stories about industrial accidents and an array of experiments, the discourse on kuru is embedded within a long tradition of European anthropology, which has been criticised in recent decades in regards to how it constructs its subject.

In *Kuru Sorcery*, Lindenbaum defines kuru as ‘... a fatal neurological disorder very common among the Fore, and present to a lesser degree among neighbouring groups, but unknown elsewhere in the world’ (1979:9). As Lindenbaum points out, the thinking in the West has been to consider kuru ‘...a slow virus infection spread by the ingestion of human flesh’. This western view can be contrasted with the perceptions held by the people of the region who believed that kuru was caused by the malevolent activity of sorcerers. As well as being the name of the particular type of sorcery that caused the disease, *kuru* was a word that meant trembling. This is picked up in
western discourse on the disease which Lindenbaum notes is ‘... marked by loss of balance, incoordination [ataxia], and tremor’ followed by ‘complete motor incapacity and death in about a year’ (1979:9).

In respect of Western views about the cause of the disease, at first it was believed to be of psychosomatic origin. This idea was prevalent in Australia when the first case was sent to the Australian government hospital at Kainantu in 1955 and was given a provisional diagnosis of “acute hysteria, in an otherwise healthy woman” (Lindenbaum 1979:14). In the late 1950s, it was proposed that kuru was a hereditary disorder because it was observed that the disease ran in families and was localised to a small interrelated population. A single autosomal gene that was dominant in women, but recessive in males was thought to be the cause. Lindenbaum (p. 16) also points out however, that with a deeper understanding of Fore kinship relationships it was seen that many of the victims were not biologically related at all. More recent studies support this view by showing that there are no genetic markers for kuru, unlike the sporadic and familial forms of Creutzfeldt-Jakob disease (Gajdusek 1994:176). Nevertheless, during the late nineteen fifties consideration was given to quarantining the kuru region because the disease was perceived to be genetically mediated.

Based on this belief, it was felt necessary to return to the area any person who had left the area for whatever reason. Medical researchers and government officials debated whether an invisible fence could be constructed around the area. As Lindenbaum says, it was thought that this would contain the problem of the supposed deadly kuru gene and the affected people would ‘... continue to transmit the disease one to the other until their tragic extinction’ (Lindenbaum 1979:15-16).

In 1962/3, together with Robert Glasse, Shirley Lindenbaum presented evidence that ‘... kuru had spread through the Fore population in recent times, and that its high incidence in the early 1960s was related to the cannibal consumption of deceased kuru victims’ (Lindenbaum 1979:19). Lindenbaum remarks that the cannibalism hypothesis seemed to fit the epidemiological evidence, which showed that women and children were the groups affected most and it was these groups who were said to engage in ritual cannibalism.
The cannibalism idea did not fit all of the evidence though. For the evidence to ‘fit’, it was necessary to rely on the idea of surreptitious cannibalism. Lindenbaum explains that the first Australian government patrols reported the practice throughout the entire kuru region during the late nineteen forties (Lindenbaum 1979:19) and by 1951, when the anthropologists, Ronald and Catherine Berndt, worked in the Northern Fore region, they reported that government intervention had ‘put a stop to cannibalism’. Lindenbaum suggests however that cannibalism ‘was still practiced surreptitiously farther afield’, particularly in South Fore where the largest number of deaths were occurring. According to Lindenbaum, cannibalism did not stop in South Fore until the road was built from Okapa - in the centre of the entire kuru region - to Purosa in the south.\(^{14}\) Arens (1990 & 1997) highlights the inconsistency in the narratives related to the time when cannibalism is said to have stopped in the area. Another possible interpretation is that the Berndt’s and Lindenbaum worked in different areas; the former worked in the North Fore, while the latter worked in the south, thus the different findings.\(^{15}\) But even if Lindenbaum’s account of cannibalism is correct it could not have accounted for all of the cases of kuru that occurred when the following

\(^{14}\) This information came from an elderly man, who is reported to have told Lindenbaum in 1962 that when the government told the people to stop being cannibals the message went unheeded and the practice continued. The man is reported to have said, “We hid and ate people still. Then the tuluaits [government appointed local leaders] and tutuls [their appointed assistants] tried to stop us, but we hid from them too” (italics in original Lindenbaum 1979:19). Lindenbaum goes on to describe in detail how the bodies were prepared ready for human consumption and notes that not all dead were eaten. People who had died of dysentery or leprosy were not seen as suitable for this purpose. Kuru victims, apparently, although sick, were eaten as they died quite suddenly. In Lindenbaum’s words, ‘... the layer of fat on those who died rapidly heightening the resemblance of human flesh to pork, the most favoured protein’ (p. 20).

\(^{15}\) See appendix 1b for a general map of New Guinea and the Eastern Highlands region. Appendix 1c shows Okapa. The Fore region surrounds the Okapa patrol post. North and South Fore were names of census divisions defined by the Administration. The areas immediately north of the Okapa patrol post were deemed Nth Fore, while those south of the patrol post were deemed South Fore. In 1951 and 1952, R. and C. Berndt worked in North Fore on field work auspiced by the Department of Anthropology, University of Sydney, and Catherine Berndt’s work was supported by an Ohio State Fellowship from the International Federation of University Women and the Research Committee of the Uni. of Sydney (Berndt 1952, Berndt 1958). R. Glasse and S. Lindenbaum worked in the Southern Fore area between 1961 and 1962. This work was auspiced by the University of Adelaide Department of Genetics under the terms of a Rockefeller grant, and the PNG Department of Public Health (Glasse & Lindenbaum 1971 in Berndt, R. & Lawrence, P).

In relation to the population of the area affected by kuru, in 1971, Glasse and Lindenbaum said that the population of the Fore region was around 13000, with around 7000 living in South Fore. The census figures mentioned in a Patrol Report in 1954/55 is given as 22000, with Okapa being in the centre of the region (Letter dated July 1955 from West to District Commissioner, Goroka. Report 14 of 1954/5 Microfish 18 of 102. Menzies Library, Canberra). In the mid 1960s (no date Burnet Papers Series 10/7) Glasse gives the figure of 33,000 for the population of the Okapa sub district. I have found it difficult to establish exactly how large the population was at a given time as each paper seems to be speaking about a slightly different area.
is taken into consideration.

Gajdusek remarked at the Washington meeting on *Slow, Latent and Temperate Viruses* held in 1964, that

We looked for antibrain antibody early in the disease in sera from Kuru victims, by many types of reactions, but found nothing. We began to doubt the importance of cannibalism, too, when we found cases of Kuru in children who had been reared on the police station from birth and had never been near a cannibalized dead body. It is possible that they went off surreptitiously of an evening and consumed some flesh, but we doubted this very much, and thus we were forced to say that if cannibalism had anything to do with the disease it could only have introduced hypersensitivity in some individuals one generation back which was then transmitted vertically to offspring from parents (Gajdusek in discussion following the presentation of a paper by Alpers in Gajdusek, Gibbs & Alpers 1965: 81-82).

Gajdusek went on to say that

Certainly, cannibalism has never been omitted from consideration and has been thoroughly studied by the two anthropologists Robert and Shirley Glasse [Lindenbaum], but we do not believe it is the answer to Kuru; the suggestion is more in the nature of an embellishment to other etiological hypotheses.

This seems to suggest that at least as far as some of the leading scientists in the field were concerned during the mid nineteen sixties, cannibalism was little more than embellishment to a very complicated story that involved a scrapie-like agent, regardless of the work of anthropologists.

Since the 1960s there has been differing views on the relationship between kuru and cannibalism. What happened over time was a shift from the view that it was necessary to eat bodies to a view that the disease could have been transmitted during the cannibal ritual through the agent getting into mucosa and conjunctiva. In 1979, as Arens has pointed out, Gajdusek wrote, “even today, we have no evidence that eating the bodies caused the spread” of the disease (Arens 1998: 52 quoting Gajdusek 1979:28). In 1984, a cause and effect relationship between cannibalism and kuru was written about by Klitzman, Alpers and Gajdusek (cited by Arens 1998:52). In 1985, Gajdusek wrote that the disease has been transmitted by ‘the contamination of close
kinsmen with a mourning family group by the opening of the skull of dead victims in a rite of cannibalism...'. This is said to have occurred following autopsy, when unwashed hands were able to scratch skin thereby producing ‘hundreds of intradermal and deeper inoculations’ (Gajdusek 1985:1531 & 1547). As he also points out however, this does not explain the origin of the disease. Questions then, still remain.

Turning to the issue of sorcery, which the indigenous people believed to be the cause of kuru, I want to highlight here only how sorcery is tied up in the meaning of the word Fore. This might not matter were it not for the fact that Fore is also the name of the area where kuru occurred.

Fore is an ambiguous term. In 1958, Ronald Berndt remarked that it was ‘not easy to locate the actual region noted as Fore’ (1958:15). To the Administration and the people of the Kamano region at the Kainantu patrol base, Fore was ‘a vague term covering a relatively wide area embracing Usurufa, some Jate and the “true” southern Fore’. In the Goroka region, Fore was used in reference to ‘people living several days walking distance to the south’, however even in this particular area the name apparently covered more than one language.

As Glasse & Lindenbaum (1971:379) have pointed out, as well as being the name given by the administration to the kuru region, the term Fore also referred to the “source” people and products for making sorcery\(^\text{16}\). John Colman’s Patrol Officer Report written in 1955, captured this meaning in the following terms:

The term FORE to the Henganofi and Kainantu natives has much the same meaning as the term BOMAI has to the Chimbu people - both meaning the area or people to the South. It generally refers to people or an area, of which little is known but is known to exist. The term does not mean an actual direction on the compass but it so happens that all these little known areas lie generally South. The Henganofi and Kainantu people believe that if the tools, method or ingredients for sorcery are required they can be obtained from the FORE. A man absent from his village for any considerable time without reason is believed to have gone to the FORE to make sorcery. This illustrates how loosely the term is applied - it does not mean that the man, in order to

\(^{16}\) Fore also refers to the hot, low-lying regions in contrast with the cooler, higher areas of the region.
make sorcery, has come to this area known linguistically as FORE (emphasis in original p 1)\textsuperscript{17}.

This indicates that while the idea of sorcery went hand in hand with the name Fore - meaning a general area south of Kainantu, the Fore area in which kuru occurred was not necessarily the same area. The distinction seems to have been lost in later stories told about the people of the Fore linguistic area who suffered from kuru. Instead, the people of the Fore linguistic area have been stigmatised as sorcerers. One example found in the popular literature will highlight this. Mick Anglo wrote:

In the ‘land of the Kuru’ ... the Foré, ... detested by their neighbours in the Eastern Highlands as sorcerers and killers who ate their own dead, were discovered. Half of the Foré women, and one tenth of the men were suffering from an incurable virus disease, resembling Parkinson’s disease, never encountered anywhere else in the world. Until recently, it was thought that the disease, Kuru, was transmitted through the Foré custom of eating the brains of their dead relatives (Anglo 1979: 71).

Anglo’s account is found in a book with the title *Man eats man: the story of cannibalism*. It shows the effect being associated with sorcery had and how far derogatory images have spread about the people living in the Fore linguistic region. What is interesting about the account is that Anglo seems to imply, through his use of the phrase ‘until recently’, that he did not believe that cannibalism was any longer considered to be the answer to kuru.

Such negative stereotypes as those that have evolved of the people who suffered from kuru have not been applied to all groups living in New Guinea. James B. Watson has remarked on the generally positive stereotypes that have arisen about people living in Mount Hagen and Chimbu compared to others (Watson 1964:7). Watson also noted that a colleague remarked to him that it is easy to gain the impression from reading New Guinea anthropological accounts that one group are sorcerers and cannibals,

\textsuperscript{17} John Colman, Patrol Officer, Report Kainantu Sub-division of South Fore Census Area, Kainantu 14 of 1954/55. Colman patrolled the South Fore area from the 14\textsuperscript{th} to the 18\textsuperscript{th} of May 1955 and from the 23\textsuperscript{rd} of May to the 10\textsuperscript{th} of June 1955. The patrol was made for the purpose of carrying out ‘routine administration and consolidation where necessary’, conducting a medical check by the native medical assistant (NMA), ‘tribalisation work’, and the capture of an escaped prisoner. This was the first routine administration patrol to the South Fore area since the initial patrol carried out in 1953, according to Colman. An earlier report of the South Fore conducted by MacArthur in 1954 shows that the Fore area had been divided into north and south regions for census purposes in 1953 (see MacArthur report October 1954/55 ‘General Summary’ page 8, Microfilm 8 of 102).
another is devoted to land tenure, while the primary concern of another group is related to something else. The colleague suggested that this is perhaps a reflection of the anthropologist’s interests rather than the native custom (Watson 1964:14). These comments need to be considered when reflecting on how this might have affected the way that kuru has been written about. Kuru did not just happen anywhere, but rather happened in a place whose label - Fore - also happened to be the name given to a general area associated with sorcery.

Kuru and “Nakuruitis”

During the search for meanings of kuru I was surprised to find the term nakuruitis used in connection with a disease of animals, given that kuru is regarded as a human disease. L. Dudley Stamp, Professor of Social Geography at the University of London, mentioned in one brief paragraph in chapter eight of his book entitled Africa: A study in tropical development (1953), that there were parts of Kenya where the ‘mysterious disease “nakuruitis” swept away the settler’s cattle and sheep’. The meaning of the term is unclear, but nakuru possibly relates to the name of a town north of Nairobi. There is also a Lake Nakuru18. In medical terms, itis is a suffix used to refer to inflammation. Exactly when this disease occurred Stamp does not say, but the disease was said to be due to a deficiency of the trace element cobalt. On a speculative level, given the association between kuru and scrapie following Hadlow (1959), and knowing that scrapie has been transmitted through louping ill vaccine (Zlotnik 1965), it is worthwhile being open to the idea that reasons other than trace element deficiency might have been responsible for nakuruitis. Further research on the cause of this disease might provide additional information. The link, however tenuous, between kuru and nakuruitis is that the information provided in chapter eight of Stamp’s book is based on unpublished material supplied by the School of Hygiene and Tropical Medicine (University of London), the Wellcome Museum of Medical Science and the Institute of Medical Research, Johannesburg. Given that an exhibition on kuru was held at the Wellcome Medical Museum in 1959, nakuruitis seem worthy of further research.

18 Thanks to Professor Nelson for this information.
On a more certain footing, it is clear that kuru had as devastating an effect on the people of New Guinea as nakuruitis had on the settler’s sheep and cattle in Kenya, when the following account is considered. In 1957, Jack Baker’s Patrol Officer report indicated that there had been 45 deaths from the disease in the region since the last census. The figures for the census division of the North Fore for the previous five years showed that there had been ‘approx. 200 deaths from KURU’. In the total area known to be affected - the Fore linguistic group, and the sections of Kimi and Keigana bordering the Fore - there had been ‘over 600 deaths’ during the same period\textsuperscript{19}. This suggests that between 1952 and 1957 more than 600 deaths had occurred from kuru. While many reports have been written about the prevalence of the disease, this number does not seem to have been incorporated as the reports begin only with the figure for 1957\textsuperscript{20}. Given the number of deaths that occurred in the five years prior to this there is no doubt that kuru had devastating effects on the people of the Fore region.

This survey has tried to show how, where, and when, the terms ‘prion’ and ‘kuru’ are used within scientific, anthropological and popular accounts. When prion diseases are discussed, the analogy of industrial accidents (EDT) and the metaphor of Ice 9 are provided to help understand the disease. The metaphor of Prion Bay also serves to impress upon us the complexity of prion research, and Prion turtur reminds us of the need to be open to the incorporation of new information.

\textsuperscript{19} Okapa Patrol Report 6 1957:6, also known as Kainantu Report 7 1956-57 held at the Menzies Library, ANU, Canberra and available on microfilm 24 of 102.

\textsuperscript{20} Alpers (1965) provides an excellent analysis of the figures from 1957.
Chapter 2

Kuru and X Disease

Many of the written accounts of kuru begin in early 1957 with Dr D. Carleton Gajdusek, an American scientist, arriving in the Fore region. Gajdusek is reported as having been making his way back to the United States following a stay with Sir MacFarlane Burnet at the Walter and Eliza Hall Institute for Medical Research, in Melbourne, Australia. The narrative is one of serendipity, a new disease investigated by a qualified person who happened to be in the right place at the right time. Here, I want to briefly describe the narrative and then go on to review what is known about X disease in order to highlight the relevance of X disease for the investigation of kuru.

One reason for exploring the pre-history of kuru is the juxtaposition of the two diseases in the title of Series 10 of the Burnet Papers. The title, ‘X Disease, Kuru and Gajdusek files’, suggests a relationship. Folio 10/1 focuses our attention on the relationship between X disease, louping ill virus and the possibility of post-vaccinal encephalitis. When X disease reappeared in 1951 in the Murray Valley in Victoria, Australia, it was renamed Murray Valley encephalitis (MVE). The relevance of MVE research lies in the fact that Dr S. G. Anderson, an Australian investigator of MVE, was scheduled to study kuru, but Gajdusek began the work along with Dr Vincent Zigas, who was a European emigré medical officer working for the Territory Administration. The next chapter deals more specifically with events during the nineteen fifties. Here I want to begin with a few examples of the serendipity narrative.

Jennifer Cooke, in Cannibals, Cows & the CJD Catastrophe (1998), writes that ‘Gajdusek had been on his way back to an undefined role within the NIH in America, and had stopped in PNG to further his pilot studies on child growth. He also planned to visit Sir Mac’s son, Ian.... But soon after he landed in Port Moresby, a courtesy meeting with a public health official set Gajdusek on the trail of a strange “new” disease in the Highlands’. And it was from this point that ‘he decided to detour via Kainantu, north-east of the kuru region for a closer look’ (Cooke 1998: 5). Richard Rhodes tells much the same story in his book Deadly Feasts. Here, Gajdusek is
reported to have ‘stopped off in New Guinea in March 1957 on his way home from research work in Australia, [where] he expected to spend a few months exploring what he called “child growth and development in primitive cultures”’ (Rhodes 1997: 27). June Goodfield’s account of this event in her chapter ‘Kuru Mystery’, is similar, although written from the perspective of a medical historian rather than a journalist. Goodfield writes that following a ‘two-year period, as a visiting investigator’ at the Walter and Eliza Hall Institute, and following a prior visit to West New Guinea in 1956, Gajdusek ‘wanted to pass through the country again on his way back to Boston’. The purpose of the visit is also described here as a wish to ‘visit some primitive cultures’, as Gajdusek ‘had planned a program devoted to child growth and development, and disease patterns in primitive cultures, to be centred at the Children’s Medical Center at Harvard’. To achieve this, ‘he would just move through Papua New Guinea once again with a patrol officer, Ian Burnet, the son of Sir MacFarlane Burnet, who was opening a new uncontrolled area in the highlands, and then would hasten home’ (Goodfield 1985:6-7). The common element in all of these versions of the story is that Gajdusek did not go to New Guinea in 1957 with the intention of studying kuru but rather to begin a pilot study of child growth and development before heading home.

It is not surprising that these accounts have been written as one of the published letters notes that Gajdusek stumbled into the problem of kuru (Gajdusek 1975:174). He informed his Australian colleagues, Drs Burnet, Anderson and Wood at the Hall Institute in Melbourne, of his whereabouts, in a letter he wrote on March 13th 1957; ‘I arrived in Port Moresby and started to plan out sites for potential child study’. Gajdusek went on to explain, that while speaking with the newly appointed Director of Public Health in Port Moresby, he ‘mentioned the possibility of seeing Ian Burnet at Lufa’. This occurred ‘just at the time when Dr. Scragg ... was dealing with correspondence on kuru, the disease’ about which Anderson and the Department’ had been corresponding at length. The letter goes on to describe what Scragg told Gajdusek about the disease:

He told me the story, and I soon got all details of the anthropological study of the region and kuru sorcery from the State anthropologist, Julius, and agreed to have a look at the Okapa area as well as Lufa as possible areas for long-term child study of the sort I am interested in as well as looking at kuru cases-
especially those located in children .... The more I read of the literature and correspondence, the more obvious was it that I had no intention of stepping into your project (Farquhar & Gajdusek 1981:5-6).

The contents of this letter tend to indicate that Gajdusek did not have previous knowledge of kuru prior to entering the region, and also suggest that ‘the project’ was Australia’s. A letter written by Dr John Gunther in late February¹ suggests likewise. Gunther wrote:

I understand Gajdusek has gone to the highlands. Unfortunately I did not see him in Port Moresby. No doubt, though, he will be hot on the trail if he gets the opportunity. It is particularly interesting as your son is at Lufa which, of course, is next door to our problem (Farquhar & Gajdusek 1981:3).

This letter, makes it clear that the problem that kuru presented was considered to be ‘our’ problem, an Australian problem.

In the letter he wrote to his Australian colleagues, Gajdusek expressed disappointment that he had ‘heard no word about kuru and its interesting and intriguing problems’ while at the Institute (Farquhar & Gajdusek 1981:6). Nevertheless, he was willing to put his disappointment aside and agreed to look into the problem in what he described as ‘an attempt to be of some help and service’ to his Australian colleagues and the Administration.

Vincent Zigas commented on Gajdusek’s arrival and foreknowledge of kuru in Laughing Death: The Untold Story of Kuru (1990:227). He was puzzled why Gajdusek told him that Scragg had been the first to tell him about the disease. Zigas found this unusual and wondered why the disease had not been discussed with Gajdusek while he was working at Walter and Eliza Hall Institute during 1956. This suggests that either a degree of secrecy on the part of those at the institute prevailed, or that Gajdusek knew about kuru prior to his arrival.

¹ It is not possible to assess the exact date of this letter as the date has been inserted in square brackets as an approximation of an undated letter.
In 1977, Gunther was a little more frank about the events of twenty years past, when he wrote the following in a draft paper entitled, *Australia, Kuru, and a Nobel Prize* (Gunther 1977):

The situation was that Gajdusek knew about kuru, he knew that the Hall Institute had been asked to study it, and in the first half of March 1957 he went into the Fore territory without much ado (Gunther 1977:10).

Gunther then continued by writing, “Thank God he did”.

My purpose here is to show that whereas the accepted kuru story maintains that it was serendipity that connected Gajdusek with the investigation of kuru, it would appear that this might not have been the case. Hank Nelson captures the confusion in the narratives on kuru when he writes, Gajdusek ‘had heard about kuru in Melbourne – for Anderson had been contacted and the first brain and blood samples had arrived before he left’ (Nelson 1996:191). Further support for the idea that Gajdusek knew about kuru before arriving comes from the recollections of William Tomasetti, a member of the New Guinea Administration at the time of the investigation, whom I interviewed.3

Whilst discussing the life of administration officials in 1957, Tomasetti remarked that the study presented extra workload for patrol officers and the administration. This meant that ‘people like Gajdusek and Sir MacFarlane Burnet who were more or less internationally well known figures in the field, in those fields of medical science, … couldn’t be ignored’. Furthermore,

They [Burnet and Gajdusek] were doing the same work and ... Carl came to Melbourne, ostensibly, and probably eager to see the MacFarlane Burnet Institute. Whilst he was there, and this was I think in either late 56 or mid 56 ... Vincent Zigas … realised that there was something very very interesting there, and he’d been sending material I think down to Melbourne. It may have

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2 Nelson highlights the fact that ‘in the most simplistic reports the scientists seem to arrive suddenly among villagers with no previous contact from the outside world, and the practical and moral implications of the research are ignored’ (Nelson 1996:178).

3 Interviews for the research conducted September 21st 1999 and February 2000. Tomasetti served the Administration from 1945-1978 in a number of positions. In July 1957 he was Acting District Officer of the Eastern Highlands District, at the District Headquarters, Goroka (Letter from Tomasetti to The Director of Native Affairs, Port Moresby dated July 31st 1957. Ref. 30-3-1/10 (328). Microfilm 24 of 102 held at Menzies Library, ANU, Canberra).
gone direct, or it may have gone direct through Public Health in the Headquarters in Port Moresby. Anyway, ... Gajdusek saw that stuff, so he came to New Guinea, ostensibly he said, he was interested in the pattern of child development, the disease pattern, but in fact, he wanted to, he probably did some work on that, but also he wanted to get into the kuru scene, which he did, and indeed became the central figure in it for some time⁴.

Tomasetti’s recollections suggest not only that Gajdusek knew about kuru but also that he and Burnet worked together on the research. This is inconsistent with many accounts that emphasise the animosity between the two men. On checking my interpretation of Tomasetti’s account with him, he replied that it was his understanding that the two men worked together. While this recollection is inconsistent with many of the narratives that highlight the conflict aspects of the relationship, it is consistent with Gunther’s opinion that Gajdusek knew about kuru before going to New Guinea. Gunther’s paper and these recollections put into question the serendipity narrative. The question is why did Gajdusek express disappointment at not being told about kuru while at the Institute in Melbourne, when it seems that he knew about the disease, and why then, did he express a willingness to help the administration in spite of his disappointment? Because of the inconsistency in accounts, it seems worthwhile drawing attention to the pre-history of kuru to provide a context that involves research on acute diseases like MVE and X disease.

To understand X disease we need to look at a mysterious group of diseases that occurred in Australia beginning in 1917. Over the next few decades the mysterious disease continued to appear at intervals and underwent a number of name changes. During the nineteen fifties when it occurred in the Murray Valley in Victoria, the disease was renamed MVE and research at this time suggested that MVE and X disease were the same disease⁵. Many causes of the disease were proposed over the decades. At first, an aberrant form of poliomyelitis virus was thought responsible. By the nineteen thirties, louping ill virus, which primarily affects sheep, was suspected. By the fifth decade when MVE became manifest louping ill virus lost favour as causative agent and the disease was likened to, although found to be not identical to Japanese B encephalitis.

⁴ Interview conducted with William Tomasetti September 1999
⁵ More recently the same disease is referred to as Australian encephalitis.
Here I want to track the line of research that began with the mystery diseases of 1917-1918, through X disease to MVE with the objective being to explore this relationship and thereby suggest how it might have a bearing on how we look at the study of kuru. As already noted the relevance of this line of research for the kuru investigation is that plans had been made for Dr Gray Anderson, who worked on Murray Valley encephalitis, to study kuru but for some reason, these plans went awry and Gajdusek began the work. Another point of relevance relates to the similarity between the pathology and symptomatology of X disease and kuru. This is not to suggest that the two diseases are identical but there are enough similarities to take into account. Another reason for examining this earlier research is to show how the level of anxiety caused by X disease during the first two decades of the twentieth century was very similar to the anxiety produced by prion diseases throughout the last two. Similar anxiety was expressed also in the intervening period when kuru became particularly problematic in 1957. A further reason relates to the unusual nature of X disease and its causative agent. That poliomyelitis virus was at first thought implicated is significant in light of problems associated with polio vaccine encountered at the time of the kuru investigation. Chapter four examines these problems in detail. Polio vaccine also featured prominently in the BSE story with the withdrawal of batches of the vaccine by the British government in October 2000, a decision taken ostensibly as a precautionary measure to prevent cases of the new variant form of CJD (nvCJD). Whether precautionary or not, this serves to highlight the potential of transmitting prion disease through iatrogenic means. Chapters five, six and seven deal with these issues, but in this chapter I want to highlight only the relationship between poliomyelitis virus, loping ill virus, X disease and post-vaccinal encephalitis. And so it is time to turn back the clock to explore this other group of “mysterious diseases” that became known as X disease.

**X disease: An unusual syndrome**

In May 1917 Dr W. F. Litchfield brought the case of what was then described as a “mysterious disease” to the attention of the British Medical Association in Australia and pointed out that ‘the symptoms resembled to some extent those of meningitis’. At the meeting Dr Robert Day noted that ‘these mysterious cases were still occurring in
the neighbourhood of Bourke in considerable numbers’. Furthermore, adults had recently begun to become infected (BMA News 1917:384-385). At the time, it was thought that the organism may have been ‘an aberrant form of pneumococcus’ (1917:384). In children the disease manifested with a ‘sudden fever and convulsions’ with ‘no other symptoms’, and ‘death followed in a comparatively short time’. Understandably, the disease caused ‘much uneasiness’ in the region. The President of the Sydney Association, Dr R. Gordon Craig, who Chaired the meeting, noted that an ‘official investigation was being carried out by the officers of the Board of Health’. Craig ‘had no doubt but that the nature of the disease would be ascertained’.

A short time later, another report appeared, when Burnell, the House Surgeon of the Broken Hill Hospital and a junior member of the medical profession, wrote a paper about ‘The Broken Hill Epidemic’. Burnell brought his account of the disease to his colleagues at a meeting of the South Australian Branch of the British Medical Association, and he did so with some trepidation for the aetiology of the disease was ‘very obscure’. But with a case mortality of 80% Burnell believed that the problem was in need of a full discussion in order that abler minds than his could fathom the problem and offer valuable suggestions. Burnell carried out a number of tests, but as he explained to the meeting, the night after he had inoculated a rabbit intraperitoneally with Bacillus No 1, ‘it was stolen’ (Burnell 1917:159). Although thwarted in his endeavour to transmit the agent, Burnell was able to conclude that ‘we have a symptom complex which does not conform to any of the ordinary diseases’ (Burnell 1917:160).

For inspiration and insight into the disease, Burnell drew on the work of J. P. McGowan. McGowan began writing about scrapie, a disease of sheep similar to kuru,

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6 The patient to whom Litchfield referred, a child aged two years, had been ‘taken ill suddenly and within three hours was in convulsions, with a temperature of 103°C’ (BMA News 1917:384). Two children who had lived in the same house had also died of the disease two weeks previously. The new patient was in hospital for ten days by which time the temperature had subsided. On the fourth day antimeningococcal serum was given, presumably as treatment for suspected meningococcal infection. The cerebro-spinal fluid taken from the patient was clear which means that there was no detectable infection within the central nervous system, although the CSF was under increased pressure. A blood specimen taken produced a diplococcus (gram positive) which was ‘indistinguishable from the meningococcus’.

7 This paper was published in the August 25th edition of the Medical Journal of Australia (Volume 11 No 8 pp 157-161).

8 Bacillus No. 1 had been found in ten of the thirteen cases studied.
in 1914. One aspect of McGowan’s work particularly interested Burnell, notably, the difficulty involved in researching the agent responsible for this mystery disease. This was because the ‘organism’ was polymorphic. Burnell, citing McGowan, characterised the problem in the following terms:

We have observed variations of biological activity among different strains of the same organism, derived from different sources, whether it be that of chicken cholera, swine plague, or sheep septicaemia. [...] Not only does this group ... vary greatly with regard to its biological activity, but it also varies greatly in its power of altering its shape (McGowan quoted in Burnell 1917:160).

The polymorphism associated with the agent with which Burnell worked was ‘more marked than is the case with any other group of organisms’. This is a characteristic of the agent/s that are thought to be responsible for prion diseases like scrapie, kuru, CJD, BSE and nvCJD. The ability of the agents to appear in many forms must have been most disconcerting for the scientists, doctors, and veterinary specialists working during the early decades, and no less disconcerting than for the scientists working on prion research in the subsequent decades. Because Burnell drew on McGowan’s work, there is a direct relationship between X disease and a prion disease – scrapie dating back to the beginning of the twentieth century. This also shows that disciplinary knowledge was shared between animal and human health. At the end of the twentieth century, this crossover seems to have been forgotten.

A recent example of this came to light at an Inquiry held in England during the 1990s into iatrogenic CJD. A learned Professor of human medicine scoffed at the idea that an ‘animal doctor’ might have something to contribute to the subject of prion disease.

The mysterious diseases of the early decades of the twentieth century and prion diseases in the subsequent decades have another common feature in that in both cases

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9 McGowan wrote a paper in 1922 entitled ‘Scrapie in Sheep’ which was published in the Scottish Journal of Agriculture. Another article, ‘A further contribution to the subject of scrapie’, was published in 1925. In 1917 Burnell referred to McGowan as representing the Royal College of Physicians. By 1922, McGowan was working at The Rowett Institute, Aberdeen.

10 Interview with Jennifer Cooke September 1999, who attended many of the hearings of the Inquiry held in England into the use of human pituitary hormones that resulted in a number of deaths. A similar Inquiry was held in Australia in 1994 (Allars 1994).
the agent/s can be transmitted experimentally to animals. The early studies showed that the agent responsible for the mysterious diseases was capable of infecting monkeys. One commentator remarked that ‘at present we cannot say more than that he [J. B. Cleland] has succeeded in producing a disease of an encephalitis type in monkeys by the intracerebral injections of emulsion of the spinal chord of a person dead of the disease’ (‘The Broken Hill Epidemic’ 1918:195). This shows that the idea that an agent could affect people as well as animals, in this case monkeys, was readily accepted at the end of the First World War. And then as now, knowledge that the same agent could infect animals and humans, if only experimentally caused concern. The report in 1918 tried to dampen speculation about the causative agent by saying that ‘[n]o useful purpose would be served by a speculation at the present juncture as to whether or not this disease is caused by Flexner’s organism of anterior poliomyelitis’.

The perceived link between the mysterious diseases of 1917 and 1918 and acute anterior poliomyelitis needs to be given special attention because of the parallels in terms of the concern and anxiety produced then and similar concerns during the nineteen fifties when polio vaccine and kuru were being studied. The situation was repeated again during the nineteen eighties and nineties when CJD, BSE and variant CJD were investigated in greater depth. Here I want to simply describe what lay at the base of the concern: the uncertainty surrounding the identification of the causative agent of X disease.

**Poliomyelitis virus as a possible cause of X disease**

During 1917 and all throughout 1918 there were many articles published about the possible cause of the disease. Burnell offered thanks to Dr J. B. Cleland for his assistance in being able to independently isolate Bacillus No 1. from one of Burnell’s cases. Cleland had particular expertise in ‘the more technical part of the bacteriological investigation’, and finally was successful in “placing” the organism (Burnell 1917: 160). This meant that the causative agent was loosely placed in the category of agents that included poliomyelitis virus. Nevertheless, this did not stop speculation about its nature.
In June 1917, Cleland and Dr Burton Bradley co-authored a short letter in response to one published in the *Medical Journal of Australia* by Dr. Anton Breinl in the May 26th edition\(^1\). Cleland and Burton Bradley remarked on the similarities between the mysterious disease occurring in Townsville described by Breinl and the disease occurring in New South Wales. These authors suggested a similarity between acute anterior poliomyelitis and the mystery disease and noted work in progress along these lines. They noted that

> It is quite possible that the disease in question is not acute anterior poliomyelitis, as generally understood, but that it may be due to some virus allied to that of acute anterior poliomyelitis producing a disease which has hitherto not been differentiated from the latter (Cleland & Bradley 1917: 499).

This highlights the difficulty in categorising the agent.

In March the following year, Breinl published a paper in the *Medical Journal of Australia* entitled ‘Clinical, pathological and experimental observations on the “mysterious disease”, thought to be a clinically aberrant form of acute poliomyelitis’\(^2\). The article concluded by saying that

> ... the cases of “mysterious disease” occurring in Queensland and New South Wales are caused by the same virus as acute poliomyelitis (Breinl 1918: 234\(^3\)).

There was however one important difference. The disease differed pathologically from typical acute poliomyelitis because of ‘extensive involvement of the brain’ (Breinl 1918: 234).

The mysterious disease, which by this time had been given the vague name of X disease, was discussed again at a meeting of the Victorian Branch of the British Medical Association on March 6th 1918. A Dr Robertson, who attended the meeting, ‘referred to cases of polio-encephalitis at Shepparton, and read a letter he had received

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\(^1\) Dr Anton Breinl was the Director of the first medical research centre in Australia founded in Townsville in 1911, which was named the Australian Institute of Tropical Medicine (Denoon 1989: 21-22).

\(^2\) Volume 1 No 11 pp 209-213.

\(^3\) The article was split between two editions, number eleven, noted above, and Volume 1 No 12 pp 229-234.
from Dr. Cleland, of New South Wales\textsuperscript{14}, in reply to enquiries he had made in reference to the disease ‘X’. In Robertson's opinion, ‘these cases of ‘X’ were undoubtedly polio-encephalitis’ (‘Scientific’ 1918:294). Only one month earlier however another report had tried to dampen speculation about the causative agent, as previously mentioned, by saying that ‘[n]o useful purpose would be served by a speculation at the present juncture as to whether or not this disease is caused by Flexner’s organism of anterior poliomyelitis’ (‘The Broken Hill Epidemic’ 1918).

It would seem that there was just as great an attempt to dampen speculation about X disease, then, as there was much later in regard to the cause of BSE. And then as now the anxiety reducing measure did not achieve its goal. The cause of X disease continued to be a subject of debate throughout the nineteen twenties and thirties, and it is to this period that I now turn to show how loping ill virus was proposed as an alternative causal agent. This does not negate earlier comments about polio vaccine and its possible implications for the transmission of prion diseases but rather serves to highlight the complexity of the research that existed in both timeframes.

\textbf{X disease, loping ill virus and the possibility of post-vaccinal encephalitis}

During the nineteen thirties, Burnet worked with Jean Macnamara, who worked on polio convalescent serum as a treatment for polio sufferers. The complex story of the perceived link between acute anterior poliomyelitis and X disease has been described above. Now, the aim is to explore events between October 1933 and 1935 in order to establish a link between X disease and loping ill and explore the iatrogenic relationship that was perceived to exist.

It is not clear when Burnet’s interest in X disease began, but in 1932 he went to work at the National Institute for Medical Research in London on a special fellowship to study virus diseases of animals and man. While there, he shared a laboratory with the veterinarian Ian Galloway, who was working on foot and mouth disease of cattle (Burnet 1968:88-90). Having returned from the Institute, between 1934 and 1935,

\textsuperscript{14} It is not clear whether the Cleland to whom Robertson referred was the same Cleland to whom Burnell referred.
Burnet corresponded at length about X disease, louping ill, poliomyelitis and foot and mouth virus transmission with researchers in London. Series 10/1 of the Burnet Papers, entitled ‘X-Disease – Correspondence and Clinical Records’, begins the series of files that Burnet kept together with his papers on kuru\textsuperscript{15}. X disease therefore seems to be an important component in understanding kuru. And the same is true for louping ill in that by 1934 Burnet was suggesting that it might be the causative agent of X disease. A few years later, louping ill vaccine research transmitted scrapie to sheep. So let us again venture back, this time to the nineteen thirties, in order to review what was known about louping ill virus with respect to post vaccinal encephalitis and show how the virus came to be in Australia by 1934.

In May 1934, Burnet published a paper entitled ‘Louping Ill virus as a possible cause of the X diseases epidemic of 1917-1918’, in which he cited the work of Drs Greig and Gordon, two prominent researchers in the field of louping ill vaccine research. Burnet published his paper in *Medical Journal of Australia* in the hope that it ‘... may stimulate the interest of country medical practitioners and veterinary surgeons in the possibility of louping ill being present in Australia’ (Burnet 1934:681). The paper was a plea to researchers from human and veterinary disciplines to be on the look out for louping ill in Australia, which is significant because it indicates that both would be reading the journal and demonstrates a sharing of knowledge between disciplines at this time. What is perhaps more significant though, is that it is inconsistent with a suggestion made by J. H. L. Cumpston the Director General of Health, that louping ill virus did not exist in Australia. Cumpston made the suggestion in a letter he wrote the previous year (1933), in which he advised Burnet not to bring the louping ill virus into Australia without further advice, something for which Burnet had requested permission. So whereas louping ill was believed to be non-existent in Australia in 1933, by 1934 Burnet was postulating that it was the possible cause of X disease.

While in London at the NIMR in 1933, Burnet requested permission from Cumpston

\textsuperscript{15} Burnet’s papers were archived as part of the Australian Science Archives Project, now AUSTEHC, and have been maintained in the order in which they were accessed (Gavin McCarthy personal communication). The X disease research precedes Series 10/2 which covers negotiation held in 1956 to visit New Guinea preliminary to the Kuru Project - the subject of the following chapter - which precedes the kuru investigation files (Series 10/3 – 10/38). Thus the ordering of Burnet’s papers promotes a reading of the material that makes sense of the connections.
to bring a number of viruses into Australia. In October that year, Cumpston replied noting that after consultation with the Acting Director of the Commonwealth Serum Laboratory, it had been decided that there was no objection to importing ‘various bacteriological cultures of dysentery salmonella groups and a series of bacteriophage active against these’. The situation with regard to some other viruses was quite different however. Significantly, Cumpston stressed that in regard to

vaccinia virus, herpes virus and virus of louping-ill, the possibility of introducing the agent - whatever it may be - of post-vaccinal encephalitis must be regarded with some gravity especially in relation to the fact that herpes virus is not above suspicion in this connection (emphasis added).

It was because louping-ill disease did not occur in Australia that it was considered ‘especially undesirable to introduce the virus of this disease’\(^{16}\).

It is clear from the above that in 1933 Burnet and Cumpston were aware that louping ill might be related to post-vaccinal encephalitis. Another researcher, J. R. Perdrau, who also published a paper on the relationship between louping ill and X disease, as had Burnet, published a paper called ‘Histology of Post-Vaccinal encephalitis’ in 1928 (Perdrau 1928)\(^{17}\). This tends to suggest that the links between post vaccinal encephalitis, X disease and louping ill were suspected during this period with respect to human disease.

When Cumpston cautioned against bringing the virus into Australia in 1933, he indicated to Burnet that if he could come up with good enough reason to counter the decision, which, he noted, would ‘limit the investigation which you are contemplating, the matter may well be re-considered’. In the meantime, ‘the decision as above indicated must apply’. However, following its passage from England in 1934, louping ill virus was ‘active on arrival’ and found its way into Burnet’s laboratory.

Details about the movement of the louping ill agent are contained in a letter written by

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\(^{16}\) Letter from Cumpston to Burnet dated October 27\(^{th}\) 1933. BP Series 10/1

\(^{17}\) In 1928 and 1936 Perdrau worked at the National Institute of Medical Research, Hampstead, where Burnet also went to work in 1932 and part of 1934.
J. A. Galloway, who wrote from the Medical Research Council, Hampstead, on July 11th 1934 thanking 'Galloway' for his letter of the 15th May. J. A. in Hampstead, remarked to Galloway in Australia, that he had 'dried some loping ill mouse brains' for him and had 'also sent some infective mouse brains in glycerin (50 percent)'. The specimens were to be sent on the SS *Oronsay* sometime 'around the 19th July'. J. A. requested that Galloway 'arrange for the collection of the package on arrival'. In September 1934, Burnet wrote to J. A. Galloway in London, who responded with a three and a half page letter, written on October 16th. Galloway was 'glad to learn that the virus of loping ill proved active on arrival'.

Was permission received from the Director General of Health to import the virus? Either Burnet had been able to provide an adequate argument to reverse the decision of the Director General of Health and the Commonwealth Serum Laboratory, or the loping ill virus entered Australia without authority.

While in London the previous year, Galloway and Burnet attended a meeting of Comparative Medicine at the Royal Society of Medicine, at which Galloway referred to 'preliminary observations on the possibility of propagating the virus of loping ill in eggs'. When Burnet left England to return to Australia, Galloway was under the impression that he 'had not received permission to work on the virus and decided to continue these investigations with Miss Faulkner' (emphasis added). It is perhaps significant that there is no letter of authorisation to bring loping ill virus into the country in the correspondence that Burnet kept between himself, J. A. Galloway and Cumpston for the period 1933 to 1934.

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18 Letter dated July 11th 1934 from J. A. Galloway to Galloway. BP Series 10/1
19 The letter continues by describing the status of the experiments being carried out at Hampstead. Much of the discussion revolved around Foot and Mouth disease. Unfortunately, the foot and mouth experiment in Duck's eggs was 'a wash out'. Nevertheless, Galloway suggested that his namesake in Australia may be interested in the 'short report on F.M.' enclosed. Filtration of the Poliomyelitis virus was 'nearly finished'. This agent 'appears to be very small and seems F. + M. very close [?]'. J. A. Galloway seemed to be saying here that the size of the polio agent and the foot and mouth agent seemed to be very similar. Other filtration studies were being carried out on 'V.S. filtration - egg passage'. But no results were yet available. The discussion then continued by describing various membranes used for filtration of the agents being tested. 'Elford ... has been unable to get the necessary membranes made viz. 0.13μ - the last batch were 0.136μ which were unsatisfactory as both guinea pig and egg passage virus came through', he reported. J. A. explained however that in his case, 'after 10 passages in eggs (without g.p. [guinea pig] intermediate passage) the virus does not come through a 0.115μ membrane, and one passage of guinea pig material through eggs is sometimes sufficient to give a virus filterable through 0.13μ'.
20 Letter dated October 16th 1934 from J. A. Galloway to Burnet. BP Series 10/1
Another question that arises is, was Cumpston aware of the importation? It seems that he might have been excluded from knowing about this event because there is also no evidence in Burnet’s papers of any reason to reverse Cumpston’s decision. The lack of documentation tends to indicate that Burnet had, with Galloway acting as intermediary, arranged for the louping ill virus to enter Australia without permission from the Director General of Health. There could of course, have been some agreement reached between the time of the meeting on Comparative Pathology and July 19th 1934 when the specimens were to set sail on the SS Oronsay. But judging from Galloway’s comments it would appear that louping ill virus may have entered Australia outside of normal channels\(^\text{21}\). The possible implications of this are difficult to assess, but one of them is that the louping ill experiments discussed in the correspondence might have been more closely associated with W. S. Gordon’s louping ill vaccine research conducted around the same time than has formally been recognised or acknowledged.

One commentator has remarked that Gordon’s ‘... louping-ill accident and its implications for pooled tissue transmission of diseases remained largely unknown by the wider scientific world ... [with] only five (mostly brief) references to the louping-ill accident in veterinary literature between 1940 and 1954’ (Cooke 1998: 30-31). This might be the case, but Burnet, who played a significant part in the kuru story some decades later, was probably aware of Gordon’s work, and therefore probably knew about the transmission of scrapie through an iatrogenic route during the ‘thirties. Even if he was not aware of the vaccine accident in the field of animal disease, he was aware of the possibility of louping ill virus being associated with post-vaccinal encephalitis in relation to human disease from 1933.

There was not only familiarity with the work of Gordon. J. A. Galloway explained to Burnet that he ‘asked Gordon about the horse as regards louping ill’ and there was ‘no evidence either one way or the other’. He also explained that ‘27 consecutive passages in eggs during a period of 180 days’ had been made and that the ‘virus passages through eggs’ were ‘still infective for mice’, and they were ‘now having the egg virus

\(^{21}\) Clearly, other interpretations are possible. For example, correspondence may have taken place directly between Cumpston the Director General of Health, J. A. Galloway in Hampstead, and Galloway in Australia.
tested on sheep’. A test had ‘in fact already [been] made on sheep but unfortunately the test animals were unhealthy and although the virus was recovered from the brain and cord of the sheep neither had shown typical symptoms of louping ill and lesions of pneumonia and pleurisy were found on p.m.’. For this reason, the test was being repeated on ‘healthy sheep’. A few months earlier, in July, J. A. Galloway described these proposed experiments when he wrote to his namesake in Australia saying: “We have carried on Louping Ill in eggs -18 passages and are now getting it tested on sheep -3 months out of mouse. We shall write it up when sheep test is complete.” It would appear that this sheep passage experiment had to be repeated, and there is little doubt that Burnet was being kept informed about the work conducted in London on louping ill transmission.

According to Burnet’s autobiography, he was working with poliomyelitis and louping ill in 1936 (Burnet 1968:118). This means that he was quite possibly aware of the transmission of scrapie through Gordon’s louping ill vaccine research two decades before he became involved in the study of kuru and might have been familiar with scrapie when the investigation of kuru was conducted. While it is not exactly clear why Burnet’s papers on X disease (louping ill) have been kept with the kuru papers, given the predominant theme of the early correspondence it would appear that kuru is being viewed within the framework of iatrogenesis. Alternatively, the arrangement of the papers might simply be because Burnet was aware of Gordon’s work, knew about scrapie, and therefore kept this correspondence with his papers on kuru because scrapie and kuru were later found to be similar diseases; something pointed out by William Hadlow the veterinarian in 1959 (Hadlow 1959). Another scenario might be because X disease and MVE were linked during the nineteen fifties and MVE and kuru occurred in such close proximity. But neither of these possibilities negate the fact that Series 10/1 revolves around X disease, louping ill and post-vaccinal encephalitis and that the research on this earlier group of diseases has been linked to the research on kuru.

22 Letter from Galloway to Burnet dated October 16th 1934. Series 10/1 BP.
23 Letter from Galloway to Galloway dated July 11th 1934. Series 10/1 BP.
In a way, the fact that Burnet might have known about Gordon’s louping ill vaccine experiment that transmitted scrapie to sheep, is not as significant as the fact that he was occupied with the complexity of differentiating between poliomyelitis virus which affected humans, foot and mouth which affected cattle, and louping ill, a disease of sheep. These preoccupations occurred while Gordon’s vaccine was in preparation between 1931 and 1934. The possible iatrogenic link with louping ill virus relative to humans was suggested in 1933. This means that louping ill was suspected as a possible cause of X disease in humans through an iatrogenic route before the results of Gordon’s louping ill vaccine experiment had even been published in the veterinary literature. Members of the medical profession, including Burnet, had their own concerns, albeit about a form of acute encephalitis as opposed to a disease like scrapie which took longer to manifest from the point of exposure.

Before leaving the subject of X disease, I want to highlight the similarities in pathology between it and kuru.

**Comparative pathology: Kuru and X disease**

The kuru story is so often confined to events that occurred in the late nineteen fifties and after as if there is nothing to be gained from looking back in time. I would argue that there is much to be gained because rather than there being a discontinuity between kuru and anything that went before it, there are some interesting continuities. The unusual nature of the agent that was thought responsible for X disease, which had some commonalities with the agents which McGowan the scrapie researcher described at the beginning of the century, is one similarity that has already been flagged. Another, is the anxiety and uncertainty that X disease caused, a situation not unlike the concerns raised during later decades with respect to prion diseases. An additional common link exists through certain aspects of the pathology of X disease and kuru.

It was noted earlier that Breinl in 1918 remarked that X disease differed pathologically from typical acute poliomyelitis because of ‘extensive involvement of the brain’ (Breinl 1918: 234), which typically did not occur in polio. This highlighted
the difficulty in categorising the responsible agent. It is interesting to compare Breinl’s pathology findings with those of kuru because while there were some differences there were also noticeable similarities.

Breinl remarked on the presence of neuronophagia, a process whereby the ganglion cells in the brain alter during the course of the disease changing from ‘the earliest signs of degeneration to complete disappearance’. Many of these cells were ‘surrounded by leucocytes, lymphocytes and large mononuclear cells, possessing a large, irregular nucleus, with a fine chromatin network – a process known as neuronophagia’ (Breinl 1918: 234). Neuronophagia is present in kuru pathology (Klatzo, Gajdusek & Zigas 1959). Klatzo found ‘very spectacular neuronophagia’ of the caudate in the Basil Ganglia region\(^\text{24}\). In 1959, at a Symposium on the encephalitides held in Antwerp, attention was drawn to this sort of lesion in reference to kuru. Webb Haymaker from the Armed Forces Institute of Pathology, Washington, D.C., in reference to the neuronoplagic type of lesion, wrote: ‘It was demonstrated to us not only in the infectious encephalitis, but also in Drs. Klatzo, Gajdusek and Zigas’ kuru’ (Haymaker 1961: 699-701).

Looking to the outward signs of the disease, which represent pathological changes inside the body, there were two other common features of X disease and kuru. The first of these is dysphagia, which means difficulty in swallowing. The second feature is that both diseases are marked by fluctuating symptoms.

When Burnell published a further account of ‘The Broken Hill Epidemic’ in the *Medical Journal of Australia* in April 1918, he pointed out that the features in 1918 differed slightly from the cases he had seen in 1917 but one aspect that remained constant was ‘difficulty in swallowing’ (Burnell 1918:280\(^\text{25}\)). There was confusion over this term in one of the first published accounts of kuru in the scientific literature where dysphagia was reported as dysphasia by mistake\(^\text{26}\). Dysphasia is the inability to

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\(^{24}\) Kaltzo to Smadel in a Memorandum dated August 15\(^\text{th}\) 1957 in Farquhar & Gajdusek 1981:112-113.
\(^{25}\) Burnell notes in his opening remarks of this paper the change of name from “mysterious disease” to “X”: ‘On 25\(^\text{th}\) August, 1917, I published in the *Journal* an account of an epidemic at Broken Hill of what is now called the “X” disease’ (Burnell 1918:278). *MJA* 1918 April 6\(^\text{th}\) pp 278-289

\(^{26}\) Letter from Gajdusek to Robertson dated December 24\(^\text{th}\) 1957 in Gajdusek 1975:377. See Zigas & Gajdusek (1957:748), where dysphasia is used instead of dysphagia.
arrange words in their proper order due to a central lesion (Miller & Keane 1987: 386). Dysphagia can have ‘numerous underlying causes’. The question that arises in this context is whether the dysphagia of kuru and that of X disease were aetiologically related.

Brieni observed that a variety of symptoms tended to fluctuate in X disease. Burnell drew attention to the same phenomenon. For example, ‘nystagmus was present now and again’; ‘neither Babinski’s nor Kernig’s sign was constant’ (Breinl 1918:233). Burnell mentioned that one of the striking features of the symptoms caused by this peculiar agent was their ‘transient nature’ (Burnell 1917:157). ‘In the morning’ he wrote, ‘one may find the patient with marked …[symptoms] … while an hour later he may be lying quietly in bed with none of these symptoms’ (Burnell 1917:157). The same fluctuating phenomenon was observed in kuru in December 1957: ‘the nystagmoid eye jerking can approach true nystagmus one day and later that day have almost disappeared’ (Gajdusek 1975:338). The commentary of Kuru: A Clinical Study (1958), a film made in 1957, noted that strabismus, a disorder involving the eyes and the central nervous system, comes and goes.

As well as the neuronophagia, fluctuating symptoms and dysphagia common to both kuru and X disease, destruction of the Purkinje cells in the brain featured in both diseases as well as in louping ill.

The involvement of the Purkinje cells is a significant feature of kuru pathology. In 1970 Burnet delivered a paper on kuru in Kuala Lumpur in which he remarked on the fact that the pathology in both man and chimps is similar ‘with major involvement of the Purkinje cells of the cerebellum’.

Other commentators have noted that this is one of the marked features of both kuru and scrapie (Beck & Daniels 1969). In 1957, two of the preliminary pathology reports noted the destruction of Purkinje cells in the cerebellum. Igor Klatzo wrote:

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27 The film is held at the Australian National Sound and Film Archive, Canberra, and was made by the Department of Information, Commonwealth Government Film Unit, Australia, MCMLV111.
28 The paper ‘Introduction to Kuala Lumpur talk on Kuru’ is held at the Adolph Basser Library, Canberra, Ref. 98/2/C8
There are numerous “torpedoes” (ballooning of the Purkinje cell axon) and bizarre deformities of Purkinje-cell dendrites (in Farquhar & Gajdusek 1981:113).

Dr E. G. Robertson wrote in summary on his ‘Preliminary (Incomplete) Report on Case 1’:

... regressive changes with degeneration and disappearance of Purkinje and granule cells (in Farquhar & Gajdusek 1981:115).

Both reports indicated that the process did not appear to be inflammatory. Robertson’s report included the opinions of Webb Haymaker and Dr G. J. Greenfield, to whom Robertson had shown the specimens while in America just prior to the report being written.

During 1957, Dr G. J. Greenfield worked as a Visiting Investigator at the National Institute of Nervous Diseases and Blindness in America. His role at this time was to provide details on how to collect kuru brain specimens to best effect. This information was sent to Gajdusek who was conducting the investigation in New Guinea (Farquhar & Gajdusek 1981:306). Greenfield had written many papers and books and was considered an expert in the field of pathology by 1957 when he was involved in the kuru investigation. It is useful to look for a moment at the sort of expertise Greenfield might have brought to the kuru study.

The dominant kuru story suggests that no one had any idea what sort of disease they had in kuru. The association between kuru, scrapie and Creutzfeldt-Jakob disease did not occur until 1959. In 1957, kuru was presented as a ‘new’ disease unlike any other. Greenfield however, might have had some very relevant knowledge about Creutzfeldt-Jakob Disease at this time because he had helped to make a differential diagnosis for Jakob’s syndrome in 1937. Along with MacDonald Critchley, he published a note in the Proceedings of the Royal Society of Medicine, which stated that:

In 1923 Jakob described a case of senile dementia associated with Parkinsonism, and distinguished this condition from arteriosclerotic dementia, which it resembled closely in its clinical features, but not in its pathology (Critchley & Greenfield 1937:94).
The authors describe a 64 year-old Belgian woman who was admitted to the National Hospital, Queens Square, on March 11th 1936 who died eighteen days later. It was found on examination at autopsy that ‘the microscopical picture...like the clinical picture, was that of a combination of senile dementia with paralysis agitans’. The onset of these two conditions within a few months of each other was suggestive, the authors remarked, of a more than chance association (Critchley & Greenfield 1937:94-95). At this time it was unclear whether Jakob’s syndrome should be seen as a distinct clinical entity, ‘or as the association of two diseases’. Grünthal, who had discussed the issue in the literature, was inclined to see the disorder as an association of two diseases. Critchley & Greenfield concluded however that due to the ‘morbid process of senility severely implicating the palato-striatum’, which was a ‘striking clinico-anatomical phenomenon’, it was ‘convenient to isolate this syndrome under the term Jakob’s disease’ (Critchley & Greenfield 1937:95). Greenfield was therefore involved with the differential diagnosis of Jakob’s disease in 1937 and therefore was well placed in 1957 to comment in relation to kuru pathology.

Back in 1934, little was known at all about the significance of the destruction of Purkinje cells, but one thing that was known was that in both X disease and louping ill this feature was evident. Galloway referred to Greenfield in a letter he wrote to Burnet on October 16th that year while discussing the significance of the Purkinje cells in relation to X disease. He remarked that

... our knowledge on the possible existence of Purkinje cell destruction in other conditions is very meagre and there is that condition “Parenchymatous cortical cerebellar atrophy in the human being described by Parkes and Kerrohan [?] 1933 and Greenfield also described ... a case in which there was affection of the Purkinje cells. On the other hand the conditions referred to above appear to be chronic and evidence as to Purkinje cell destruction in acute cerebral infection other than louping ill is not yet forthcoming.

In other words, Purkinje cell destruction was evident in both X disease and louping ill, as it was in the disease Greenfield described. The diseases described by Greenfield and Kerrohan however had a much longer course than X disease or louping ill. One of

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29 This term can be spelt with either a J or a G.
30 The reference for Greenfield’s paper just published in Brain (57:161) was passed on to Burnet for his information.
31 This case is not to be confused with Greenfield’s later publication about Jakob’s Syndrome.
the reasons for speculation about louping ill was that Perdrau had been ‘in touch with Cleland in Adelaide and received some histological material from the 1917-18 cases of X disease [from] humans monkey and sheep’\textsuperscript{32}. He also obtained ‘some material from the more recent cases at Broken Hill’ from 1925 and 1926, to which Burnet had referred in his paper (Burnet 1934). Galloway informed Burnet of the results of Perdrau’s research that had found destruction the Purkinje cells ‘to a slight extent in the human and to a more noticeable degree in the experimental animals’\textsuperscript{33}. Also, it was found that rabbits were not susceptible to either X disease or louping ill whereas sheep, monkeys and humans were. Burnet had recorded the same specificity in his paper with the addition of cattle. Galloway, well aware of the difficulties that might lie ahead of Burnet in terms of louping ill research, closed his report on the subject, many paragraphs long, with the words: ‘Here’s good luck to you!’\textsuperscript{34}.

The letter gives the impression that the likeness was enough to suspect louping ill, whereas previously it was thought that an aberrant form of poliomyelitis was the agent responsible for X disease.

Apart from the involvement of Purkinge cell destruction, there is other evidence that suggests a link between X disease and louping ill that specifically relates to some of the cases that appeared in 1926. On May 30\textsuperscript{th} 1934, Dr Cedric Duncombe, a general practitioner from Hobart, Tasmania, writes to Burnet immediately following the publication of his paper on the proposed link between the two diseases (Burnet 1934). Duncombe points out that in 1926 he treated some of the cases of X disease in Broken Hill District Hospital and brain material taken at autopsy from a patient that died was made into an emulsion and injected into sheep. This left a lasting impression on Duncombe who writes:

\begin{quote}
It is rather a vivid recollection I have of one sheep, I think about a fortnight later, which began ataxic circling. To the best of my recollection the cases
\end{quote}

\textsuperscript{32} That Perdrau was in contact with Cleland indicates another continuity in the research, at least relative to X disease/louping ill in England and Australia over a decade and a half between 1917-1918 and 1934.

\textsuperscript{33} Perdrau had proposed to Cleland, whose permission he awaited, to write a short note on his observation probably in \textit{Brain}. The article proposing the link between X and louping ill was published in the \textit{Journal of Pathology and Bacteriology} in 1936.

\textsuperscript{34} Letter dated October 16\textsuperscript{th} 1934 from Galloway to Burnet BP Series 10/1
were either definitely lethargic or definitely delirious and at the time we regarded them as X disease\textsuperscript{35}.

Duncombe understood that Cleland had been sent some specimens and these had been sent to ‘Flexner or some well known “bird” like that’. No doubt to differentiate the agent from a form of poliomyelitis. It would seem quite probable that these specimens were the ones that Perdrau had also been sent. It appears from the sheep experiment described by Duncombe that when X disease was inoculated into the sheep it manifested as loping ill. It is significant though that when scrapie is passaged through goats it has been found that two different syndromes can emerge. Pattison and Millson in 1961 (cited by Pattison 1988) refer to these as the ‘nervous’ type and the ‘scratching’ type. The nervous type was later referred to as a drowsy form of the disease, which seems to resonate with the lethargic type described by Duncombe. So it is difficult to be sure if what Duncombe observed was actually X disease/loping ill, but a fortnight incubation period tends to suggest acute encephalitis like loping ill rather than scrapie. Nevertheless, in both cases two syndromes emerged on serial passage of an agent, which only serves to highlight how complex both fields of research can be.

Complexity was no stranger to Jean Macnamara and Burnet, who by February 1935 were trying to differentiate between polio, loping ill and X disease. That year, Macnamara searched the parliamentary papers for the survivors of X disease to trace the cases that had been reported. Macnamara understood from the picture described that the cases that occurred in Moree in 1918 were more like polio than X disease. She wrote a note to Burnet saying, ‘do you know if anyone has tried to inoculate our Polio into sheep. I don’t think they have at the Rockefeller Institute – but if you have an active cord it may be worthwhile buying a few lambs’. A letter was going to be sent along with a copy of one of Burnet’s papers to a couple of dozen doctors in order to trace the more definite cases of X disease and thereby ascertain exactly what had caused the disease\textsuperscript{36}.

\textsuperscript{35} Letter dated May 30\textsuperscript{th} 1934 from Duncombe to Burnet. BP Series 10/1
\textsuperscript{36} Letter from Macnamara to Burnet dated February 2\textsuperscript{nd} 1935, Series 10/1 BP
To sum up what was known about the mysterious diseases during this early period and comparing the situation with what transpired later with respect to prion disease research, it is fair to say that both led to a degree of anxiety, uncertainty and speculation about the agent/s responsible. The fact that they could be transmitted experimentally to monkeys and sheep in the early cases demonstrates that at least some scientists during this early period were familiar with the idea of transmission of disease across species. These early experiments were no different from the transmission studies that took place during the nineteen sixties in which kuru, CJD, and scrapie was transmitted to chimpanzees and monkeys, albeit with a longer incubation period than had been experienced during the early part of the century\(^\text{37}\). But the idea of a longer incubation period was not a particularly new one given what was known about Gordon’s louping ill vaccine research which had brought to light this idea in the middle of the nineteen thirties. What was similar was the fact that in both time frames, when inoculated into experimental animals both X disease and scrapie could produce more than one syndrome.

The causative agent/s responsible for X disease remained elusive, although it was known that the agent was able to manifest in many forms. In other words the agents were polymorphic, as were the agent’s with which McGowan, the scrapie researcher, worked. And whereas an aberrant form of poliomyelitis was the preferred suspect in the early decades of the century, by the nineteen thirties X disease and louping ill seem to have been associated through the possibility of an iatrogenic pathway. With this in mind, it is interesting to look at what occurred relative to any discussion about louping ill in 1951 when another mysterious disease appeared in the Murray Valley.

**X disease and Murray Valley encephalitis**

In order to explore the occurrence of this disease it is necessary to by-pass events during the war years and move onto work conducted during the early nineteen fifties.

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\(^{37}\) For an outline of some of these later studies, Gajdusek, Alpers & Gibbs article (in Hornabrook 1976) is useful.
when outbreaks of an unknown encephalitic disease occurred in the Murray Valley in Victoria, and in Adelaide, Australia. There are a number of reasons for exploring this line of research. The first of which is that two of the researchers involved in the study of Murray Valley encephalitis (MVE) in 1951-52 – Drs S. G. Anderson and E. G. Robertson - were engaged a few years later in the investigation of kuru. Another reason is that in spite of the fact that studies during the nineteen fifties led to the conclusion that MVE was X disease, any idea of loping ill virus being associated with the disease seems to lose favour. This is curious because the 1934 correspondence between Burnet and Galloway show that the loping ill virus was closely associated with X disease, based on the characteristic destruction of Purkinje cells and the specificity of the agent in that it was pathogenic for monkeys and sheep, but not for rabbits.

On October 22\textsuperscript{nd} 1952, Burnet explained his views on MVE in a paper presented at the Joint Session of the Epidemiology and Laboratory Sections of the American Public Health Association at the Eightieth Annual Meeting in Cleveland, Ohio\textsuperscript{38}. There, he remarked, that ‘during the last two years I believe that my colleagues in Australia have solved a problem that has interested me ever since modern virological technics were developed in the early 1930’s [sic], i.e., the nature of the Australian X disease epidemics of 1917 and 1918’. He noted the similarity between these outbreaks and one that occurred in 1951 from which fatal cases had resulted in northern Victoria. Drs French and Miles isolated the virus and Burnet suggested that the agent behaved in ‘experimental animals like that transmitted to monkeys and sheep in 1918 by Cleland, Bradley and Campbell’. A ‘Special Article’ printed in the Medical Journal of Australia in 1951 notes that Cleland’s virus had been lost. Burnet (1952) pointed out, however, that transmission studies made with the virus isolated by French and Miles demonstrated that it could be transmitted in a similar way as the X disease virus of 1917-1918 and was specific for the same animal species.

There is no mention of loping ill virus in the description of MVE made by Burnet. Instead, the virus is placed in the same group of mosquito borne viruses as that which

\textsuperscript{38} A version of the paper was published in the December edition of the American Journal of Public Health (Burnet 1952: 1519-1521).
causes Japanese B encephalitis. A possible reason for this might be due to the influence of studies conducted by Miles, Fowler and Howes (1951), who reported on the pathology of a case of MVE in Adelaide that occurred in February that year. J. B. Cleland provided the specimen. These authors make a point of saying that their ‘case differs from the classical picture of X disease in that there was a lack of cellular infiltration in the brain’. In the monkey however, the clinical picture these researcher’s found was ‘exactly like that of X disease’. Nevertheless, their conclusion was that the ‘picture in both experimental animals and eggs is quite different from that in louping ill, of which one of us has had considerable experience’ (Miles, Fowler & Howes 1951:800). This was in spite of finding that the MVE virus was non-pathogenic for rabbits just as in X disease and louping ill studies conducted decades earlier, whereas other researchers had found that Japanese B virus was pathogenic for rabbits (Miles et al citing Koprowski and Cox 1946 and Webster 1938\textsuperscript{39}). So while this group of researchers found a similarity between MVE and X disease, louping ill virus was sidelined\textsuperscript{40}.

Robertson (1952), on the other hand, who examined the pathology of the cases that occurred of MVE in Victoria in 1952, and who incidentally also examined kuru pathology five years later, seemed more willing to keep an open mind on the possible causative agent. He makes a point of referring to Perdrau’s re-examination of Cleland’s pathological specimens from 1917-1918 and mentions the Purkinje cell destruction and the presence of ‘empty baskets’ in the brain, which had been used as a criterion for the diagnosis in both louping ill and X disease. He remarked that the same pathology was a feature of MVE. Robertson acknowledged that the pathology of MVE was similar to Japanese B encephalitis, but the fact that he concluded his article by pointing out the similarity between MVE and X disease with an emphasis on Perdrau’s findings suggests that there was room for doubt about the agent that caused MVE.

\textsuperscript{39} Koprowski and Cox’s paper was entitled ‘Studies on Chick Embryo Vaccine against Japanese B encephalitis virus’ and was published in the Journal of Immunology Volume LIV page 357. Webster’s work in 1938 focussed around ‘Japanese B encephalitis virus: Its differentiation from St Louis encephalitis virus and relationship to louping ill virus’ and was published in the Journal of Experimental Medicine Volume LXVII page 609.

\textsuperscript{40} Miles et al note that in the cerebellum the Purkinje cells were selectively attacked in a patchy fashion (1951:799).
Ian Wood, in a paper entitled ‘Burnet and Medicine’ (1969) summarises this line of research⁴¹. He makes reference to Litchfield’s account of the 1917 outbreak of the “mysterious disease” from Bourke and remarks that Burnell, and Cleland and Burton Bradley, following on from Litchfield’s work in 1917, ‘suggested an infective cause but no organism was isolated’ (Wood 1969:305). He also notes the isolation of Murray Valley Encephalitis virus carried out by ‘French, Anderson, McLean and their colleagues’ under the direction of Burnet. And then in 1952 Anderson found elevated antibody titres to the MVE agent in ‘persons who had lived in the area in 1917’. This, Wood then suggests, demonstrated that the St. Louis encephalitis virus (SLE) in U.S.A. and the Japanese B encephalitis virus (JBE) in Asia were closely allied. And, Subsequent surveys of Anderson provided good evidence that the cases of encephalitis from Bourke N.S.W. (“The Mysterious disease”), reported by Litchfield in 1917 and later known as “Australian X disease”, were cases of Murray Valley encephalitis’ (Wood 1969:305).

There is no mention of louping ill in Wood’s account either. Somehow it too, gets bypassed. Nonetheless, the mysterious disease of nineteen seventeen was linked to X disease and X disease was linked to MVE.

Here, it is important to remember the discussions held between Burnet and Galloway in 1934 and the work published in 1936 by Perdrau that proposed that the agent responsible for the X disease outbreaks was louping ill virus. Galloway was also willing to entertain this idea. In view of this, it seems curious that louping ill was not found to be associated with MVE, even though MVE was considered to be the same as X disease. It was not that the earlier suggestion of a causative relationship between X disease and louping ill was not revisited during the nineteen fifties when a form of X disease - MVE - came along. As the paper by Miles Fowler or Howes indicates, one of these researchers had considerable experience working with louping ill virus. Rather there seems to have been uncertainty regarding the relationship. Perhaps the agent of MVE was similar to that which caused Japanese B encephalitis, but if this is the case and MVE and X disease were considered to be the same disease, then how is

⁴¹ Wood’s paper is published in The Australian Annals of Medicine (Volume 18:4 pp 1-9), and it was from this paper that I obtained the reference to Litchfield.
it that MVE was not more firmly associated with louping ill virus? If we let A stand for X disease, B stand for louping ill virus, and C stand for MVE, its like saying that A is probably caused by B, A and C are the same, but C is not caused by B.

A question that arises from this conundrum is from where did the louping ill virus with which Miles et al worked come? Was this research related to the outbreaks of scrapie in Australia during the early nineteen fifties that were caused by louping ill vaccine? A separate question relating to the research conducted during the nineteen thirties is, was the louping ill virus with which Burnet in 1936 the virus that quite possibly had not been given clearance into Australia during the nineteen thirties? If this is so and the louping ill virus did enter Australia in 1934 without permission from Canberra there would have been good reason to keep this earlier line of investigation quiet. This is not to discount that in 1968 Burnet mentions his research with the virus. Whatever the answer to these questions, the title of Series 10 of Burnet's papers - 'X-disease, Kuru and Gajdusek files' - implies a connection between research conducted during the nineteen thirties, research carried out some two decades later on Murray Valley encephalitis/X disease beginning in 1951, and the investigation of kuru.

Given that the correspondence in Series 10/1 revolves around louping ill virus being suspected as a cause of post vaccinal disease, could this have provided a reason not to openly discuss this issue in scientific publications? As Jennifer Cooke points out, Gordon's research on the effects of louping ill vaccine was published in only a few articles and was not widely disseminated. But even though the findings of Gordon's research might not have been widely disseminated, there is evidence that at least some influential individuals like Burnet and the Australian Director General of Health knew about the links between X disease, louping ill virus and post vaccinal disease. Moreover, Burnet was aware of Gordon's preliminary work on the preparation of the vaccine, even before the vaccine was shown to produce scrapie. What matters in interpreting these events is not an assessment of the actual numbers of scientists and medics who shared this knowledge, but rather their influence.

Burnet as Assistant Director of the Institute during the nineteen thirties and as Director of the WEHI during the nineteen fifties, was in a position to alert the medical community more broadly about the possibilities of post vaccinal disease generally.
And he did this to a certain extent during the late nineteen twenties when he was involved in the investigation of the Bundaberg incident, in which children died from the use of a particular vaccine (see Wilson 1967). He was therefore aware of problems associated with the use of vaccines. But when it comes to the case of louping ill virus and vaccine research relative to the sharing of knowledge between the fields of human and animal health, there seems to be no interest in acknowledging what was known across this divide.

By exploring the field of research that involves X disease/louping ill and MVE I have shown that cross-fertilisation of knowledge existed between human and animal health from the nineteen twenties onwards. This is particularly evident in the line of work that extended between the mysterious outbreaks of disease in 1917 and 1918 to the work conducted on X disease and louping ill. Burnell’s knowledge of McGowan’s work on scrapie is one example. The close relationship between Burnet, Galloway and Gordon, who was working on louping ill vaccine research at the time, is another. Even by the nineteen fifties trans-disciplinary sharing of knowledge existed, evidenced by Miles, Fowler and Howes’s contribution to the literature on MVE, which was conducted at the Institute of Medical and Veterinary Science in Adelaide. For some reason, this sharing of knowledge seems to have been forgotten.

June Goodfield in *Quest for the Killers* (1985) makes the point that none of the scientists involved in the kuru transmission studies conducted during the early nineteen sixties had ever heard of William Hadlow, the veterinarian. Hadlow is said to have suggested that the studies required a long time frame because he knew that scrapie required a long period of incubation (1985:27). The implication is that there was a lack of cross-over between research on animal and human health. And yet, this was precisely the focus of Burnet’s research. Challenging this narrative is important in that it is sometimes used to explain why little knowledge about prion diseases of animals (scrapie, mink encephalopathy and BSE) was known to scientists working in the field of human health (kuru, iatrogenic CJD, nvCJD). This argument has been used in the CJD Inquiry in England as I mentioned.

It is difficult to say why Burnet’s papers on X disease and kuru have not been researched and the relationship discussed. Perhaps this is because the material was not
collated until 1985 when the Australian Science Archives Project began. Nevertheless, fifteen years have elapsed since this time, and the material had been housed at a medical library in Australia prior to this time. Perhaps also, there are a greater number of research topics available than researchers. But when the documents are considered, it is clear that Burnet along with the Director General of Health knew about the possible relationship of post-vaccinal encephalitis and louping ill relative to X disease in 1933. X disease was then associated with Murray Valley encephalitis. And then came kuru. Any mention of the possibility of transmission of various agents via vaccines is absent in the dominant kuru narratives, whereas this is precisely the focus of Burnet’s papers collected during the earlier period of research.

The similarities between the pathology of kuru and that of X and louping ill - the dysphagia, the neuronophagia, the Purkinje cell destruction in the cerebellum, as well as the fluctuating symptoms – are difficult to overlook. I am not arguing that all three disease were the same. What I am reminded of is a story told by Michel Morange in his book A History of Molecular Biology (1998). Morange recounts how during the nineteen fifties Alfred Hershey and Martha Chase were credited with discovering the genetic role of DNA. And yet, eight years earlier Oswald T. Avery had made similar findings. Morange shows how the discoveries of Hershey and Chase had a long history. Their work was the culmination of a long series of experiments. One wonders whether there are any parallels in the kuru story. Could it be the case that the study of kuru was somehow the culmination of the research on louping ill or possibly another vaccine, perhaps against MVE or Japanese B encephalitis? The following chapter explores Series 10/2 of the Burnet Papers, with this question in mind.
Chapter 3

From Murray Valley encephalitis to Kuru

The relationship between X disease, louping ill and poliomyelitis virus has been examined and the similarities between X disease and kuru have been brought into the foreground. Where the previous chapter focussed on research between 1917 and 1951, at which time X disease reappeared and was named Murray Valley encephalitis (MVE), here, relevant research conducted between 1951 and 1958 is explored. By this time the first year of the kuru investigation was drawing to a close. Events pre and post the investigation in 1957 can be seen as ‘book-ends’ that might help to understand the conflict story that has been written about kuru.

A conflict is said to have developed in the kuru investigation related to two factors, the first of which was Gajdusek’s arrival in New Guinea in March 1957. One commentator has written that ‘Gajdusek wrote copious letters trying to smooth out the trouble over his presence’ and ‘while the discord swirled between Port Moresby and Melbourne, Gajdusek, intent on his work, dismissed it in one of his letters to Smadel’ (Cooke 1998: 6 & 7). Dr Joseph Smadel at this time was the Associate Director of the National Institute of Health in America and Gajdusek’s mentor. Rhodes states that ‘Mac Burnet had expressed his displeasure that native-born Australians weren’t handling the investigation (Zigas was a refugee from Estonia who had found his way to Australia after the Second World War). Gajdusek brashly wrote his Australian mentor [Burnet] to lay off’ (Rhodes 1997:39). As Hank Nelson has written in reference to Gajdusek’s arrival, ‘[h]ere then was a strange situation’, for Gajdusek had ended his ties with Burnet in Melbourne, had no position with the Public Health Department in the Territory, no funds, and was ‘directing a major project’ that had been planned for Dr S. G. Anderson (in McCalman, Penny & Cook 1998:145). The second factor relates to events that occurred towards the end of 1957, when Burnet is said to have withdrawn completely from the kuru investigation because specimens

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1 According to a letter written by Gajdusek in 1957 Zigas was Lithuanian, not Estonian (Gajdusek 1975:56)
were being sent to America without his knowledge (Gunther 1977. Nelson in McCalman, Penny & Cook 1998:145). Dr E. G. Robertson, the first Australian pathologist to examine kuru specimens referred to this situation in a letter he wrote on October 31st 1957 to Dr J. G. Greenfield, Visiting Scientist at the National Institute of Neurological Diseases and Blindness in the United States. There, he noted that only recently had Australia been made aware of the situation and said, ‘I am baffled by it all, and obviously do not understand all the facets - therefore the less said the better’. Burnet is said to have agreed (in Farquhar & Gajdusek 1981:229-230). I want to show that, whilst there was a degree of conflict over Gajdusek’s arrival and his sending specimens to America, a different emphasis emerges when the correspondence in Series 10/2 and the events preceding, and immediately following, the investigation are taken into consideration. As previously mentioned, many of the accounts written about kuru begin only with events in 1957, thereby leaving events that occurred during the years leading up to the investigation that might have a bearing on the situation overlooked. Reading accounts of kuru can sometimes leave the impression that there were no affiliations between Australian and American researchers prior to the investigation in 1957, apart from Gajdusek’s stay with Burnet at the Walter and Eliza Hall Institute in Melbourne from the end of 1955. My aim is to highlight the collaborative relationship that existed between Burnet and Smadel throughout the nineteen fifties related to research on MVE and other members of the Group B encephalitides, such as Japanese B encephalitis and Yellow Fever. In addition, I will review some of the research concerns of the period of the nineteen fifties, thereby providing a fuller account that might help to understand the conflict that is said to have developed.

**Burnet and Smadel: Early collaborations**

Smadel and Burnet were involved in many different studies conducted on diseases of Group A and Group B encephalitides during and after the outbreaks of Murray Valley encephalitis in 1951. Much of the research of the period was aimed ultimately, at the manufacture of effective vaccine to protect against these diseases.
In May 1951, an Australian National Health and Medical Research Council report, states that:

“If it is finally proven that the virus [of MVE] is that of Japanese encephalitis B steps should be taken to obtain all available information as to the efficacy of the vaccine used by US troops in Japan against this disease and if necessary to produce such a vaccine here”.

The following year, some of the work conducted on MVE was briefly discussed in the ‘Report on Virus Epidemiological Unit’ (1952) of the Walter and Eliza Hall Institute. Also discussed was a ‘field study of the efficacy of gamma globulin from convalescent serum in the early stages of a rubella epidemic in a service camp’. Dr S. G. Anderson and D. McLean, a Junior Medical Fellow working at Walter and Eliza Hall Institute, conducted this study and found that gamma globulin was not wholly successful. Anderson thought however that there might be a possibility that the globulin ‘may have a capacity to protect 60% of those so treated’. Whilst this last example does not specifically refer to Group B encephalitides, the records show that preventative medicine was uppermost in the minds of some of the researchers involved in the study of MVE.

In 1951, when Anderson, Burnet and other researchers in Australia studied MVE, American scientists assisted in the studies (Burnet 1952). Anderson, French, and Miles worked in ‘well equipped laboratories’ with American researchers in Australia, Japan, and Okinawa (Burnet 1952:1520). Burnet points out that the virus of MVE was categorised along with Japanese B, St Louis, and West Nile virus as Group B encephalitides and all were mosquito-borne viruses. In a paper on the isolation and

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4 The Director’s Thirty-Sixth Annual Report to the Board of The Walter and Eliza Hall Institute of Medical Research, July 1955, indicates that at the end of 1954 Dr Anderson succeeded in growing the virus of rubella in tissue culture ‘prepared from monkey kidney cells’. Hopes of advances in rubella in pregnancy had been dashed however, as it had been found that ‘normal appearing monkeys rather frequently carry a latent virus in their kidneys and early in 1955 this virus was found to be contaminating the tissue cultures and rendering work on rubella impossible’ (p6). Anderson then repeated the work ‘with elaborate safeguards’ in place, which, it was hoped, would ‘eliminate the unwanted virus’ (p6). (BP Series 8/42). The following year, ‘because of insuperable technical difficulties, two years work with tissue culture of rubella virus terminated’ (The Director’s Thirty-Seventh Annual Report to the Board of The Walter and Eliza Hall Institute of Medical Research, July 1956).
characterisation of the causative agent of MVE, French notes that testing facilities in Australia at this time were not available to differentiate between the agents of arthropod borne encephalitides (French 1952). Consequently, Colonel J. E. Smadel, Chief of the Department of Virus and Rickettsial Diseases, Army Medical Service Graduate School in Washington, received specimens from French to see how closely aligned MVE was to the other members of the group. In 1954 and 1955, Smadel co-authored two relevant papers that highlight the research in this area. One of these is entitled ‘Japanese encephalitis in Malaya’ (Pond et al 1954). The other is called ‘Murray Valley encephalitis: its serological relationship to Japanese B encephalitis-West Nile-St. Louis encephalitis’ (Pond et al 1955). This brief survey shows the close alignment of the interests of Smadel and Burnet during the years leading up to the investigation of kuru and the collaboration between them. Having established the collaborative efforts I turn to the research climate during the nineteen forties and fifties as it is important to understand the relationship between military and scientific research at this time.

The climate of scientific research during the 1940s and 1950s

The importance of both Group A and Group B viruses was realised in military circles at least by 1945, when Colonel Arvo Thompson addressed the Chief of the Chemical Warfare Service, Special Projects Division in America. At this time, American intelligence was receiving word that the Japanese might deploy balloons carrying biological weapons. Thompson had the following to say about the subject:

The type of agent chosen would depend upon the degree of accuracy with which the balloons could be sent. In the event that city limits were to be the site of landing, effective agents would be either epidemic in character (e.g. pneumonic plague) or non-epidemic agents easily transmissible via the respiratory tract (e.g. psittacosis). If non-accurate dispersion were attempted, insect-borne agents or those affecting livestock would be indicated .... One of the most likely agents might well be Japanese B Encephalitis since it has been shown that this virus can be transmitted by every mosquito capable of carrying equine encephalomyelitis (Eastern and Western) .... If this type of wholesale

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5 Anderson (1952) makes the same point.
distribution were used on our pasture lands with our important food producing animals as the ultimate target, the bacillus of anthrax, the viruses of Foot and Mouth, and Rinderpest might be effective (quoted by Avery in Macleod 2000:241).

Avery’s quotation highlights the problems associated with the agent responsible for producing Japanese B encephalitis, and indicates the concern this agent posed to military authorities in 1945, just as it did a decade later. In September/October 1955, Dr John Gunther, the Director of Public Health in the Territory, discussed research being conducted by an American army team in Kuala Lumpur with Dr Hale, the Rockefeller Professor of Bacteriology. The meeting occurred at the University of Malaya in Singapore and Gunther was fascinated by the work Hale’s team was conducting on Japanese B encephalitis.

The above quotation also shows the problem associated with rinderpest. This provides a rationale for a joint American and British covert rinderpest vaccine trialed on a significant number of sheep in Kenya in 1945. The temporal relationship between this trial conducted in Kenya and Stamp’s 1953 report on nakuruitis mentioned in chapter one, are obvious. What is not so obvious is the similarity between the Kenyan rinderpest research and the investigation of kuru. One similarity relates to the idea that America did not want to be seen to be associated with the work. In 1957, when the Australian Commonwealh Film Unit, Department of Information, made a film on kuru in New Guinea, it was also suggested that efforts should be made to disguise the fact that an American scientist was involved (Gajdusek 1975:398). Perhaps this

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6 It is not entirely clear whether Avery meant to write our instead of out. Either way my interpretation of the quotation in full is not altered.
7 Letter dated April 19th 1956 from Gunther to Burnet. BP Series 10/2
8 Appendix 3 provides information about the Kenyan trial.
9 The film Kuru: A Clinical Study is archived at the National Sound and Film Archive, Canberra
10 In a letter to Smadel dated February 22nd 1958, by which time, Gajdusek was in Kuala Lumpur at the U.S. Army Medical Research Unit having just left New Guinea after the first year of investigation, he wrote about a film made by the Commonwealth Film Unit. The relevant passage reads:

The long 35mm film on Kuru clinical features is completed with my sound track and in a note from Professor Eccles of Canberra, I learn that he has been asked to be photographed reading or talking, at least, an “introduction” to the film, which is in the script he sent us embarrassingly laudatory of our work. However, the “political” pressures worrying the Territorial government, i.e. to disguise the fact that I am American. They trace all our work to the basic ‘discovery’ of Kuru by Berndt (which I was first to point out, although Berndt is still confusing Kuru with hysterical mimicry and arguing by mail to me that we are really “not trained” to study it, that he is “Australian”, etc., etc.) ... all very amusing and inconsequential. However, Eccles is doing a fine piece of work for us and helping on every hand (Gajdusek 1975:398).
possible relationship should not be given too much weight because the soundtrack of the film did mention the involvement of America, and it would have been difficult to disguise American involvement in the kuru research, for by November 1957 the study had been reported in a scientific journal. Regardless though, of what actually transpired, talk of an intention to disguise American involvement was the same in both kuru research and the Kenyan rinderpest research.

What should also be taken into account is that while the Kenyan research was framed as ‘visitors studying work on diseases or stock being carried out in Kenya’, the study was related to testing a vaccine in order to protect against possible biological attack. This indicates that the real purpose of the visit of the scientists was concealed through a distortion of information. Distortion of information also occurred in a project conducted by Gajdusek in 1954 judging by a letter written to Smadel on May 5th. At this time, there were concerns that an ‘incident’ might occur. Gajdusek tried to allay fears expressed by Smadel and Dr T. Woodward. The two men were told not to worry when reports appeared in the press as the Embassy had given Gajdusek all the “dope” and told him ‘not to worry if local papers carry notes about a “team with an American doctor” … working in the “closed” border areas’. His superiors were assured that everything ‘will be completely cleared and sponsored by the Iran and Afghanistan government’ (Gajdusek 1991:317 ellipsis in original). It was pointed out that the work had been ‘cleared with our Embassy on all matters, including with our Embassy political advisor… and we shall be working directly with the military and civil governors of the Meshed area and of any other district we enter… so have no fear about “incidents”’ (Gajdusek 1991:317 ellipsis in original). This suggests that what might have been reported in the press, was not necessarily an accurate representation of events. I should stress that there is no intention here to suggest wrongdoing. It is important to note that the Second World War pressed into service scientists from many institutions, including academic faculties, in order that knowledge and skills could be brought to bear on the war effort (Macleod 2000). The situation was little different during the nineteen fifties.

Following the Second World War, not only was there still a threat of war - the Korean War was waged between 1950 and 1953 - there was also the possibility of nuclear, biological and chemical threats, just as there had been in 1945. When hostilities
abated between the Allies and Germany after the Second World War, the tensions did not cease but rather changed focus and greater emphasis was given to the threat of Communism. It was during this period of heightened tension that Burnet, in his role as a member of the Commonwealth Defence Research and Development Committee, was called on to examine allegations made by the Chinese People's Committee for World Peace that United Nations troops had used germ warfare\(^\text{11}\). These allegations were denied. Nevertheless, Burnet was only too well aware of the dangers of meddling with bacteria and viruses, particularly in terms of recombination experiments as is evident from his book *Viruses and Man*, written in 1953.

After a discussion about animal and plant viruses, he wrote

... at this point we reach the characteristic dilemma of the twentieth century. Every aspect of virus disease justifies full investigation. From such investigation we may hope for improved means of preventing or curing disease, but equally the fuller our knowledge becomes the greater the threat of the use of that same knowledge for the deliberate dissemination of disease in war or in some other phase of the incessant struggle for power amongst men. We cannot refuse to face the position that we have just entered an era of extraordinarily effective biological research, an era that may be as productive as the golden age of atomic research that reached a provisional culmination in Hiroshima and Nagasaki. Laboratory research is at least as potent to produce new evils as to counter existent ones (Burnet 1953:195-196).

The allegations, mentioned above, might well have been on Burnet's mind when he wrote this passage, although he does not make reference to these events in his book. In contrast, he remarks that it would probably be two or three decades before viruses would be able to be used in biological warfare. Yet, biological weapons were being used experimentally from the late nineteen thirties, particularly by Japan (Williams & Wallace 1989). To counter the potential threat, America began its own biological research station early in World War II at the 'well-equipped laboratories at Fort Detrick in Frederick, Maryland' (Fothergill 1964:8)\(^\text{12}\). L. D. Fothergill suggests that

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\(^{11}\) *Exhibition on Bacteriological War Crimes Committed by the Government of the United States of America* (Williams & Wallace Chapter 11 Note 3). See also Sexton 1991:128. Relevant papers for the period in question (1952-3) are kept at the Adolf Basser Library, Canberra. Ref 98/2a/6.

\(^{12}\) L. D. Fothergill (1964) notes in his paper 'Biological Warfare: Nature and Consequences' that it was known during the nineteen fifties that biological weapons could devastate people and yet leave the infrastructure of a community intact. Biological warfare (BW) is 'the use of living microorganisms to reduce the military capability of an enemy'. 'Its most important effect would be in disrupting the economy of a nation through incapacitation of its working force and reduction of its agricultural crops.
this ‘form of warfare has never been employed on a planned, overt, military basis’. But saying that biological warfare has never been used in this way does not preclude the possibility that it might have been employed covertly. Because of the nature of the work conducted during this period, secrecy was the order of the day. To go back to the Kenyan trial for a moment, it was only one example of post-war co-operation between the United States and Britain that involved using a tropical country to test either a vaccine or biological agent\textsuperscript{13}. Brigit Goodwin’s (1998) recent studies indicate that parts of Northern Queensland were used to test the effects of chemical weapons under tropical conditions\textsuperscript{14}. These examples show that tropical countries were seen as places where drugs and vaccines could be trialed by Western governments. During this period, along with conscripts of war, it was common for prisoners, people with mental illness and poor people to be used for trials without consent to further the security effort\textsuperscript{15}. McNeill (1993) points out that after the Second World War military policies were wound back, but this did not necessarily bring a halt to military thinking within health administrations. Rather it was only the setting that changed while much of the work continued, albeit from within the confines of government institutions rather than military ones\textsuperscript{16}. Therefore, the ethico-cultural context needs to be taken into account.

\textsuperscript{13} Dr E A Perren, a British scientist from the Biological Research Establishment at Porton Down, in 1948, recommended a 25-30 square mile site near Benin, Nigeria, as suitable to test chemical and biological weapons in “hot, humid conditions” (Letter from Ministry of Supply to L H Gorsuch, Colonial Office, October 28\textsuperscript{th} 1948 in Williams & Wallace Chapter 10 Note 16). Arrangements for supplies of small animals “for research” were approved including mice and rats “bred at the Rockefeller Yellow Fever Institute, Yaba, Lagos...” A few years earlier, George W. Merck from Fort Detrick highlighted the potential threat of biological warfare in a “Report to the Secretary of War by George W. Merck”, January 3\textsuperscript{rd} 1946, Fort Detrick (Williams & Wallace Chapter 11 Note 2).

\textsuperscript{14} Mellor (1958) also writes about Britain and Australia’s testing of chemicals in Australia during the post war years, but Goodwin’s work reveals the extent of the tests.

\textsuperscript{15} Society saw this as a way for prisoners to repay their debts to society for being ‘bad’. Freund (1972: xvi) comments on the belief that prisoners could get a sense of satisfaction and a resultant increase in self esteem by consenting to experiments for the ‘good of society’. A member of a symposium on human experimentation in 1972 said that he thought it was ignorant not to submit to experimentation ‘for the greater good’. This included submitting to immunisation. Not many other members of the forum agreed with their colleague (Freund 1972: xiii).

\textsuperscript{16} McNeill highlights the close ties that exist between medical research and the military, and suggests the machinery of scientific and medical research go hand in hand with war efforts. After the Second World War $25M was given to universities, hospitals, institutes, and companies, to find ‘cures’ for malaria with the ultimate aim being to benefit soldiers fighting in war (McNeill 1993:20).

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The aim here is to show how these events occupied the mind of Burnet and other eminent scientists throughout the years leading up to the kuru investigation in 1957. This information is useful as a gauge of the times and any analysis of the scientific history of this period is decontextualised without the interconnections of military and medical research.

As to whether the investigation of kuru should be rightfully seen within the context of military research is a difficult question to answer. There is little doubt though, that surplus military supplies were used for the kuru transmission studies during the early nineteen sixties at the Patuxent Wildlife Research Center (Rhodes 1997:73 & 86)\textsuperscript{17}. And that four members of the Armed Forces Institute of Pathology assisted in the autopsy of the first chimpanzee that succumbed to kuru after having been inoculated with brain suspension taken from a patient that had died of the disease (Goodfield 1985:38). It is not surprising therefore that the text, *Pathology of Tropical and Extraordinary Diseases*, which includes the chapter on kuru by Heffner and Strano, was published by the Armed Forces.

I have found no evidence that either biological weapons research or a vaccine trial to guard against such attack has been conducted in New Guinea at some time in the past. But the circumstantial evidence of military work having been conducted around this period leads me to conclude that the issue should be more thoroughly researched. The thesis aims to provide some of the background information from which further and more widely enquiring studies can move forward and discussion about kuru can be opened up before it is relegated to a footnote in medical history.

During the period in question, problems were identified with the routine use of some vaccines. Yellow Fever, a member of the Group B viruses, also had a problematic history within a military context. In 1942, ‘28585 young American soldiers inoculated with yellow fever vaccine developed jaundice and 62 died’ (Zuckerman 1996:127). That is one in four hundred and sixty one deaths as a result of the vaccine. Jaundice

\textsuperscript{17} Patuxent was located between Washington and Baltimore, south of Fort Meade, and was operated by the Rare and Diminishing Species Program of the Department of the Interior’s Fish and Wildlife Service (Rhodes 1997:73). Gajdusek is said to have reported to Rhodes that a site was needed for the transmission studies “where nobody could see what we were doing”.

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could result from either syringes and needles improperly sterilised between successive use in different patients or from the incorporation of convalescent serum into a vaccine. Convalescent measles serum, batches of yellow fever vaccine in which human serum had been incorporated in the virus culture medium, and mumps convalescent plasma, all resulted in cases of hepatitis. In 1954, the Australian Report of the Director-General of Health for the year ended 30th June noted, that Yellow Fever vaccine produced at Commonwealth Serum Laboratory had been approved for international use by the World Health Organisation following tests carried out overseas (undisclosed). The report indicates that ‘the keeping qualities of the vaccine were better than was generally assumed’. In 1956, the Rt. Hon. Mr. P. M. C. Hasluck, the Minister for Territories, made reference to Colonial Office interest in Yellow Fever in a letter written to Burnet on May 10th in relation to increased air travel. Burnet suggested that the prevention of the spread of this disease was ‘perhaps the most important public health problem in the world’, although no mention is made in this correspondence of any research related to vaccine. Nevertheless, preventing the spread of Group A and Group B encephalitides was seen as vitally important at this time, as were the efforts to produce vaccine or sera to immunise against these diseases.

During the nineteen fifties, at the same time as Smadel and Burnet were attempting to differentiate between Murray Valley encephalitis and Japanese B encephalitis, there was also intense interest in the study of variants of hemorrhagic fever, a disease of considerable importance militarily between 1952 and 1955. In 1952, Smadel headed the team sent by the American Armed Forces Epidemiological Board, Office of the Surgeon General, to Korea to conduct studies on the aetiology, epidemiology, and arthropod phases of hemorrhagic fever (Traub, Hertig, Lawrence, & Harriss 1954). In September 1954 it was thought that hemorrhagic fever bore similarities to ‘foreign serum reaction’ described in the latter part of the nineteenth century (Gajdusek

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18 The Report of the Director-General of Health for the year ended 30th June 1954 (1955 pp 23-28). The Report was the first report of its kind issued by the Commonwealth Department of Health. At this time, an International Conference between Dutch and Australian Health Authorities was held at Minji in the Western Highlands of New Guinea (p 54).
19 Letter dated May 10th 1956 from Hasluck to Burnet BP Series 10/2.
20 Letter dated April 12th 1956 from Burnet to Hasluck. BP Series 10/2.
21 Hale was particularly interested in Group A viruses allied to Mayaro and Chikungunya. Letter dated May 2nd 1956 from Hale to Burnet. BP Series 10/2.
1991:362). At this time, research was also being conducted on rabies and loping ill vaccine, and Gajdusek wrote: ‘I prostitute myself in enslaving myself to the vaccine bottles’ (Gajdusek 1991:218). By 1956, a number of diseases (hemorrhagic fever from Omsk, loping ill disease, Russian Spring Summer encephalitis (RSSE) and another meningo-encephalitis from Slovenia, Austria and possibly Czechoslovakia), were all attributed to the same agent\textsuperscript{22}, and in 1957, kuru was likened to Epidemic Hemorrhagic Fever (EHF). It is not that the two diseases manifest in the same way. The similarity lies in the fact that kuru was proving as difficult to deal with as EHF had been in Korea\textsuperscript{23}. Given that HF has been spoken about in terms of a possible reaction to a vaccine or sera, the problems that were identified related to the production and use of vaccine, sera or other manufactured product need careful consideration.

In New Guinea, when regions were opened up for ‘exploration’ it was routine to implement vaccination campaigns. In May 1951 a paper written about the expanded health programme for the Territory of Papua and New Guinea noted that ‘during the current year 100,000 calf lymph vaccinations have been carried out’ (Gunther 1951). A policy of mass immunisation was implemented with \textit{Bacillus Calmette-Guérin} commonly known as BCG against tuberculosis and in 1951 the programme was proceeding at a rate of 300,000 vaccinations per annum, sourced from the Commonwealth Serum Laboratories (CSL) in Melbourne (Gunther 1951). CSL worked closely with the Walter and Eliza Hall Institute. The region in which kuru occurred was one of the last areas to come under the control of the administration, a process that began during the early years of the nineteen fifties, although patrols began in the kuru region as early as 1947. By 1953, ‘people were themselves choosing to walk to Moke for injections against yaws’ as medical supplies had been in the area for some time (Nelson 1996:186)\textsuperscript{24}. In March 1957 at Lufa, the area adjacent to the kuru region, the administration of 12000 injections of penicillin as a treatment for Yaws took place. Judging by a comment made by Ian Burnet, Patrol Officer of the

\textsuperscript{22} Letter from Gajdusek to Smadel dated April 26\textsuperscript{th} 1956 in Gajdusek 1975: 32.

\textsuperscript{23} Letter from Gajdusek to Smadel dated August 6\textsuperscript{th} 1957 Gajdusek 1975:174. Gajdusek wrote to Smadel again in September saying, ‘as with EHF, we are thus far licked’ (Letter from Gajdusek to Smadel dated September 18\textsuperscript{th} 1957 in Farquhar & Gajdusek 1981:159).

\textsuperscript{24} Moke is the name of one of the areas in the Kuru region where the main patrol post was built. This is often referred to as Okapa Patrol Post.
Lufa region, who accompanied a European Medical Officer and an Agricultural Assistant on the patrol where the injections were administered, it is clear that he had difficulty in understanding their purpose. He wrote to his father saying, ‘we saw only about 20 active cases which made the whole thing seem pretty futile’. Administration of any injection is a serious business regardless of whether it contains a vaccine, sera or antibiotic. That so many injections have been given in New Guinea under difficult conditions is a cause for concern. Kuru however, did not happen everywhere. It was confined to one particular area. Therefore something unique seems to have happened in this area. Cannibalism, of course, is the widely believed route of transmission. But cannibalism is also said to have occurred in other areas, so it too, is not specific to the area in question. Another possibility is that a particular batch of product was used in this area that might have been contaminated. The next chapter assesses the likelihood of this possibility. For now, I want to return to the negotiations held in 1956 in relation to Groups B viruses to show their complexity and the confusion evident in the correspondence for the period.

**Significant conversations and meetings: 1956 to 1958**

Following on from the collaborative research conducted between 1951 and 1955 on MVE and other members of the Group B encephalitides, mentioned at the beginning of this chapter, a series of meetings took place in 1956 between American and Australian institutions to study these diseases in New Guinea. In 1955/1956 Burnet made an ‘unusually extensive series of visits to Ceylon, India, United States and Great Britain’. In India, he met with Telford Work, a virus specialist and in America, Smadel, Burnet and Dr Robert Morison from the Rockefeller Foundation, New York met and agreed that there was a need for a “staging laboratory” and a five-year plan of research in New Guinea. This laboratory was seen as a valuable addition to a number of other facilities around the world that had taken responsibility for dealing with local aspects of what Burnet termed the ‘general problem’. The proposed base in New

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25 Letter from Ian Burnet to Burnet dated March 11th 1957. BP Series 10/4
26 The Director’s Thirty-Seventh Annual Report to the Board of the Walter and Eliza Hall Institute of Medical Research, July 1956 p. 6 (BP Series 8/42).
Guinea was to accompany existing bases stationed in Cairo, Entebbe, Poona and Kuala Lumpur\textsuperscript{27}, which were operated respectively, by the United States Navy, the British Colonial Medical Service, the Rockefeller Foundation, and Britain and the United States jointly.

In April 1956, having returned from his overseas trip Burnet submitted a proposal with an outline of the research to Hasluck, Minister for Territories\textsuperscript{28}. He noted that Australia would ‘be in a far better position to understand the behaviour of Murray Valley encephalitis in Southern Australia if we could study it in what is presumably its “home base”, New Guinea and adjacent areas’\textsuperscript{29}. By 1956, one death had occurred from MVE in New Guinea, which is reported to have drawn ‘renewed attention to the importance of mosquito-borne diseases in the Territory’\textsuperscript{30}.

Hasluck was first alerted to Burnet’s proposal in early February by Dr John Gunther, Director of Public Health, who had been told by Robert Walsh, Director of the Sydney Blood Transfusion Service that Burnet was ‘keen to make certain epidemiological studies in the Territory’\textsuperscript{31}. Gunther informed Burnet that he hoped the studies would go ahead, that he had spoken to Hasluck in Canberra about them and that he had been fascinated by the United States Army research being carried out in Kuala Lumpur under Dr Hale. He had received word from Kuala Lumpur that Audy would like to visit Papua and New Guinea ‘in regard to the fauna side’ of the studies\textsuperscript{32}.

It is at this point that the correspondence is difficult to fathom. Gunther wrote back to Hale saying:

\textsuperscript{27} The Kuala Lumpur laboratories were actually based in Singapore.
\textsuperscript{28} Letter dated April 12\textsuperscript{th} 1956 from Burnet to Rt. Hon. Mr. P. M. C. Hasluck, Minister for Territories, Parliament House Canberra, ACT. BP Series 10/2.
\textsuperscript{29} \textit{Ibid.} Evidence available at the time suggested that ‘only under special conditions of rainfall, bird migration, etc.,’ was the virus brought south from the tropics as it was in 1917, 1918, 1925 and 1951. These were the years when outbreaks of what until 1951 was termed X disease occurred. As noted in chapter two, X disease was at first thought to be similar to a form of acute anterior poliomyelitis, but was later believed to be caused by louping ill virus.
\textsuperscript{30} The Director’s Thirty-Seventh Annual Report to the Board of the Walter and Eliza Hall Institute of Medical Research, July 1956 (BP Series 8/42).
\textsuperscript{31} Letter dated April 19\textsuperscript{th} 1956 from Gunther to Burnet. Series 10/2.
\textsuperscript{32} Audy was one of the co-authors of the paper written by Smadel in 1954 mentioned earlier.
Recently, we have been negotiating with Sir MacFarlane Burnet to follow up his Murray Valley Encephalitis work in the Territory. It is a coincidence that, within the last week or so, he has made certain specific proposals to the Honourable the Minister for Territories about his epidemiological survey in Papua and New Guinea.\(^{33}\)

There was no coincidence though, as Burnet must have alluded to a problem that might flow from the submission of his proposal to Hasluck, either in a letter to Gunther or in a telephone conversation.\(^{34}\) Gunther wrote back to Burnet saying ‘I personally do not feel that I am placed in an awkward position.’\(^{35}\) He believed that the field in New Guinea was a fascinating extension of Burnet’s Murray Valley encephalitis research and he hoped that Burnet would soon hear from Morison in New York so that the matter could be settled. Nevertheless, he indicated that he would prefer the work to be ‘performed by Australians, if that is at all possible’ (emphasis in original). The correspondence suggests that the submission of Burnet’s proposal simultaneous with the developments from the Kuala Lumpur laboratories led to a potentially awkward situation developing for Gunther. It also indicates that while Gunther might have preferred to see the work carried forward by Australian researchers, Burnet’s preference is not so clear.

On May 8\(^{th}\) 1956, Morison wrote to Burnet expressing his regret that ‘something of a mix-up may have developed’, but he felt sure that there would not be too much difficulty ‘in straightening it out’. Morison explained that he had ‘stopped by Singapore in February and spent some days talking to Dr. Hale about his work on Japanese B encephalitis’. While his memory of the discussion was patchy, he explained that ‘the question of extending the work to include the islands which stretch down from the Straits of Malacca to the northern parts of Australia’, came up only ‘in the most general way’, if at all, whilst talking with Hale. ‘The possibility of some aid from the Rockefeller Foundation’, was a matter however that he did remember discussing. Since then, he had received Hale’s ‘formal request’, which was awaiting a response and while no grant had yet been made, the proposal was receiving ‘very serious consideration’\(^{36}\).

\(^{33}\) Letter dated April 26\(^{th}\) 1956 from Gunther to Hale. Series 10/2. Hale’s letter to Gunther is not included in Burnet’s papers, and so it is impossible to say exactly when his letter was written.

\(^{34}\) This piece of communication is not included in the Burnet Papers.

\(^{35}\) Letter dated May 7\(^{th}\) 1956 from Gunther to Burnet. Series 10/2

\(^{36}\) Letter dated May 8\(^{th}\) 1956 from Morison to Burnet. BP Series 10/2
The following day, obviously without receipt of Morison's letter by this stage, Burnet wrote to Hale with an apology, and outlined, in what could be perceived as an over-justification, his reasons for the submission of his proposal. It was pointed out that the significance of the New Guinea region was realised following discussions with Telford Work in India. There was also a 'continuing interest in MVE and its relation to Jap. B with the strong impression that New Guinea may well be the enzootic area of MVE from which it spreads south'. Burnet's preference was 'to defer the start of the active work to the beginning of 1957 when McLean' returned, as he was 'now well trained in this general field including mosquito handling'. It was thought that New Guinea would be a 'very appropriate niche' for McLean. Burnet did not object to Hale's team visiting the area in 1956, as he thought this would enable a 'useful reconnaissance' and perhaps 'provide pointers as to what should be looked for in appropriate parts of Indonesia between New Guinea and Malaya'. These areas, he said, would be more relevant to Hale's Malayan problems than New Guinea, just as the New Guinea problems seemed likely to be specially important in relation to what Burnet described as 'our own "baby" M.V.E.'. There was also the fact that Burnet's team had collaborated in a small way with 'the recent Sydney University-Nuffield Foundation expeditions'. In summary, there was an interest in the 'general medical "exploration" of New Guinea at present'. Burnet expressed the view that he was prepared to co-operate with Hale providing that the Territory of New Guinea was Australia's responsibility from 1957 onwards. But while this gives the impression that Burnet preferred that research in New Guinea should be Australia's responsibility, at least from 1957 onwards, as did Gunther, he also stated that he would be prepared to

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37 Letter dated May 9th 1956 from Burnet to Dr Hale, Singapore. Series 10/2. Burnet wrote, 'I am sorry that our plans in regard to New Guinea should both have been initiated almost simultaneously'.

38 By July 1955, McLean had returned from a year in America on a Rockefeller Fellowship and had won the Harrison Watson Studentship at Clare College, Cambridge (The Director's Thirty-Seventh Annual Report to the Board of the Walter and Eliza Hall Institute of Medical Research, July 1955 p6 (Series 8/42).

39 The studies in the Highlands showed that herpes anti-bodies were found in 100% of the sample tested from the Highlands. This comment is interesting for the current thesis, as it indicates that work had already been carried out in the Highlands, if not directly in the region where kuru occurred. Why herpes antibodies should have been so widespread in the Highlands at this time is unexplained, but herpes virus is one of the viruses forbidden entry into Australia in 1933 by Cumpston. Burnet's trip to the UK and America was made possible with the help of the Nuffield Foundation (The Director's Thirty-Seventh Annual Report to the Board of the Walter and Eliza Hall Institute of Medical Research, July 1956. p6. Series 8/42). In America, Burnet also met with W. M. Stanley, University of California to discuss 'the new treatise on viruses', which is referred to in Chapter One of the current work. The main purpose of the visit to England was to 'take part in a Royal Society discussion on Immunological Tolerance' (March 8th 1956), and to take part in a CIBA symposium on the 'physics and chemistry of viruses'.

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withdraw from the project if Morison thought it wise, due to his ‘wider picture’ of events.

The question is what was Morison’s wider picture and was Burnet’s willingness to step aside in 1956 related to his withdrawal from the study of kuru in 1957? It would be surprising if the mention of withdrawal one year and actual withdrawal the following year are not in some way connected in light of the confusion that is said to have developed in kuru research. And what is to be made of the coincidence of the two proposals submitted almost simultaneously? Coincidences seem to litter the kuru landscape, but this is not the kuru landscape, yet. It is work proposed on a group of diseases which include dengue, Japanese B encephalitis, yellow fever, and MVE, all of which were closely associated diseases and of interest to military medical schools during the nineteen fifties. Here, the boundary between the negotiations in 1956 and the study of kuru in 1957 becomes blurred because the controversy that developed in 1956 about who should take the proposal for research in New Guinea forward seems to have been repeated the following year.

To shed some light on this issue we need to take into consideration Gunther’s comment about his preference for Australians taking the study of viral diseases in New Guinea forward, remembering that it was made in relation to Group B diseases, not kuru. The comment needs to be compared with a similar comment made in the ‘Leading Article’ on kuru published in the Medical Journal of Australia in November 1957, the relevant part of which reads:

Some regret may be felt that the work which originated and was developing in Australian hands should be diverted overseas, when it could have been done at least as effectively by Australian investigators. This feeling is natural enough. However, perhaps we should not be too sensitive about this, lest we be suspected of Chauvinism …. The important thing is, by whatever means, to unravel the mystery of this devastating syndrome (Anonymous 1957:765).

The similarity between what was said in 1956 relating to MVE and what was said in 1957 relating to kuru is striking. It could be argued that Australian authorities would have preferred that both MVE and kuru research were coordinated and conducted by Australian researchers. I would argue though, that the words in the article published in the MJA are significant in that they can be seen as a point of contact between MVE
research and kuru research. When kuru was first described in the correspondence on February 4th 1957, it was simply described as the Okapa outbreak of encephalitis and Burnet wrote to Gunther saying that he had been in close touch with Anderson ‘in regard to the possibility of his undertaking an epidemiological investigation’⁴⁰. This suggests the possibility of a direct relationship between the work on MVE conducted in the previous five years by Anderson, Burnet, Smadel and possibly McLean, the proposed plans in 1956 and the investigation of kuru.

The article in the journal in 1957 gives an impression that Gajdusek appeared as if out of thin air and belies the complexity of the negotiations in 1956, for it begins by saying:

... at the beginning of 1957, tentative arrangements were made for the Walter and Eliza Hall Institute of Medical Research to cooperate in the investigation of kuru with officers of the Administrative Medical Service, of whom V. Zigas in particular had taken a great interest in the syndrome. The Hall Institute was, of course, particularly attracted to the investigation by the possibility accepted at that stage that kuru was a post encephalitidic manifestation. However, at this point a specially qualified American investigator, D. C. Gajdusek, came on the scene and the work was spread to include American laboratories (Anonymous 1957:765).

The article overlooks the previous collaboration between Smadel and Burnet, and the fact that Gajdusek had been working with Burnet up until the time he went to New Guinea. It also overlooks the fact that Gajdusek and Smadel had worked together since 1951 at Walter Reed Army Medical School, Hale, Audy and Smadel were aware of each other’s research and Burnet, Smadel and Morison had agreed on the need for research to be conducted in New Guinea in 1956. Given these associations, why could not a suitable arrangement be made, where all members would have been clear about who would conduct and coordinate the proposed research in New Guinea, which originated as a study of encephalitides and ended up as a study of an encephalopathy?

And why was it problematic that Gajdusek was sending specimens to America during 1957, when virus specimens had moved between Burnet’s and Smadel’s laboratories during the earlier years? It is true that Burnet wrote to the editor of the journal on

⁴⁰ Letter from Burnet to Gunther dated February 4th 1957. BP Series 10/4
November 8th 1957 saying that it would not hurt to hint that 'it is a pity this investigation was diverted to America instead of being done by our own people'. As the end of the article suggests however, the main issue seems to have been to unravel the mystery of kuru regardless of who carried forward the research. In a letter he wrote to Dr Roy Scrugg, Director of Public Health in the Territory of New Guinea on March 26th 1957, Burnet said that the main thing was 'to get the investigation done as well as possible' (quoted in Sexton 1991:170). This is not to discount that he noted his preference was for the investigation to be an Australian affair, but he also remarked that Gajdusek had 'certainly been most conscientious' in keeping the Institute informed of developments and that he was not unaware

... that Gajdusek has a tempestuous enthusiasm that may make him feel that he is doing both the Administration and the institute a service in tackling the kuru problem and that the value of that service is more than enough to counterbalance the effect of his somewhat unorthodox intrusion into the problem (quoted in Sexton 1991:170).

Christopher Sexton has written that 'it is an interesting reflection on Burnet's capacity for objectivity and professionalism over personal politics that, notwithstanding his initial annoyance at Gajdusek's field-pinchng manoeuvre, he acquiesced in Gajdusek taking charge of the kuru investigations' (Sexton 1991:170). My interpretation is slightly different in that Burnet has noted in his letter to Hale the previous year that he would be willing to withdraw given Morison's wider view of events.

My interpretation comes also from the fact that Burnet noted in his letter to Scrugg that he would cooperate with the Administration at their request. This implies that Gajdusek was acting with the authority of the Administration. Gunther's view contradicts this as it was his view that he found Gajdusek's intrusive behaviour unethical because he had only been given permission to conduct child studies (Gunther 1977). Furthermore, Gunther expressed his preference in 1956 that he would prefer the research in New Guinea to be carried forward by Australian researchers. This raises a question. If Gajdusek did not have Gunther's approval, on whose authority was he acting? As I will show in a moment, Burnet was cooperating with Dr

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41 In Farquhar & Gajdusek 1981: 236.
Tom Rivers, Medical Director of the National Foundation for Infantile Paralysis and with Smadel, thus the role of the NIH and the NFIP should not be underestimated.

There seems to have been a general reluctance to talk openly about the kuru investigation. ‘The less said the better’, Robertson’s comment made in October, is but one example. Another example is the Director’s Annual Report to the Board of the Walter and Eliza Hall Institute for July 1957, which was brief in its description of the kuru project. The report noted that the Institute had ‘taken a minor part in the investigation of a serious disease of the nervous system observed in natives in a restricted area of the Highlands of New Guinea and known as “Kuru”’. Fieldwork had ‘been in the hands of the Administration team for whom we have acted as a base for supplies and special tests’ (p. 6). Under the miscellaneous section of the Report on page 24, it states:

Some preliminary enquiries were made by Dr. Anderson and, after investigations on behalf of the Administration by Drs. Zigas and Gajdusek had been under way for some time, he made a visit to the area for consultation with them. The condition is one of extreme interest medically and, because of the tradition that sorcery is its invariable cause, of considerable importance from the administrative angle. … The Institute staff are co-operating with the field investigators in carrying out or arranging for laboratory tests on specimens sent down from New Guinea. As the Institute has no primary responsibility in the investigations no attempt will be made to discuss the problem in any more detail.\footnote{The Director’s Thirty-Eighth Annual Report to the Board of the Walter and Eliza Hall Institute of Medical Research, July 1957. p. 24. BP Series 8/42.}

Burnet explained to Rivers in a letter written on May 27th 1957 that the main problem regarding Gajdusek’s involvement was that the Territory Administration might be seen by the international community and the United Nations to have been slow to act on an important health problem. This, however, was not the case, as arrangements were being made for Anderson to investigate the disease. Nevertheless, there was the possibility of a perception of negligence in not investigating kuru sooner.

It is the case that in his letter to Rivers, Burnet mentioned that Gajdusek had no business in ‘that field at all’ (meaning kuru). He went on to say however that things had now settled down, everything was in order, the Walter and Eliza Hall Institute
were acting as a base for any laboratory work and full administrative support was being given to Gajdusek and Zigas. Gajdusek, in Burnet’s opinion, had ‘behaved admirably in co-operation with the clinical man, Dr. Zigas, who was seconded by the Administration to work with us’\textsuperscript{43}. Burnet had enjoyed having Gajdusek around for the previous eighteen months and had found it interesting, especially the recent kuru period which was ‘the most interesting of all!!!’, and let Rivers know that he had received a cheque that had been sent for Gajdusek from the Polio Foundation and awaited Gajdusek’s ‘instructions on how to act’\textsuperscript{44}. This suggests that Burnet was working cooperatively with Smadel and with the NFIP, which is the institution that supported Gajdusek while he worked with Burnet prior to leaving for New Guinea to begin the kuru study\textsuperscript{45}.

In June, Gajdusek wrote to Burnet and Anderson thanking them for getting the money from the NFIP to him. As well as the $1000 ‘extension grant’ for the kuru project, the NFIP provided an additional $1000 to cover the research at the WEHI conducted by Gajdusek while working there in 1956. Gajdusek was ‘deeply grateful’ to Burnet and thanked him for handling ‘all the administrative details’ with which he had ‘been saddled’\textsuperscript{46}. The money arrived in New Guinea passed on through Ian Wood, who took care of the matter in Burnet’s absence while he was overseas\textsuperscript{47}. Burnet made a similar apology in a letter he wrote to Scrapp in April, which closed by saying: ‘I am sorry for all the trouble that you have been let in for’\textsuperscript{48}. The question is, was the trouble

\textsuperscript{43} Letter from Burnet to Rivers dated May 27\textsuperscript{th} 1957. BP Series 10/17
\textsuperscript{44} Letter from Burnet to Rivers dated May 27\textsuperscript{th} 1957. BP Series 10/17. Gajdusek’s fellowship with the NFIP had expired by this time and Smadel attempted to obtain further funds. Rivers phoned Smadel on April 9\textsuperscript{th} to discuss the finances having heard about Gajdusek’s situation in the territory (in Farquhar & Gajdusek 1981:40). Smadel was pleased that the medical director was able to use the administrative authority vested in him to make available certain funds to take care of Gajdusek (1981:40). After giving his assurance that negotiations were in progress for Gajdusek’s employment at the NIH, Smadel suggested that Rivers ‘allocate something between $700 and $1000’. This would provide support for several months to enable the work to continue on what was described as ‘... the new neurological disease (kuru) among the New Guinea cannibals’, as well as provide enough money ‘for passage back to Washington’ (1981:40). ‘It would be a good idea’, Smadel wrote, ‘to send the money and a covering note to Sir MacFarlane Burnet at Melbourne ... [as] Burnet is well aware of Gajdusek’s casualness so will probably not be surprised at such an arrangement’. Smadel understands that, Burnet ‘can be expected to find Gajdusek to deliver the money’ (1981:41). During the 1950s, Rivers headed the Virus Research Committee of the National Foundation of Infantile Paralysis (Hooper 2000:206).
\textsuperscript{45} The Director’s Thirty-Seventh Annual Report to the Board of the Walter and Eliza Hall Institute of Medical Research, July 1956. p7. Series 8/42.
\textsuperscript{46} Letter from Gajdusek to Burnet and Anderson dated June 4\textsuperscript{th} 1957 in Gajdusek 1975:113.
\textsuperscript{47} Ian Wood is the author of the paper which beautifully summarises the work on MVE and X disease outlined in the previous chapter.
\textsuperscript{48} Letter dated April 5\textsuperscript{th} 1957 in Farquhar & Gajdusek 1981:32.
referred to related to Gajdusek ostensibly taking over the study from Anderson or does the reference to ‘trouble’ relate to an event that occurred prior to this, which might have created the disease?

In contrast to Burnet’s reason as to why an American scientist’s intrusion into the field was problematic (because the Administration might be perceived to have been negligent in not acting on the problem sooner), Gunther’s reason for being upset by Gajdusek’s involvement is somewhat different. For Gunther, the main issue was that by Gajdusek stepping into the project there was a potential to alienate Burnet and the Walter and Eliza Hall Institutes’ goodwill from the Administration of the Territory (in Farquhar & Gajdusek 1981:42). On April 16th he wrote to Gajdusek saying that while he could not blame him for wishing to be part of the research, he just wished he had gone about it differently. It was most unfortunate that he had bypassed Gunther and inserted himself into a situation he knew was reserved for Burnet’s supervision. As Gunther explained to Gajdusek, ‘You must be well aware that whilst we had to do something, the urgency was not such that we couldn’t wait a few weeks’ (1981: 42). Gunther felt sure that when the discord settled, Gajdusek would agree that all he had written about his actions would be seen as justified under the circumstances and he awaited further advice from Burnet in the meantime. Burnet’s letter to Rivers, written one month later, reveals however that Gajdusek behaved admirably, at least in cooperation with Zigas once the discord had settled. This is not to deny that both Gunther and Burnet might have preferred Australian scientists involved, but it is because of the different nuances in the accounts that make it difficult to establish exactly what Burnet’s preference was.

In a letter written to Gunther in mid April 1957, Burnet noted that Gajdusek had told him that his main reason for ‘going again to New Guinea was to find centres for subsequent child study projects which he expected to develop with support from one or another foundation (e.g., Ford) when he returned to the U.S.’. It was suggested that the two men had ‘parted on excellent terms’ and Burnet thought Gajdusek particularly well suited to carry out the investigation. The letter contains personal information about Gajdusek, which Burnet hoped would be helpful in dealing with

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49 Letter from Burnet to Gunther in Farquhar & Gajdusek 1981:41-42.
someone who was quite unique in his experience and he guessed also in Gunther’s. Unique Gajdusek might have been, but according to the correspondence of Lois Larkin, who worked at the Institute with Burnet and Gajdusek before he left to begin the kuru project, rather than Burnet and Gajdusek having parted on excellent terms, something had marred his stay at the institute. On March 13th she wrote that this was something she ‘would have given anything to prevent’. By way of consolation she pointed out that he was ‘not the only one in the dark about their plans’. I turn to Lois Larkin’s letters for another voice that might provide some clues about these plans.

In April 1957, Larkin wrote that ‘there seems to be a hell of a mix-up with the arrangements about the kuru investigations’ (Larkin in Farquhar & Gajdusek 1981: 298). It is important to note that Larkin’s comment about a mix-up refer specifically to specimens of CSF being sent to WEHI, rather than having the tests carried out in Port Moresby, as arranged (1981:298). Larkin was concerned that Burnet considered that Gajdusek was ‘cutting across Moresby in sending the CSF’s down here’. While this comment could be read as referring only to the correct protocol in sending specimens out of N.G. to Australia, another comment made by Larkin – ‘the politics and petty jealousies behind the New Guinea work are very complicated and I cannot make head nor tail of the whole mess’ – indicates that the mix-up extended to the whole of the New Guinea work. Attention by early 1957 had turned towards the study of kuru and the study of child growth and development, rather than MVE. The question though, is how closely associated was this mix-up with the one described the previous year?

There were two studies referred to by Larkin, one of which was for child studies and the other was described as ‘the peculiar illnesses among the natives’. Larkin had

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50 The Lois Larkin letters were written to Gajdusek during 1957 and appear as an Appendix to the Early Letters and Field-Notes from the Collection of Carleton D. Gajdusek (Farquhar & Gajdusek eds. 1981). Between March 12th and March 16th Larkin wrote four letters, which were all posted together on the sixteenth. When Gajdusek left the institute for New Guinea in February 1957 to study kuru, Larkin continued his work related to the auto-immune complement fixation (AICF) test. In the middle of 1957 she visited New Guinea for what can be described as a working holiday and throughout 1957 acted as a liaison between Gajdusek and other collaborating scientists.

51 A letter written in 1968 by Burnet supports the idea that the kuru investigation was related to the negotiations. He notes that he has been ‘continually’ in touch with the subject of kuru since mid-1956, but has not taken part in the elucidation of the disease. Letter dated August 23rd 1968 from Burnet to Gajdusek. BP Series 10/13.
discreetly tried to find out what was behind the silence and noted that the ‘security in high places’ around the Institute defied even her ‘spy system’ (in Farquhar & Gajdusek 1981: 297). She understood that Ian (Mackay? Wood? Burnet?) was the only other person who knew anything about the plan for the child study besides Anderson, Burnet and Gunther, and noted that this was a ‘little out of their field, don’t you think?’\textsuperscript{52}. Both studies were ‘behind the curtain’ and it was suggested that the secrecy was having a great effect on morale at the Institute; the place was described as stagnant. The institute might have been stagnant but there was much news to report to Gajdusek. In her letter written on March 12\textsuperscript{th} 1957, Larkin opened by saying: ‘So much has been happening that I hardly know where to begin’ (in Farquhar & Gajdusek 1981: 296).

Three problems had been discussed at the Institute since Gajdusek had left. These included, the role and mechanism of antibody production in the body, the problem of Macroglobulinaemia, and the problem of recombination using \textit{Vaccinia virus}\textsuperscript{53}. Macroglobulins are proteins of high molecular weight (around 1 million mw) that are observed in the blood in a number of diseases (Miller & Keane 1987:732). Macroglobulinaemia is not a sign specific to kuru although it was found in some patients suffering from the disease. The problem of recombination is a little more complex to interpret in terms of its possible relationship with the investigation of kuru, but the prospect of \textit{Vaccinia} virus fragments recombining with another agents’ genetic material was worrying for scientists during this period\textsuperscript{54}. The possible relevance of recombination for the study of kuru is that it involves animal virus. Of greater relevance is that Larkin also tells Gajdusek that she has heard that kuru along with Foot and Mouth disease was discussed at a lunch meeting\textsuperscript{55}. This means that as early as March 1957, an informal meeting had taken place where it seems possible that kuru was viewed as being associated with animal disease. It is perhaps not so surprising that kuru was discussed within this context considering that animal disease

\textsuperscript{52} \textit{ibid} p 296. In parenthesis, Larkin wrote, (who “they” is I am not quite sure, but I think it means Dr Gunther, FMB [Burnet] and SG [Anderson]).

\textsuperscript{53} This is another of the viruses forbidden entry by Cumpston in 1933 along with louping ill and herpes virus because of the possibility of post vaccinal encephalitis.

\textsuperscript{54} In 1952 it was found that when more than one strain of an agent (any agent) was used together with another, recombination could occur (Anonymous article published in \textit{MJA} February 9\textsuperscript{th} 1952).

\textsuperscript{55} At the time, foot and mouth was thought to be an enterovirus, not unlike poliomyelitis virus. These were the simplest of all viruses made up of protein and RNA (ribonucleic acid) only arranged into a crystal lattice and spherical in shape (Burnet & Stanley 1959:8-9).
has always been associated with prion research since research began on scrapie, but this aspect of the kuru story is rarely given prominence. And neither is the secrecy that is said to have surrounded the two studies to which Larkin referred. There are though, not two but three sets of studies to consider: the child study, the kuru study, and the studies that had been negotiated in 1956 related to Group B diseases like MVE. Here, I attempt to clarify what was said about these studies.

Burnet mentions a proposal for child studies in his letter to Scragg written in April. He looked forward to receiving Scragg’s reaction to a memorandum he had sent to Canberra a few weeks earlier ‘in relation to Dr Gunther’s report on the possibilities for research on disease in infants and children’. He suggested that Anderson would make a visit, possibly in May, which would allow him to discuss the implications of Gunther’s report, visit the areas where sentinel fowl had been placed related to the studies of MVE, and to see any cases of MVE that had occurred in Port Moresby.\(^5^6\)

Gunther refers to a proposal for child studies in his 1977 Draft paper, while talking about the events that led up to him authorising permission for Gajdusek to come to New Guinea to work in 1957 (for the child studies only). He explains that in August 1956 he met Gajdusek when he visited Burnet’s laboratories in Melbourne to further the formation of a programme on child health. He writes, ‘we badly needed to know more about child growth and morbidity, not just physical development and sickness, but psychological development; how concepts were developed, with the establishment of some kind of time scale as set down by Jean Piaget’ (Gunther 1977: 5-6). As he went on to explain, Gajdusek had previously been to New Guinea with an American Anthropology research group who worked in New Britain and expressed a desire to come back and work in New Guinea explaining that he would not need recompense. Gunther told him to let him know when he was to arrive (p 6). Sometime between February 12\(^{th}\) 1957 and March 8\(^{th}\) Gunther was asked whether a Permit should be issued and assuming that this followed his discussions in August the previous year, he had no hesitation in supporting the Permit, which he understood would be for the child studies (p 9). One thing that is difficult to understand here is how Gunther could have met Gajdusek in August, because on September 28\(^{th}\) 1956 Gajdusek wrote to

\(^{5^6}\) Letter dated April 5\(^{th}\) 1957 in Farquhar & Gajdusek 1981:32.
Smadel saying that he had just returned from a three and a half month trip to New Britain etc\(^5\). It is possible that Gajdusek had been back at the institute for more than one month before writing to Smadel, or that Gunther was mistaken about the timing of the discussions, but given the inconsistencies in the accounts that have been written a critical attitude seems warranted. The point is that Gajdusek, Burnet, Anderson, French and Smadel were all working on the same studies and seemed to work collaboratively until, for some reason in early 1957, things are said to have gone awry.

Gunther drew a distinction between the three studies. I have already shown how he differentiated between kuru and the child study. He also notes in his paper that ‘quite separately from kuru Sir MacFarlane had already proposed and had offered to organise medical research in a meaningful way in Papua and New Guinea’ (1977: 9). This seems to refer to the study of Group B diseases on which the negotiations focussed in 1956.

Gajdusek also describes three studies in a journal account written in March 1957, having just found out about kuru, ostensibly, from Scragg in Port Moresby. In this case, the three studies are much more difficult to distinguish. The relevant part of the account reads as follows:

\(\text{I arrived in Melbourne filled with enthusiasm for the Territories, dug up encephalitis from here, which Anderson had filed away, and pressed my point of the need of studying arthropod-borne virus diseases here! I urged and discussed my every find to disinterested ears and started planning my own trip here. Six months later [March 1957] I was presented with the fact of Sir Mac’s and Anderson’s extensive correspondence with the Territory and plans for}

\(^5\) Gajdusek opened his letter of September 28\(^{th}\) by saying, ‘I have recently returned from New Britain, the Trobriand Islands, Samarai, New Guinea and Papua and Furguson Island in the D’Entrecasteau Archipeligo’, following ‘three and one-half months of field work’. The work had been ‘immensely profitable’. Specimens had been collected from 600 aboriginal children of the Cape York Peninsula, which were to be tested for ‘measles CF and neut antibodies’. The specimens were ‘ideal for studies of duration of antibody after acute infection in the absence of reinfection, since measles disappeared from the population completely after each epidemic’. The specimens could also be used for all other seroepidemiology, specifically ‘dengue, Jap B, MVE’, which came to his mind immediately (Gajdusek 1975:40). Anderson and French were ‘rushing through the MVE problem, but not other neurotropes’ and had ‘a strain of MVE (it was not Jap B) isolated from the brain of a sick native at Port Moresby which was sent to us here in glycerol’ (1975:41). ‘Serological screening of their serum collection locates MVE antibody in some population groups, but not all, scattered in Papua’. At this time Smadel had moved from the position of Director of Virus and Rickettsial disease at Walter Reed Army Medical School to the position of Associate Director of Health at the National Institute of Health, Bethesda.
such studies here. All had been quietly done without even bothering to tell me and I was presented with “brilliant new ideas” which six months earlier I had pumped into their heads.

This account seems to link the study of Group B diseases and kuru as the diary entry continues by saying,

I went on with my plans and my trip, never being offered the chance to take part in theirs and treated as an intruder into “their territory” ... (in original) as Sir Mac had treated Hale of Singapore with a polite “hands off” ultimatum. They dared not suggest “hands off” to me – the author of their scheme, and I proceeded to ignore both their plagiarism and their discomfort (Gajdusek 1996: 77).

The account then reverts back to when Gajdusek returned to Melbourne in 1956, when he ‘again shared with Sir Mac and Anderson all their interests’ and told them of his work by emphasising ‘the need for child growth and development studies and for correlation of these with ethnology and cultural anthropology’ (underline in original). This line of investigation, he writes, was ‘totally my own approach’ (pp 77-78). This account shows how the three studies - kuru, child studies, and Group B arthropod diseases - are difficult to distinguish.

Recall that Gajdusek knew about kuru. This means that, whether or not there was secrecy about the kuru project, or the child study project (if the two can be differentiated), there are too many inconsistencies in the accounts and the so-called conflict that developed calls for reinterpretation. Gajdusek’s version of events is not consistent with that which emerges from the correspondence in the Burnet Papers regarding Burnet’s ostensible ultimatum to Hale. As I have shown, Burnet was willing to compromise, and even withdraw should Morison feel it necessary, even though he said that the region should be Australia’s responsibility post 1956. The significant point is that it was Morison at Rockefeller who said that the matter should not need much straightening. It appears that straightening the matter was more difficult than expected judging by the situation that developed in 1957.

Given that a separation has been made between the child study and the kuru study in the recollections of Larkin, of Gunther, and in Burnet’s letter to Scrugg mentioned above, it is interesting that research on kuru has been framed within the context of
studies of child growth and development. Clarence Gibbs, who conducted many transmission studies using kuru material passaged in primates, has written about the naming of the kuru studies in the following way:

[S]low virus investigations at NIH [National Institutes of Health], under the direction of D. Carleton Gajdusek, evolved in an unforeseeable and circuitous manner. It was established within the Division of Collaborative and Field Research, National Institute of Neurological Diseases and Blindness (NINDB), as part of Gajdusek’s laboratory of “Child Growth and Development and Disease Pattern in Primitive Cultures” and was designated as the “study of slow, latent and temperate virus infections” (Gibbs 1992: 55).

The process described by Gibbs began following Gajdusek’s return to America in April 1958. The relevant questions are why was it said that the study of kuru and the child studies were secret and was this related to the production and/or use of vaccine or sera?

I began this chapter by showing that there was a collaborative relationship between Burnet and Smadel during the early part of the nineteen fifties. I then showed that Burnet, Smadel and Morison agreed on the need for a five-year plan of research in New Guinea. This followed Burnet’s discussion with Telford Work in Poona where the importance of the New Guinea region was realised. I now want to bring the chapter to a close with a focus on events towards the end of 1957. Here it is important to recall that kuru and hemorrhagic fever were likened to each other in that both diseases proved difficult to understand.

Towards the end of 1957, Burnet corresponded with Telford Work about a variant of RSSE, known as Kyasanur Forest disease that was occurring in India. Burnet sent the information to Gajdusek, who wrote back to Burnet on September 23rd 1957 thanking him for the information from Work in Poona. When Gajdusek left New Guinea he travelled to Poona to visit Work where they discussed kuru and Work thought the story a fascinating one. This letter was copied to Robert S. Morison and therefore shows the collaboration between Morison, Work, Gajdusek, and Smadel.

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60 Letter from Work to Smadel dated March 20th 1958 in Gajdusek 1975:401-402.
Perhaps it is significant that Burnet was not sent a copy of this correspondence. However given that Burnet had been in touch with Work only a few months before, and that the initial prompt for the New Guinea research had come from Work in 1956, it would not seem unreasonable to assume that Burnet was involved in all of the negotiations and plans. Nor would it seem unreasonable to assume that he was aware that kuru specimens were being sent to America towards the end of 1957, which, it has been said, was part of the reason for his withdrawal from the kuru project. This interpretation is consistent with the idea, expressed by Burnet in 1956 that he was prepared to withdraw should Morison feel it necessary.

Work wrote to Smadel on March 20th 1958 from the Virus Research Centre at 30A Wellesly Road, Post Office Box 11, Poona 1, India, indicating that Gajdusek was about to leave for Moscow\textsuperscript{61}. This letter is important as it shows how Gajdusek was able to operate outside of normal channels. The visit to Russia was arranged through Drs Chumakov and Smorodintsev. Where others would have been required to take a Russian plane to somewhere in Western Europe, a more direct route via Kabul and Tashkent had been arranged to allow Gajdusek to disembark in order to see the places where hemorrhagic fever and Russian Spring Summer encephalitis occurred. Gajdusek wrote to Smadel from Moscow (Gajdusek 1975: 399). Chumakov was trying to obtain a few books, which he and Telford Work required.

Gajdusek’s ability to operate outside of normal channels was mentioned in a letter written to Scrapp in April 1958, in which Smadel remarked:

\begin{quote}
Since you also know Carleton well, you will not be surprised by his numerous delays enroute or his individual capacity to do things which could be arranged only with difficulty through official channels\textsuperscript{62}.
\end{quote}

Given this ability, the pressing question becomes did the kuru project also operate outside of normal channels?

Daniel and Robbins, in a monograph called \textit{Poliomyelitis}, note that for a number of

\textsuperscript{61} Letter from Work to Smadel dated March 20\textsuperscript{th} 1958 in Gajdusek 1975:401-402.

\textsuperscript{62} Letter from Smadel to Scrapp dated April 3\textsuperscript{rd} 1958 in Gajdusek 1975:408.
years during the nineteen fifties, a group of polio investigators from the United States and U.S.S.R. ‘met regularly to discuss progress and exchange information’. The story, they say, ‘of Albert Sabin’s collaboration with Russian investigators at the height of the Cold War is a fascinating one’ (1997:16)\textsuperscript{63}. Included in the list of names of the Russian workers are, Chumakov, Soloviev, Zhadnov, Voroshilova, Smoridentsev, and Drosdov, who were all ‘important figures in the field of polio research’ (pp 16-17). As Daniel and Robbins point out, this was not the only joint Russian-American collaboration ‘but it was one of the early ones’. Clearly, Gajdusek was also familiar with some of the scientists named by Daniel and Robbins. These scientists conducted research on hemorrhagic fever as well as polio. In 1953, while a Captain in the United States Army, Gajdusek authored the book entitled \textit{Acute Infectious Hemorrhagic Fevers and Mycotoxicoses in the Union of Soviet Socialist Republics} (Gajdusek 1953). I am suggesting that this earlier work needs to be seriously considered when interpreting the kuru project, not only because kuru was likened to hemorrhagic fever, but also because collaborating with Russian scientists would not have been seen as a particularly endearing characteristic given the Post War tensions that existed. Some Australian colleagues, because of his associations with Russian researchers, might have been suspicious of Gajdusek. This might explain in part why the negotiations were not straightforward in 1956 and why the details of the kuru project have also been difficult to interpret.

There were however Australian scientists who had also been to visit Russian researchers. In June 1956 Gajdusek wrote to Smadel saying, ‘from reports I have of recent Australian visitors, the Russians are quite willing to take them wherever they have sense enough to ask to go’ (Gajdusek 1975:39). On December 29\textsuperscript{th}, Gajdusek sent a small pamphlet to Smadel entitled \textit{Spring-Summer Tick Encephalitis} by N. N. Gorchakovskaia, and noted that it was available in the Russian language distribution houses in Australia for about three American cents. That ‘so much of this sort of stuff, and stuff of far higher caliber [was] now rolling off Soviet presses’, Gajdusek found utterly astounding (Gajdusek 1975:46). That there would be Russian literature rolling

\textsuperscript{63} During the late nineteen forties, Frederick Robbins wanted to go to work with Tom Rivers who was forming a special laboratory of the Rockefeller Institute for Medical Research, but as Robbins was the property of the Army rather than the Navy he was not able to do so and went instead to an army laboratory as Head of Bacteriology directing work on virus and rickettsial diseases. Before 1948, Rivers held the rank of Admiral in the Navy (Robbins in Daniel & Robbins 1997:122).
of the presses and available in Australia for three American cents and that Australian
scientists and Russian scientists were collaborating according to this account,
indicates that while tensions might have been high politically, the same was not the
case scientifically. Nevertheless, everybody would not have felt amenable to an
interest in Russian affairs.

This is a different version of events than that which has been described in the some of
the popular versions of the kuru investigation, which have Gajdusek in New Guinea
either on holiday or simply collecting data for child growth studies before returning to
America, and coincidentally becoming aware of kuru from Scragg at Port Moresby.
Recall that Gunther saw the proposed study as a fascinating extension of the earlier
work on MVE, which Burnet referred to, as our ‘baby’. MVE was likened to X
disease, a disease thought to be caused by louping ill virus in the earlier years. Scrapie
was known to be transmissible through louping ill vaccine. Louping ill was likened to
hemorrhagic fever and hemorrhagic fever was likened to kuru.

When Gajdusek arrived in the Eastern Highlands in 1957, his first port of call was the
Experimental Agricultural Station, having met Zigas and Michael Foley, the Assistant
District Officer, at their headquarters just above the airstrip at Kainantu. They
‘immediately laid their plans’, which meant driving for half an hour to the adjacent
Aiyura Valley where the Experimental Station was well staffed and equipped to study
‘the agricultural possibilities of the region’ (Gajdusek 1996: 80-81). In a tale full of
references to animal diseases such as louping ill, foot and mouth and all of the
mosquito and tick borne viruses, this diary entry, published forty years after the event,
needs consideration.

Whether Burnet was fully cooperative with Gajdusek’s collaboration in the kuru
project is difficult to say with confidence. My interpretation of the events would be to
suggest that Burnet was supportive of Gajdusek’s involvement and the assistance
American laboratories could bring to a very complex situation, which from later
documents, we know involved a scrapie-like agent. This is not to say that the source
of the problem had been identified at this time. But the finances obtained by Tom
Rivers at the Polio Foundation in April 1957 did not occur without a great deal of “red
tape” being cut. I argue that this was not an ordinary project, but that it is difficult to see the full implications when reading the kuru story in isolation from its context.

If the idea of vaccine or sera as a mode of transmission for kuru is to be taken seriously it is necessary to examine the institutional context from the perspective of plant, equipment, and personnel during the relevant period, which is the work of the following chapter.

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64 On April 22nd Smadel wrote to Gajdusek letting him know that Rivers’ was now having trouble cutting the red tape. It was ‘the same damn color no matter what the large organization’. Gajdusek was informed that he could ‘be thankful that Tom’ was ‘a good red tape cutter’ (Gajdusek 1975:75).
Chapter 4

Kuru and its institutional context 1955-1964

I have aimed to show that the kuru investigation in 1957 was a project closely associated with the study of Murray Valley encephalitis and that a collaborative relationship existed between Burnet and Smadel throughout the nineteen fifties. Earlier, I aimed to demonstrate the perceived similarities between Murray Valley encephalitis and X disease and between X disease, poliomyelitis and loping ill virus. Here, I propose to examine the situation that existed relative to plant and equipment at two of the principal research institutions in Australia: Commonwealth Serum Laboratory (CSL) and Walter and Eliza Hall Institute (WEHI) during the period 1955 to 1964. At the same time as members of these institutions, along with government officials, grappled with the problems presented by kuru, there were other equally pressing concerns related to the manufacture of safe and effective polio vaccine. Examining problems in this area at this time provides a useful framework within which to read the kuru story. The idea that kuru may have been related to these problems has never been publicly countenanced, but given the close temporal and institutional relationship that existed between the two investigations - kuru and polio - it seems worthwhile looking in this area.

Two useful sources of information in this area are A. H. Brogan’s Committed To Saving Lives: A History of the Commonwealth Serum Laboratories (1990), and the office diaries of the director of the Walter and Eliza Hall Institute from 1961 and 1962. Reading Brogan’s history of CSL enables a picture to emerge of an organisation fraught with tension in the process of re-organisation. The office diaries of Sir MacFarlane Burnet at the Walter and Eliza Hall Institute provide a similar picture in that they amply demonstrate the problems associated with vaccine production generally and provide details about various protocols devised at this time when considering use of polio and other vaccine and sera. By gaining knowledge about the specific problems that existed at the institutions mentioned and the efforts made to
manage the situation during the period in question it will be possible to situate the study of kuru within its institutional context.

Problems related to polio vaccine that existed at CSL during the nineteen fifties were not new in a global sense, although they may have been new to CSL. Similar problems were identified in America in 1955 when what is known as the ‘Cutter Incident’ occurred. This involved a faulty production method of Salk inactivated poliovirus vaccine which resulted in cases of polio. In April 1955 six cases of polio occurred in children who had received Salk vaccine manufactured at the Cutter laboratories. The Cutter product was recalled and yet cases continued to be found in some of those who had been inoculated with product manufactured at other laboratories (Wilson 1967:45). In all, 260 cases of polio occurred in vaccinated persons, of which 59 were of the paralytic type. 126 cases also occurred in family contacts of those inoculated, of which 101 were paralytic. 40 cases were recognised in community contacts, of which 32 were paralytic. 10 deaths occurred, five of which were in persons who had been vaccinated, the other five occurred in contacts of the vaccinated but who themselves had not been given the vaccination. From the eight batches of vaccine made at the Cutter laboratories only two were found to show signs of contamination. These batches had been used to inoculate 120000 people in California and Idaho. As Wilson points out, this gave an attack rate of 94 in 120000 and paralysis in 59 in 120000, or 1 in 2000 (Wilson 1967:45). It was found that the arm used to inject the product was affected more often than not. Wilson attributed the problems in the Cutter incident to the manufacturing process and the inability of formaldehyde to inactivate the agent because it did not penetrate the protein-containing particle (Wilson 1967:287). A similar situation prevails in research associated with prion diseases like kuru in that the agent responsible for the disease also is not inactivated by many of the known chemicals.

Following the cases of vaccine induced polio Eklund, Bell, and Hadlow (1956) identified particles in the contaminated batches of polio vaccine. William Hadlow’s involvement in this field of research is significant because he went on to point out the

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1 A similar finding was mentioned in reports made in England (Anonymous 1952: 191-192) and Australia (McKloskey 1951:618. Lancaster 1954:245) in relation to cases of polio that had occurred as a result of the use of pertussis vaccine given in combination with Diphtheria vaccine.
similarity between kuru and scrapie in an article published in 1959 (Hadlow 1959). This later work has been referred to in the kuru story as the second crucial element involved in unravelling the mystery of kuru (Goodfield 1985). The problems encountered relative to the manufacturing process for polio vaccine at CSL were little different to these problems experienced in America.

Polio and Kuru: Concurrent problems at CSL

Brogan’s history of CSL devotes a chapter to some of the problems associated with the manufacture of polio vaccine during the late nineteen fifties and early sixties\(^2\). It says very little about kuru. What it does say though is significant in that it places the commencement of the study of kuru in 1955 rather than 1957. Brogan remarks that CSL staff during this period forged collaborations with overseas scientists. One such collaboration ‘... which was of great significance was that between Simmons, Graydon and the American D. Carleton Gajdusek’ (Brogan 1990:221). Immediately following his description of the collaboration Brogan goes on to write:

In 1955 whilst a visiting fellow at the Walter and Eliza Hall Institute, Gajdusek began studies of a unique disorder of the central nervous system occurring only among the Fore people of New Guinea (Brogan 1990:221).

The fact that work on kuru, according to this account, began in 1955 is inconsistent with the dominant version of the kuru story. Considering Brogan had worked at CSL in an administrative capacity since 1957 it seems probable that his account is correct. This earlier date would be consistent with the ideas outlined in the previous chapter, notably that the negotiations that took place in 1956 were related to the kuru project, and that Gajdusek knew where he was going and for what purpose when he entered the New Guinea Highlands in 1957. Given the close collaboration that existed between Gajdusek and Simmons at CSL and given that the collaboration existed at the same time as CSL was in need of review relative to its manufacturing processes, questions arise about how closely related the study of polio was to the study of kuru.

\(^2\) Alf Brogan compiled a history of the institution following his retirement from CSL in May 1988. The book was written and supported by Neville McCarthy, CSL director and technical guidance was given in the preparation of the text (Brogan 1990: xiii).
If nothing else, the timing of these two episodes in scientific history coincide suggestively because the study of kuru existed at the same time and in the same institutions that were also grappling with the problems associated with polio vaccine. The problems at CSL from July 1956 onwards became the responsibility of Val Bazeley, who was appointed as Director at this time. Brogan suggests that the period of Bazeley’s directorship between 1956 and 1961 was ‘the most turbulent five years in CSL’s history’ (Brogan 1990:130). Sir Gustav Nossal, in the Foreword to Brogan’s book, commended him for skilfully articulating the tensions that existed between some of the key figures in the organisation’s history (in Brogan 1990: vi). It was perhaps around this time, that the tensions to which Nossal referred were especially evident. I begin by looking at three possible sources of tension that developed during Bazeley’s period as director. The first of these relates to his background and appointment. The second, to the upheaval caused by the re-organisation that he sought to implement. And the third point relates to Bazeley’s modus operandi, which was characterised by a determination to implement changes regardless of the outcome. This resulted in his alienation from a number of staff at CSL and government officials. Bazeley’s style was similar to Gajdusek’s in that both men were able to operate outside of normal channels, which raises questions about the circumstances involved in both scenarios.

Bazeley, a veterinary scientist, began his career at CSL on March 21st 1939, at a time that CSL was engaged in a ‘vital defence industry’ (Brogan 1990:64)³. Notice of the appointment was recorded in the Commonwealth Gazette along with the appointment of Dr C. E. Cook at the School of Public Health and Tropical Medicine, Sydney (Brogan 1990:52). Brogan points out that Bazeley and Cook became important figures during the controversy that arose in the late nineteen-fifties while Bazeley was Director and the methods of production at the organisation were under review. When Bazeley was stood down from his position as Director in 1961, Cook replaced him as Assistant Director. It is not clear from Brogan’s account whether the coincidental

³ Brogan points outs that there are no existing records of the activities of the organisation for the period 1937-8 to 1953-4. One of the reasons might have been because of the need to produce cardboard (Gavin McCarthy personal communication). However it is also relevant to note that ‘the problem of preventive medicine in the second war were even more numerous and urgent than in the first’ and results of the work conducted during this period were ‘largely concealed by the veil of security’ (Keogh 1951: 27).
appointment of Bazeley and Cook in 1939 was significance apart from the fact that both became embroiled in the polio problem some fifteen years later, but the School of Public Health and Tropical Medicine, at which Cook began his career, was the site of the Kuru Committee of Inquiry meeting held in 1959, although Cook was not a participant at the meeting⁴.

It might seem surprising that the Director of an institution charged with the responsibility for producing vaccine and serum for human use was a veterinary scientist. But when taking into account the fact that many of the products produced at this time could not be made without the use of animals, it is not so surprising. Animal vaccines were also made at CSL. The relationship between animal and human health in the area of vaccine research is often overlooked but is critical in understanding how contamination problems can arise. The problems related to the manufacture of polio vaccine came about, at least in part, due to action that Bazeley had taken in 1954 and 1955 when he imported 200 monkeys per month for the polio project (Brogan 1990:119). In all, 2354 monkeys arrived at CSL, and the kidney cells of the monkeys were used to grow the poliovirus. More than one third of this number died before they could be used. Bazeley noted in a 1961 (April 7th) report the problem involved in the manufacture of polio vaccine and the presence of Simian virus 40 (SV40) (Brogan 1990:124). So Bazeley’s expertise as a veterinary scientist was useful in addressing the problem of animal viruses and their potential transmission to man through the process of vaccination. This is particularly relevant when considering the study of kuru because only a few years after the initial investigation in 1957 the disease was associated with a scrapie-like agent by Hadlow (1959), who three years earlier had identified contaminants in batches of polio vaccine. Furthermore, scrapie was also known to be associated with loping ill vaccine. And extending this line a little further, confusion during the early decades of the twentieth century existed as to whether X disease was actually caused by an aberrant form of poliomyelitis virus or loping ill virus. Thus, the complex relationship between X disease, polio, loping ill, and scrapie was something that had been acknowledged and grappled with for two decades by the time Bazeley was appointed Director of CSL in 1956.

⁴ Series 10/6 of the Burnet Papers provides a detailed account of this meeting.
With prion research in mind - which involves animal as well as human research - it is interesting that Bazeley targeted veterinary research for increased funding in the reorganisation of CSL (Brogan 1990:53). Within ten weeks of his appointment as Director on July 4th 1956, he began a thorough reorganisation of the institution (Brogan 1990:130). The details of the changes were circulated at CSL in Staff Circular No 12 dated September 20th 1956, one month before the submission was put before the Board of CSL on October 11th (Brogan 1990:276). In a way this demonstrates Bazeley’s influence over a complex situation. What it also shows is that these changes coincided with the negotiations relative to MVE that took place in 1956, which were the focus of the previous chapter.

One of the reasons for the reorganisation was that up to the time of Bazeley’s appointment as Director, CSL had many different sections. These included an antibiotic section, a biochemistry section, an endocrine section three sections devoted to microbiology, one to virology, another to veterinary and diagnostics procedures and finally a research section. Bazeley sought to place like operations with each other and created five main divisions: Production, Research, Engineering, Consultancy, and Administration (Brogan 1990:131). Here, I want to focus on two of the new divisions - production and consultancy - as Brogan’s account of these areas provides the rationale for the reorganisation, notably the imperative to upgrade plant and equipment.

Within the new production division a completely new development section was created. Before the changes, ‘development work had been additional to and a subsidiary of normal production work’ and as a result of this arrangement ‘the introduction of new biologicals or improved methods was often delayed’ (Brogan 1990:131). It would seem from this account at least, that production problems were real and recognised by September 1956. This does not prove a causal relationship in

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5 Report of the Director-General of Health for the year ended 30th June 1954 (1955 pp 23-27). I noted in the footnote in the previous chapter under the section on yellow fever vaccine, that this vaccine was produced at CSL and had been approved for international use by the WHO. The report does not disclose where the tests took place, but mentions only that the tests showed that ‘the keeping qualities of the vaccine were better than was generally assumed’ (p28). Considering that the negotiations in 1956, mentioned in the previous chapter, were in part aimed at the prevention of the spread of yellow fever, this might indicate that the yellow fever vaccine was not perceived to be as useful in 1956 as it had been in 1954.
terms of the many cases of kuru that were investigated the following year, but it does indicate that problems relative to production and safety apply to the situation in earlier years.

In 1957, following a Cabinet review of the situation at CSL, ‘the obsolete and limited plant and the lack of adequate maintenance and redesign of buildings’ was also noted in a report written by the Board (Brogan 1990:132). The Cabinet review began on April 11th 1957 within a year of the start of Bazeley’s reorganisation (Brogan 1990:132). This means that prior to Bazeley’s appointment as Director in July 1956, CSL obviously had considerable problems in terms of vaccine safety, particularly with reference to animal viruses, and these problems existed simultaneously with the events outlined in the correspondence of the Burnet Papers Series 10/2 whose original title was ‘1956 visit to N. G. Preliminary to Kuru New Guinea Project – 1956’.

The date of the review is significant as it began only a few weeks after Gajdusek’s arrival in New Guinea, and simultaneous with the series of correspondence that flowed between Gunther, Burnet, Gajdusek, Smadel and Rivers at the National Foundation for Infantile Paralysis, as discussed in the previous chapter.

It is important to note the comments made on the Cabinet submission were not directed at Bazeley’s management but rather towards the role of the previous director. Here, it is perhaps best to view the situation that existed as systemic rather than the problem of any one individual, as it was not long before opposition started to mount towards Bazeley’s mode of operation.

Tension developed, in part at least, from the fact that scientists were put in charge of the production process whereas up until this time medical graduates carried out much of the scientific work involved in production. This suggests that there might have been problems in terms of safety created by the previous arrangement. Within the new consultancy division some of the senior professional staff were ‘suddenly divorced from line functions’, which led to feelings of resentment by some of the medical officers who had previously held production responsibilities. Others were

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6 This was the name of the file of documents when it was accessed by the Australian Science Archives Project (AUSTEHC), having previously been housed in the Medical History Unit, Brownless Medical Library, Melbourne.
incorporated into the new division as consultants. Don Oxer became Principal Veterinary Consultant and Dr Wally Hurst, Gilbert Anderson and Roy Simmons were incorporated into the re-organised consultancy division (Brogan 1990:131). Simmons is relevant in that he collaborated with Gajdusek on the study of kuru.

It is not surprising that Bazeley’s reorganisation brought with it a sense of dis-ease. The situation in which he found himself was highly complex and fraught with difficulty. Along with the creation of new divisions, new staff appointments, and a realisation that plant and equipment was inadequate to do the job required, another factor that contributed to the tension was the employment, as consultant, of Frank Partridge, the shortly to retire head of General Motors. Bazeley’s appointment of Partridge brought about a fierce interplay between Bazeley and the Board and the event exemplifies Bazeley’s style, which can be characterised by two features: a determination and an ability to seemingly work outside of normal channels.

The tension associated with the negotiation of the contract of Partridge in November 1957 revolved around Partridge’s salary and the length of his contract. Following much correspondence on the subject, the Board was “unable to accept as convincing the explanations offered by the director” as to the reasons for the appointment or why Bazeley had ‘recommended an extension of Partridge’s term for a further two months’ (Brogan 1990:134). Finally, the Board summarily terminated the consultancy on 16th April 1958 after a term of three months and ten days. Bazeley’s determination in appointing Partridge regardless of the opinion of others is but another of the reasons why he was alienated from CSL, the Department and the Board, who lost faith and patience. Brogan describes this event as a ‘storm in a teacup’ (1990:134). I am not so sure, as it reflects a pattern of behaviours that made it difficult for some officials to work with Bazeley. Bazeley’s situation can be compared with Gajdusek’s, as he too, showed a similar determination to continue researching kuru in 1957 despite being asked to leave the area because the study had been allocated to Dr S. G. Anderson.

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7 The Board wanted to limit the appointment to three months with a fee of £1000. Bazeley argued for a 3-6 contract with a fee of £1250. The Board decided that everyone concerned should sit down and discuss the matter with Partridge. Those included in the discussion were representatives of CSL, departmental officers, Board members, and Partridge. Bazeley went ahead independently and employed Partridge with a fee of £1250 (Brogan 1990:134).
Another similarity between Bazeley and Gajdusek relates to their association with the National Foundation for Infantile Paralysis. Brogan notes that Bazeley felt a debt of gratitude to the Foundation. Gajdusek, no doubt, also felt a similar debt to the NFIP as it was Rivers who supported the ostensibly, ‘unsalaried’ year of investigation in New Guinea in 1957.

It would be surprising if the two sets of events - polio and kuru - were not in some way connected. If they are not, the synchronicities are remarkable. Burnet’s proposal to study viral diseases in New Guinea in 1956 simultaneous with Bazeley’s appointment as Director at CSL, the outmoded plant and equipment and Gajdusek’s collaboration with Roy Simmons at CSL between 1955 and 1957. We must also accept as pure coincidence, the Cabinet review of CSL’s operations and the flurry of correspondence between Gajdusek, Smadel, Burnet and Rivers at NFIP in April 1957, and Bazeley’s investigation of polio and his involvement with NFIP. Given the problems related to polio focussed on vaccines and their safety, or lack thereof, it seems reasonable to speculate that a similar problem might have been in operation in relation to the study of kuru. So the managerial style displayed by Bazeley and Gajdusek might have a bearing on the situation in both series of events.

As an example of the tense situation that existed at CSL during the period in question, a letter written by Metcalfe, the Director-General of Health in 1958 is illustrative. Bazeley had written to Metcalfe (February 18th) requesting leave to accept an invitation to give advice to the pharmaceutical company Chas Pfizer and Company Inc. in the United States on “the production of the Poliomyelitis Vaccine and other incidental matters”. Metcalfe was not impressed with the request or two others that followed it, and responded by saying:

“In view of the extraordinarily complex and difficult position at the Commonwealth Serum Laboratories at the present time, I could not agree to you taking recreation leave at the present, quite apart from you visiting overseas, nor would it be allowable for you to undertake a private assignment during recreation leave having regard to the purpose for which recreation leave is proposed” (quoted in Brogan 1990:135).

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Brogan suggests that a letter Bazeley wrote to Metcalfe on March 17th 1958 makes it clear that he ‘felt a debt of gratitude’ to the polio foundation (Brogan 1990:135).
There were pressing needs at home evidenced by a letter written on February 24th that year, in which the Director General of Health remarked on the additional problems associated with the organisation of the institution, the annual estimates and the works program as well as the vaccine problems (Brogan 1990:135). In spite of this, Bazeley again defied authority and went to America, which only assisted in raising the level of tension again following the appointment of Partridge. Bazeley was prepared to collaborate with Chaz Phizer at a time when CSL was in turmoil. Metcalfe, it seems, saw things from a different perspective - an Australian perspective - where the problems at CSL took precedence.

Another reason for Bazeley’s alienation occurred when he made what Brogan terms a ‘mysterious visit to Parliament House’, Canberra in August 1958. This occurred apparently, without authorization from the Director General of Health or the Minister for Health. After leaving Parliament House, Bazeley held up an official car for two hours in order to visit Yarralumla nursery. A ‘request for an explanation seems to have gone unanswered’ (Brogan 1990:136). Without further research it is impossible to understand the purpose of the visits. What is clear though is that by 1961, after much criticism about his management style by the Board and the Department of Health, Bazeley was demoted to his former position as Head of Research⁹. He did not formally resign from the Public Health Service until 1964.

These examples provide a sense of the times and the complexity involved in producing human vaccine and sera free from animal virus contamination. When this context is considered, along with the fact that many thousands of CSL vaccines were administered in New Guinea (Gunther 1951), it seems remarkable that the possibility of an iatrogenic cause for kuru has not been investigated, or even considered, in any of the reports, papers, chapters and books written on the subject, which are in the public domain. One indirect but highly relevant exception to this comes from a paper presented by Gajdusek in 1970 at a World Health Organisation International

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⁹ Metcalfe wrote about Bazeley’s uncooperative nature, his lack of discipline, his penchant for withholding information from the Health Department, which resulted in it never quite knowing if it was getting all, or, the right, information, and his inability to respect any “restraint or control” (Brogan 1990:136-7). The Board of CSL argued that the situation had resulted in “a deterioration of relationships in his own organisation as well as with his Department” (in Brogan 1990:138).
Conference on the Application of Vaccines Against, Viral, Rickettsial, and Bacterial Diseases of Man, held in Washington, D.C. on December 14-18.

The paper deals specifically with the possible preventative strategies that could be employed to guard against chronic, slow degenerative diseases. His words are highly relevant in light of the problems related to polio vaccine:

Although the eradication of an acute virus disease by mass immunization may be expected to also eradicate a slow, latent, or defective infection with the agent and its delayed or slow pathological consequences, the production of low-level immunity and inoculation of live viruses of low virulence may actually contribute to such slow disease (Gajdusek, Gibbs & Lim in WHO 1971:566-577).

This is not to imply that Gajdusek was responsible for the occurrence of kuru, but it shows that vaccines which used live attenuated viruses were being discussed as a possible cause of prion diseases in specialist circles. This information is not widely circulated in the literature on kuru. Instead it is confined to the proceedings of specialised conferences and articles written about vaccines and biological standards, and even then kuru, as opposed to scrapie and iatrogenic CJD, is disassociated from this context because of its overt association with cannibalism.

Brogan’s account about CJD acquired from the use of human pituitary hormone produced at CSL is just about as sparse as it is about kuru. It can’t be denied however that the problems were similar during both time periods, that is, between 1955 and 1964 when polio vaccine problems and kuru occurred simultaneously, and from 1985 onwards when pituitary hormone came under the spotlight. Brogan provides beautiful accounts of the tensions at both time periods, but while his account of polio is well developed, it lacks an adequate appraisal about both the kuru investigation and the hormone studies. Both are glossed over and give the impression that neither was a significant event in scientific history. The main difference between the kuru situation and the pituitary hormone situation is that the hormone that resulted in five deaths in Australia was produced at CSL. In the case of kuru, a much larger number of people
have died - around 3000 since the beginning of the nineteen fifties\textsuperscript{10} - but these deaths have never been overtly associated with any product made at CSL. The lacunae in Brogan’s account of the hormone studies might be explained at least in part, by the findings of the Inquiry into the use of pituitary hormone in Australia (Allars 1994). This study concluded that the administration of the hormone project had both ethical and legal irregularities during its operation between 1962, when it was informally established in Australia, and 1985 when it came to a halt in Australia, America and England almost simultaneously. Given the similar absence of information about kuru in Brogan’s history of CSL there is room to consider the kuru project from the same perspective.

I want to shift the institutional focus now away from the records of CSL, although not away from the organisation’s problems, and move on to examine the records of the office diaries of Sir MacFarlane Burnet, Director at Walter and Eliza Hall Institute (WEHI).

**Polio and kuru:**

**Concurrent problems at the Walter and Eliza Hall Institute 1961-1962**

The office diaries of the Institute are useful for many reasons, the main one being that they provide quite a lot of detail about Burnet’s role in the Polio Committee established in 1961, and about the role of the polio epidemiological and technical sub-committees. The diaries reveal the extent of contamination problems related to a number of vaccines. Many of these issues were discussed at a meeting sponsored by the Glaxo Laboratories, which was held between June 12-14\textsuperscript{th} 1962. In May, one month before the Glaxo meeting, a meeting was held to establish a medical research committee in New Guinea under the direction of the Minister for Territories, and to develop recommendations in an effort to control kuru. The diaries also help to show the research interests of Drs Anderson, French and McLean, who earlier were involved in the research conducted during the nineteen fifties on MVE. By 1962,


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Anderson was in America and was shortly thereafter approached by the National Medical Research Institute in London to work in the area of biological standards. French had found a new cattle virus. And McLean brought news from Canada about the polio vaccine available from the Connaught laboratories. All of these events occurred while WEHI, not unlike CSL, updated equipment and negotiated for new buildings, while simultaneously grappling with the control of kuru.

By examining the diary entries relative to the above it will be possible to gain a sense of the situation that existed during this critical period. This information will form the basis for further consideration of the idea that kuru might have an iatrogenic cause. I begin with a general overview of the involvement of Burnet in the Polio Committee.

When Bazeley’s demise as Director of CSL came in July 1961\(^{11}\), he was given permission to work at the WEHI by Major General W D Refshauge\(^{12}\), who by this time had taken over from Metcalfe as Director General of Health\(^{13}\). The problems relative to the manufacture of polio vaccine continued and, according to Brogan, Bazeley made a request to the Minister for Health that Burnet chair the committee to investigate CSL’s production difficulties (Brogan 1990:123). An Act of Parliament, _The Commonwealth Serum Laboratories Act_, had been passed in May, which placed the laboratories under the direction of a five-person commission (Brogan 1990:145-148 & 277 note 68). On August 15\(^{th}\), the Polio Committee advised the Minister that the committee could be wound up, as the situation was now satisfactory. Dr Cameron from the Department of Health phoned Burnet the following day to thank him for the work of the committee and agreed to disband it. Later that month, on August 24\(^{th}\), Burnet received a letter of thanks from the Minister for Health for the work of the committee. It would be easy to gain the impression that the problems had been resolved by this stage, but on September 19\(^{th}\) Burnet, Cameron, and Refshauge met to talk ‘primarily on polio’ vaccine, on CSL, and on the changing nature of vaccines. Also discussed were ‘personal aspects’ of the findings of the polio committee and it was agreed that these should ‘not be put on paper’.

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\(^{11}\) The demotion occurred on July 24\(^{th}\) 1961 (Brogan 1990:153)

\(^{12}\) FMB Office Diary at WEHI 1961 August 12\(^{th}\) held at the Adolph Basser Library, Canberra. I thank Rozanne Walker at the library for her assistance during the current project.

\(^{13}\) Brogan 1990:143
Without further research it is impossible to say what the personal aspects were that were never to find their way into print. However, three weeks later on October 4th, a diary entry was made which reads ‘monkeys to be got rid of as soon as possible’, and so no matter who may have been responsible for the problem, the evidence was to be disposed of. The next day (Oct 5th), Burnet received word that the Commission for CSL would be announced shortly\(^\text{14}\) and in November that year it began its work (Brogan 1990:286).

Burnet’s involvement in negotiations about the production and use of polio vaccine continued throughout 1962. At the end of January (30\(^{th}\)) he received a call from R. W. Greville, the Director of the Commission, informing him that the ‘next batch of Connaught Labs polio vaccine has been checked for everything except presence of SV40’. Refshauge requested Burnet’s opinion as to whether the vaccine was safe to use. Burnet replied:

> In my opinion the presence of SV40 in a formalized vaccine should be no bar to its distribution, particularly as until this year probably all Salk vaccine had some of the virus in question. This batch had been approved by the Canadian Government but would not be passed by the American Drug Administration\(^\text{15}\).

It would seem from this diary entry that the America Drug Administration had a more rigorous standard than the Canadian authorities. And yet, it seems that the lower standard was accepted based on the assumption that the monkey virus SV40 was probably part of previous Salk vaccines and so the Connaught vaccine was little different in this respect. This tends to indicate that Burnet was prepared to take a chance even though he had ordered the monkey population be disposed of due to SV40 contamination.

Five months later, on May 3rd, Burnet received word from Don McLean, who as he had suggested in 1956, would be an ideal candidate to continue the MVE project in New Guinea in 1957. By 1962 he was informing Burnet about the ‘Current Canadian practice in polio immunization’ and the fact that the Connaught laboratories were now

\(^{14}\) This is the end of the account on polio from FMB Office Diary at WEHI 1961. All subsequent entries come from FMB Office Diary at WEHI 1962, also held as Adolph Basser Library, Canberra.

\(^{15}\) FMB Office Diary at WEHI 1962 January 30\(^{th}\)
testing ‘to ensure freedom from SB40’. It is probable that this should read SV40. If this is the case, only five months previously Burnet was prepared to endorse the use of vaccine that contained SV40. By May, apparently, at least at the Connaught laboratories in Canada, opinion had changed as they were now testing for the virus.

The protocols for administration of polio vaccine in Australia were again reviewed and the opinion altered at this time. The protocol in Canada included giving the standard Salk dose to infants, but also commencing in 1962, it was recommended ‘to cover the whole of the schoolchildren with oral [Sabin] vaccine’\(^{16}\). The following Monday (May 7\(^{th}\)) the meeting of the Epidemiological Committee decided to ‘initiate Sabin vaccine early winter 1963’. That evening, Burnet received a telephone call from Howes saying that ‘some misunderstanding had arisen during the meeting’ in relation to the protocol to be used in Australia. Burnet understood that ‘the decision was to cover all primary and sub-primary schoolchildren [with Sabin vaccine] irrespective of whether they had received Salk or not’. The objective was two-fold. Not only was the vaccine to provide individual protection, it was also ‘to provide a block to the spread of paralytic strains’\(^{17}\). Burnet also understood that the meeting had agreed to continue the universal ‘primary immunization with dip. Tetanus, pertussis and Salk polio’. Mr C. S. Butt, the Chairman of the C.S.L. Commission met that day with Burnet\(^{18}\), no doubt to discuss the preferred protocol.

Three months later, on August 10\(^{th}\), Burnet received a telephone call from Johnson in Canberra regarding the ‘importation of Sabin vaccine’. Burnet ‘strongly recommended that it be imported in the monovalent form so that immediate action with the appropriate strain could be taken if called for by increasing prevalence’. The vaccine was to be imported from Canada and the Canadian standard rather than the British would be used\(^{19}\).

This situation changed again in September when worrying news was discussed at a

\(^{16}\) FMB Office Diary at WEHI 1962 May 3\(^{rd}\)
\(^{17}\) FMB Office Diary at WEHI 1962 May 7\(^{th}\)
\(^{18}\) FMB Office Diary at WEHI 1962 May 7\(^{th}\)
\(^{19}\) FMB Office Diary at WEHI 1962 August 10\(^{th}\). The entry for this day actually reads Friday August 10\(^{th}\). However it would seem that this is a typographical error as the following page continues with the date Tuesday 14\(^{th}\).
meeting held at CSL on Monday the 24\textsuperscript{th}, at which Burnet saw 'letters from N.I.H. giving official account of 11 cases of polio following Sabin type 111, all in adults'. The news led Burnet to record in the diary: 'This may necessitate further consideration of Australian policy towards Sabin'\textsuperscript{20}. Three days later, he was asked for his opinion and advice on whether to go ahead with the use of Sabin vaccine. A cable he received reads as follows:

"You will be aware recent experiments Canada USA Sabin vaccine Stop In the light of this information would you advise release Sabin vaccine which has answered all tests in our hands and Medical Research Council on behalf Ministry of Health Stop Your guidance would be greatly welcomed Stop Ask Hunt Giaxo cable you reply".

Burnet immediately phoned Hunt and a return cable was sent to Jephcott, which read:

"Have discussed American and Canadian difficulties Sabin vaccine Stop Believe issue should be limited to Types 1 and 2 for general use Stop Type 3 should not be given to persons over 14 and only to children when adult home contacts have been Salk immunized"\textsuperscript{21}.

It is evident that there were problems associated with both Salk and Sabin vaccines, at least Type 111. Knowing this would have made it difficult to decide whether to keep on using Salk vaccine or change to Sabin's vaccine.

An examination of the discussions that took place at some of the committee meetings related to polio vaccine is useful as it sheds light on comments made in a \textit{Four Corners} television documentary on polio. \textit{Monkey Business} aired on ABC TV on April 13\textsuperscript{th} 1998 and was presented by Liz Jackson. The program noted that when asked, the Australian government was unable to supply a reason for why it did not change over to Sabin's vaccine, when England had done so. Could it be that the news at the meeting from NIH was one of the reasons why Australia did not decide to use the newer vaccine? The impression one gains from the \textit{Four Corners} program is that only the Salk vaccine was contaminated, while the Sabin vaccine was ostensibly safe. It is fairly clear that this was not the case judging by the diary entries. The decision to

\textsuperscript{20} FMB Office Diary at WEHI 1962 September 24\textsuperscript{th}
\textsuperscript{21} FMB Office Diary at WEHI 1962 September 27\textsuperscript{th}
review protocols of vaccine administration seems like a rational thing to have done rather than a possibly negligent act in this case.

If it was the case that the Sabin vaccine, at least Type 111, also had problems associated with it, it is conceivable that this information would not have leaked very far outside of the boundaries of these high level meetings. It would have been difficult to publicly acknowledge that there was also a problem with one of the alternative vaccines. Nevertheless, the secrecy is worrying. Also worrying is what seems to have been a willingness to use the Connaught vaccine in January 1962 when it was known to be of a lesser standard than that used in America. This is not to deny the difficulties encountered by those engaged in work on the manufacture of vaccines, which were considerable, if not insurmountable, and Burnet’s position, as adviser on these matters, was an unenviable place to be. But this information needs to be considered in relation to the possibility that kuru might also have been associated with vaccine use.

The journal entries say a lot about the picture I am seeking to create, for they reveal the problems associated with the substrate (growing media) used to produce vaccine, and with determining appropriate protocols of administration when the situation is unclear. In the case of polio vaccine, it was monkey kidney tissue substrate that had been used to cultivate the poliomyelitis virus that presented the problem of SV40, and this needs to be distinguished from the vaccine induced polio cases that I have just described. Chick embryo membrane was another problematic substrate on which a lot of attention was focussed at this time. And there was a growing realisation that the use of any animal substrate for human vaccines carried with it a degree of risk.

I turn to focus now on some of these issues and at the same time bring into view some of the research carried out by Anderson and French at this time. This will enable me to highlight some of the complexities involved in this particular institutional context, which seem to be relevant for an examination of prion diseases like kuru.

At the same time as the polio problems were being discussed during 1961 and 1962, French found a new cattle virus. Burnet agreed that the virus required identification and noted that arrangements could be made and Abbot was assigned to the job. The diary noted that this ‘would not require Abbot to be cited as co-author’. This seems
unusual considering that authorship and publication is one of the ways that careers are built in the scientific community.

A loosely analogous event occurred in 1957. When Dr Igor Klatzo, a neuropathologist in the United States, examined kuru brain specimens, he and his superior Dr M. Shy did not want to be acknowledged for their opinion when it was discussed in the first scientific paper on kuru published in the *New England Journal of Medicine* (Gajdusek & Zigas 1957, see Gajdusek 1975). On August 26th 1957, Smadel wrote a memo to Shy, the Clinical Director of the National Institute of Neurological Diseases and Blindness, indicating that a letter had been received from *NEJM* suggesting an insertion to the pathology section of the paper should be made incorporating the findings of Klatzo. Three days later, Smadel wrote to the Associate Editor of *NEJM*, Dr Ingalls, responding to Dr Garlands note of the 15th, agreeing with Garland’s suggestion that histopathological information would strengthen the paper. He also wrote that Drs Shy and Klatzo ‘are of the opinion that ...[a paragraph on the histopathology] should be used without direct reference to either of them’ (Farquhar & Gajdusek 1981:123).

There are differences in the two situations. In one case the situation involved the identification of a cattle virus, in the other an examination of kuru brain specimens. And in Klatzo’s case he requested not to be acknowledged, whereas it is unclear as to whether Abbot consented not to be a co-author. Nevertheless, there is a common factor in that the researchers would not have their name associated with their findings. Given the kudos that accrues from acknowledgments in scientific publications, it is difficult to understand the reasons for Klatzo’s and Shy’s reluctance. While it might simply have been because their report was preliminary, Goodfield (1985) suggests that Klatzo begin his work on kuru specimens reluctantly, without saying why this was the case.

I want to detour for a moment to provide a possible explanation. Dr Anthony Morris, who worked at the American Food and Drug Administration from 1960-1976, explained on the *Four Corners* program on polio, mentioned earlier, what can happen sometimes to scientists who engage in work related to the safety of vaccines. When it was first suspected that SV40 was a possible contaminant of polio vaccine, two
American women scientists, Dr Bernice Eddy and Dr Sarah Stewart, began research to transmit SV40 to small animals, with the result that cancer developed. This work was conducted between 1956 and 1960. When Eddy announced the findings, the work was silenced, Eddy lost her laboratory and assistants, and her authority to determine the safety and efficacy of vaccines. It is apparent that Eddy and Stewart were not prevented from speaking about their findings at specialised conferences and in specialised journals, as Eddy presented a paper at the meeting held in Washington in 1964 on Slow, Latent and Temperate Virus Infections, the proceedings of which were edited by Gajdusek, Gibbs and Alpers (1965).\(^{22}\)

It is ironic that in an earlier paper by Eddy et al (1958), cannibalism is mentioned on two occasions in ‘Neoplasms in Hamsters induced by Mouse Tumour Agent passed in tissue culture’. Eddy et al (1958: 748 & 750) found that out of 61 hamsters used in the study, 21 of them were cannibalised by their parent. Thus, actual cases of cannibalism were witnessed in the research that explored ‘the capacity of preparations from tumors of mice to elicit neoplastic responses in hamsters after incubation in tissue culture of mouse embryo cells’ (p 747).\(^{23}\) Despite speaking about the results in a specialised field however, this information was contained at this level. Because of what had happened to Eddy and Stewart, Morris suggests that this led to reluctance on the part of scientists who considered venturing into this line of work (Four Corners 13.4.98).

Of course, this would only provide a plausible explanation as to why either Abbot or Klatzo were not required to be cited, if the work involved vaccine production, and it does not explain why Gajdusek’s and Zigas’s names were associated with kuru research, as was Klatzo’s from 1959. Nevertheless, it is relevant to note that a Dr J. Anthony Morris worked with Gajdusek on research to test whether scrapie could be

\(^{22}\) Eddy’s paper, ‘Latency of infections with Simian Virus 40 and Adenovirus, Type 12’, includes a number of papers on this subject published between 1962 and 1964 (in Gajdusek, Gibbs & Alpers 1965:369-377).

\(^{23}\) In a footnote to another paper published by Stewart et al in 1957, the authors note that in 1951, Law and Dunn ‘reported on the effect of a filterable self-propagating contaminant’ on a transplantable subline of C, G 153. C, G 153 was an ascites tumor that had been passaged to the 98th transfer with the help of the chemical 8-azaguanine (p 419). This shows that the idea of a self-propagating agent was being investigated in cancer research. In 1951, Law and Dunn published their paper, ‘Effects of a filterable self-propagating contaminant on a transplantable acute lymphoid leukemia in mice’, in the J. Natl. Cancer Institute. 11: 1037-1055. In 1957 and 1958, Stewart was working at the National Cancer Institute and Eddy was working at the Division of Biologic Control, both of which were part of the National Institute of Health.
transmitted to mice during the early nineteen sixties. This occurred after the reported findings of Eddy and Stewart. Shortly thereafter, kuru transmission studies began, and recall that Gajdusek told Richard Rhodes in interview that a secret location was required for the kuru transmission studies during the 1960s (Rhodes 1997). If kuru was associated with vaccine research, Morris’s comments provide a plausible explanation for Klatzo’s reluctance and why a secret location was required for the transmission studies that followed. However uncomfortable this idea might be, Morris’s words should not be overlooked nor should the fact that he was directly involved in the early transmission studies of scrapie with Gajdusek.

It would appear from an article published in Science in 1976 that Dr. J. Anthony Morris, himself, suffered a similar fate to Eddy and Stewart after having spoken out publicly in 1971 about what he saw as ‘a “major breakdown in the scientific integrity” of the vaccine agency’ (Boffey 1976:1021). Morris and his attorney, James S. Turner, a consumer advocate, alleged that ‘managers were suppressing or ignoring data, failing to ensure the efficacy of vaccines, and harassing scientists (such as Morris) whose research findings might harm the vaccine market’. This resulted in a long wrangle between the agencies and Morris, and despite leading scientists like Gajdusek speaking highly of Morris’s work, attempts were made to discredit it (1976:1021).

It is uncomfortable to believe that information about the safety of vaccines or any results of research showing a possible danger to the public would be withheld. But a further reminder of this happening occurred at the same time that Klatzo was carrying out an examination of kuru specimens in August 1957. On August 8th that year, Burnet was involved in helping to prevent the publication of effects of radiation on sheep in Australia. According to Roger Cross, the author of *Fallout: Hedley Marston and the British Bomb Tests in Australia* (2001), Burnet is said to have attempted to prevent information about the possible dangers of radiation being reported in a scientific paper. At this time there was an Australian Safety Committee on Nuclear fallout, which was set up to guard the public of Australia against damage from radioactive fallout during the tests carried out on Australian soil at the behest of the British government. From the end of 1956, Marston had tried to publish a paper, which he believed showed that the radioactive fallout was not as benign as the Safety
committee had been assuring the public that it was. Much to Marston’s dismay, Burnet, whom Marston considered his friend, wrote to him on August 8th and asked that he remove any accusations made against the physicists on the Safety Committee\textsuperscript{24}. At this time, Burnet was the Chairman of the National Radiation Advisory Committee (Cross 2001:140), and wrote the letter in this capacity. Marston was particularly irked as he saw the situation as one where Burnet seemed to be helping to maintain the integrity of the Safety Committee at the expense of the public’s health. At this time, Burnet was also on the Board of the committee that overlooked the editorial process of the CSIRO journal in which Marston was trying to get his article published. Clearly, Burnet’s action at this particular point of time, in terms of what gets published and what remains hidden, is questionable and should therefore be taken into account when analysing the kuru story, which I am suggesting also has some unanswered questions attached to the publication of Klatzo’s preliminary findings. It seems remarkable that Burnet’s letter to Marston, and Klatzo’s preliminary report were written within a week of each other.

I return from this detour now and turn to Anderson’s work during the early nineteen sixties, to show that the picture becomes ever more complex in terms of possible contaminants.

In January 1962, a note was made in the diary that McIntosh from Commonwealth Health was going to write ‘in regard to the standard F. and M. [foot and mouth] sera held’ at the Institute by Anderson. The sera were to be ‘passed over to French’s keeping at Animal Health’ but could stay at the Institute until Anderson returned\textsuperscript{25}. This suggests that animal and human viruses were being housed in the same facility\textsuperscript{26}, which is a practice that was known to be a potentially dangerous one by 1967 (Wilson 1967). Here, it is relevant to recall the discussion about foot and mouth disease that took place some time either late in 1956 or early 1957, to which Lois Larkin referred when she wrote to Gajdusek in March from WEHI (refer previous chapter). This

\textsuperscript{24} Cross 2001:140-142 citing papers obtained from Marston’s papers held at the Australian Academy of Science – Marston Series 48:8. See footnote 52 Cross p. 201.
\textsuperscript{25} FMB Office Diary at WEHI 1962 January 10\textsuperscript{th}
\textsuperscript{26} At the time, Anderson was overseas. An entry for the previous year notes that a letter had been received from Uhr at New York University ‘in regard to possibility of Gray’s spending 3 months with him’(FMB Office Diary at WEHI 1961 August 12\textsuperscript{th})
discussion tends to suggest that kuru was viewed at this early stage within the context of animal disease. Knowledge about the transmission of foot and mouth disease by vaccines has been recorded since the early part of the twentieth century (Higgins 1920). More recently, vaccines against foot and mouth disease actually caused the disease in Europe during the early 1990s\textsuperscript{27}. Knowing this helps to add support to the idea that kuru might also have been associated with the same means of transmission. Whilst there is no direct relationship between the foot and mouth to which Larkin refers in 1957, Anderson’s work on MVE, and his foot and mouth sera in January 1962, there is enough evidence to be cautious before putting these events aside. When it comes to assessing the idea that vaccines might have been associated with kuru all avenues need to be considered.

In 1959, Anderson and a colleague, Dr G. L. Ada, were reporting on their own unexpected research findings that have relevance for prion agents which are known for their durability and resistance to many known processes used to purify other agents. Discussion of the research requires another brief detour at this point to look at the molecular and chemical level.

Anderson and Ada’s paper, ‘Murray Valley Encephalitis Virus: Preparation of an Infective “Ribonucleic Acid” Fraction’, was published in *The Australian Journal of Experimental Biology And Medical Science* (1959)\textsuperscript{28}. When the virus of Murray Valley encephalitis was being separated from its medium, the first application of phenol resulted not only in the virus but also an infective “RNA” particle. (RNA is a shorthand way of writing ribonucleic acid and viruses are made up of a protein coat and either RNA or DNA fragments). Other authors had also found, as did Anderson and Ada, that phenol treatment assisted in producing such fragments in ‘several animal viruses’. It was shown that the ‘infective agents’ that were produced had some of the properties of RNA when treated with phenol (Anderson & Ada 1959:353).

\textsuperscript{27} Standard vaccines [against FMD] are made from killed virus. But not all of the viruses were being destroyed, so vaccines were actually causing most of Europe’s outbreaks [of FMD]’ (MacKenzie (2001) *New Scientist* Special Report March 31\textsuperscript{st} p. 16).
\textsuperscript{28} Vol 37 pp 353-363. The MVE virus used in this study was that isolated by French in 1951 (Anderson & Ada 1959:354).
Another determining factor of the overall product related to the order in which reagents were mixed. This mattered because depending on the order, different results were obtained relative to the inhibitory factors of various substances used to RNA. For example, whereas “RNA” was inactivated by ‘ribonuclease, normal rabbit serum, and extracts of spleen and lung’, when either brain or liver were used, these extracts ‘protected the “RNA” against inactivation’ (Anderson & Ada 1959:353). It was thought that brain or liver might contain an enzyme, ribonuclease, that normally breaks down RNA, but which prevented recognition of the infective “RNA” which had been isolated. This in effect meant that while “RNA” might be inactivated by the RNAase the process might not be total leaving the infective form. The temperature at which the study was conducted also made a difference to the findings (Anderson & Ada 1959:363). The major implication of the findings was that the current methods and techniques for virus isolation in 1959 were inadequate.

This was not the first time that researchers had made similar findings. Gierer and Schramm in 1956 also ‘showed that treatment of purified preparations of tobacco mosaic virus with concentrated phenol solution at low temperatures yielded an infective agent which differed from the original virus’. These earlier experiments found that the infectivity of the product was ‘unstable at 37°C’ and due to the infectivity being destroyed by ribonuclease (RNAase) this led to the conclusion that the product was in fact an RNA fragment (Anderson & Ada 1959:353).

That Anderson reported on the inadequacy of the existing methods by 1959 and that an infective RNA fragment could result from the processes used to identify the MVE virus has relevance when considering prions because of the known difficulty in inactivating these agents and their illusive nature. The question is whether this research is related to kuru research? Steve Harris, a commentator in the magazine Sceptics, writing about a totally unrelated subject has argued that:

The art of scientific inference lies in deciding how many coincidences it is wise to accept (1992:72) 29.

29 Gajdusek also conducted research on the effects of various processes used to extract nucleoprotein from tissues. This followed on from earlier work, which had found that ‘drastic methods of nucleoprotein extraction from mammalian tissues decidedly altered the desoxyribonucleic acid protein complexes from the state in which they existed within the chromosomes’ (Gajdusek 1950:397).
Let's return now from this important detour and continue to examine Anderson's work from another perspective. A few years after Anderson reported on his findings about the "RNA" fragment, he was approached to work in biological standards in Britain at the National Institute of Medical Research. His relationship with the National Institute at this time, forms a bridge between the research conducted during the 1950s and '60s and the research conducted throughout the subsequent decades on Creutzfeldt Jakob disease. In 1962, Burnet arranged with Harrington to 'go ahead with finding a place' for Anderson and thought he 'would fit in well'. Harrington was 'a bit dubious as to how A. would fit in with his head - Bangham(?)'. Nevertheless, he would ask for a 5-7 year appointment.

Bangham's name is significant in the field of prion research generally because by 1980 he was the Director of the Medical Research Council in Britain (Allars 1994: 352-355), and the Director of the United Kingdom Human Pituitary Hormone Program. By 1980, Bangham was discussing Creutzfeldt-Jakob disease and the risks posed to scientists through inadequate purification of human pituitary hormone with Professor L. Lazarus, Chair of the Human Pituitary Advisory Committee in Australia. The discussion took place at a meeting of the Sixth International Congress of Endocrinology held in Melbourne in February. Knowledge of these risks was the reason given for the move of the pituitary program in England to the biological research establishment at Porton Down. This shows how purification problems dogged research over a number of decades not only with respect to MVE and polio research but also in relation to the research on prion diseases.

Anderson's research with MVE in the nineteen fifties, his involvement in kuru

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This report deals with physical and chemical properties of dexoyribosenucleoprotein preparations obtained from bovine spleen. It is included in a list of Gajdusek's publications that Burnet was sent in 1959 (in Burnet Papers Series 10/6).

30 FMB Office Diary at WEHI 1962 June 16th. The relationship between Burnet and Anderson seems to have been at a low by this time. Burnet noting that while Anderson was 'conscientious and competent' he had only 'minimal concern for prestige' and 'no loyalty'. Nevertheless, he felt that Anderson 'could do a good job in standards'.

31 FMB Office Diary at WEHI 1962 June 21st

32 Bangham is referred to in a letter written on February 28th 1980 by Lazarus to the Chair of the Fractionation Subcommittee, Dr Ferguson.
research, his relationship with Bangham during the nineteen sixties and Bangham’s association with the pituitary program during the nineteen eighties, form a continuity that is often unrecognised. In general, prion stories are sectioned off from discussion about other viral agents and individual prion stories that relate to kuru, scrapie, CJD, BSE are often written about in separate chapters with little if any overlap between them. Kuru, in particular, is segregated and the anthropological elements of the story tend to overshadow important scientific questions. The analysis I am attempting suggests that when kuru is viewed within its institutional context, there is reason to believe that it too, might have been associated with vaccine research. There is enough evidence related to outdated plant and equipment and worrying unexpected research findings to keep an open mind on the subject.

The problem of Kuru

Concurrent with the discussions on polio vaccines, a series of conversations were held at the Institute in relation to the control of kuru. Here, I want to focus specifically on some of the meetings and conversations held during May 1962 to highlight three main points. The first of these relates to two vaccine protocols that were discussed at a meeting that also discussed the control of kuru. This shows the context within which kuru was spoken about at this time, and the aim is to foreground how the rights of participants of research seem to have been subordinate to the needs of scientific inquiry. The second point relates to measures taken to control kuru and the administrative problems that were encountered. The third relates to what was known about kuru in 1962 and how this might have a bearing on the pituitary hormone project, which began in Australia, informally, the same year.

On May 18th and 19th 1962, a preliminary meeting was held under the instruction of the Minister of Territories to advise the Director of Public Health on Medical

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33 Apart from the fact that Gajdusek took over the project that had been allocated to him, Anderson is barely mentioned in most of the kuru narratives. He was however involved at the beginning of 1957 and continued to be involved until at least until October 1957, when discussions took place with P. J. Southern, Soils Chemist, Department of Agriculture, Stock and Fisheries, Port Moresby and with A. L. G. Rees, Assistant Chief of Division of Industrial Chemistry, Commonwealth Scientific and Industrial Chemistry, Melbourne (Gajdusek 1975:325).
Research in New Guinea. Around this time, Burnet received three visitors involved in the study of kuru. The day before the meeting, Dr Frank Schofield, Assistant Director of Medical Services in New Guinea, came to speak with Burnet 'primarily to talk about kuru and the possibility of a medical research advisory committee for New Guinea'. ‘Influential support’ was sorely needed to obtain ‘reasonable facilities’ from the New Guinea Administration. The diary note indicates that the Minister had agreed in principle to the new committee and there were to be a number of meetings in all. Burnet agreed to be a consultant if the committee met and the meeting was scheduled to coincide with the next meeting of the National Health and Medical Research Council. The ‘present’ one would deal with kuru, neonatal tetanus and the possibility of immunisation and a trial of BCG vaccination against leprosy.

One of the protocols discussed at the preliminary meeting was entitled ‘Trials of tetanus toxoid including adjuvant. Controlled Antibody Studies followed by a field trial’. (Adjuvant can refer to a number of substances that are added to a vaccine in order to gain an enhanced antibody response). This trial designated ‘Field Trial Y’ was to take place in Maprik in the Sepik District. Burnet agreed that he would use any opportunity available on his forthcoming trip to Geneva to support Schofield’s plan that a WHO epidemiologist be associated with the studies on tetanus immunization. The minutes of the meeting show that without the involvement of an expert epidemiologist Schofield considered that the results of the trial might not be recognised as having worldwide applicability (p10). Also mentioned was the fact that expert involvement would strengthen administrative support for the research. The meeting in Geneva to which the diary note refers was a meeting of the WHO Committee on Immunization.

The other protocol discussed at the preliminary meeting in 1962 was for a ‘BCG vaccination and leprosy trial’. The trial was to take place around the Karamui area. At this time, Gajdusek had informed Schofield about the situation that was developing

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34 'Minutes of a preliminary meeting to advise the Director of Public Health on Medical Research in T.P.N.G. Convened under the instructions of the Minister of Territories 18th –19th May 1962 in Adelaide', forms part of the Burnet Papers Series 10/6.
35 FMB Office Diary at WEHI 1962 May 17th
36 Minutes, BP Series 10/6.
37 FMB Office Diary at WEHI 1962 May 17th
around the aid post at Karamui vis a vis treatments being given (p 9). Many people around Karamui were suffering with leprosy and were being given treatment by the native medical orderly stationed in the area. The committee recommended that the Director of the Public Health Department ‘should endeavour to stop therapeutic administration of anti-leprosy drugs’, for without this action the ‘whole experiment that is planned’ would be nullified (p 9). While the minutes also mention that the treatments being given were without medical supervision and might have had side effects, it seems that the vaccine trial over-rode any potential benefit of the treatments being given. It was recommended that

treatment of this sort should be discontinued at once and that, apart from the factors mentioned above, the experiment was so important that its results might far outweigh, for lepers everywhere and for the Karamui people themselves, any short term benefit that might occur to individuals from the orderly’s therapeutic efforts.

This protocol suggests that it was not seen as problematic to stop current treatments in order that the trial would be more scientifically sound, even though the subjects of the trial might not necessarily benefit and might be put at risk. Given that this was the prevailing ethic in 1962, a question arises as to whether it would be wise to consider that a similar ethic operated prior to this time that enabled research to be conducted in the region of the Eastern Highlands where kuru occurred? The BCG vaccination and leprosy trial was to last for 5-10 years, not unlike the study of kuru. Given the similarities, there seems reason to assess the kuru project within the context of immunisation programs.

I turn now to examine the issue of quarantine of the kuru region and some of the administrative problems encountered. The minutes of the preliminary meeting show that a policy to quarantine the kuru region that had already been initiated was now to be ‘quietly discontinued’, because it was considered to be ‘ineffective, resented by the people’ and made registration of the people in the area difficult (p 4). At this time a register was kept of all the ‘individuals leaving the area’, but Burnet doubted that the list was complete. The committee recommended that ‘the whereabouts of all the people absent for more than two years’ should be identified. When found, they and their families could be ‘medically examined at regular intervals by their local M.O.,
who would report to the Director of the Public Health Department (p 4). The recommendations were written under a section on 'the technical aspects of kuru research' (p 3). The idea current at the time was that the disease might spread by genetic transfer, thereby providing a reason for the absent persons to be returned to the area to prevent so called 'breeding out' of the perceived faulty gene.

Another technical difficulty encountered at this period relates to the problems associated with obtaining post mortems. According to Gajdusek, who visited Burnet on May 21\textsuperscript{st}, a few days after the preliminary meeting, the administration was 'obstructive about permission for post mortems'. It was seen that the situation might improve if there was someone working under Schofield, who had a 'free hand' in obtaining permission from the families of the people who were dying. This would enable the post mortems to be carried out and the administration to be told about them subsequently. Gajdusek complained to Burnet that no one was seeing the patients with kuru and the two men discussed the issue of who would coordinate the research. Burnet sympathised with Gajdusek’s ‘objection to coordination of research’. Surely, as Gajdusek said, ‘all that is necessary is to disseminate freely knowledge of what has been done and is being done\textsuperscript{38}. It would appear that for some reason it might not have been possible to freely disseminate information at this point.

Dr Scragg, Director of Public Health, also visited Burnet at this time. On May 24\textsuperscript{th} Scragg discussed the need for a well-trained neurologist, who it was hoped could be found to spend 2 years in the area. It seemed unlikely that such a man would be found in Australia, and if this proved difficult the National Institute of Health in America would be asked to find the right person. Scragg and Burnet also discussed the situation relative to the Medical Research Advisory Committee and it was agreed that this committee, to which the Minister had agreed in principle, would be held in October to coincide with the meeting of the NHMRC. The first objective would be to work out 'ways and means of a Research Institute', preferably situated in the Highlands of New Guinea. Burnet was to follow up on the situation in the event that New Guinea would gain its independence\textsuperscript{39}.

\textsuperscript{38} FMB Office Diary at WEHI 1962 May 21\textsuperscript{st}
\textsuperscript{39} FMB Office Diary at WEHI 1962 May 24\textsuperscript{th}
It would appear from these accounts that the difficulties involved in establishing a “staging laboratory” in New Guinea that Burnet, Smadel and Morison had negotiated in 1956 during the year prior to the kuru investigation, still existed in 1962. Alternatively, this might have been the second stage, the first having been realised between 1956 and 1961. Just as there had been a problem about coordination of research in 1956/1957, the situation had not changed by 1962. The diary entries pertaining to Gajdusek’s visit indicate the ongoing collaboration that existed between he and Burnet, something that is often not acknowledged in the stories told about kuru, which tend to focus on conflict.

I turn now to examine the existing knowledge about kuru to highlight first its relationship with scrapie in 1962, then to show some of the possible intersection between the kuru project and the pituitary hormone project. During their discussions in 1962, Gajdusek informed Burnet that scrapie had been transferred to mice with a three to six month incubation period, although Burnet was equivocal about the incubation time as he wrote in brackets ‘(?18 months also mentioned)’. The scrapie agent had been transferred to only one of the strains of mice that had been used. Burnet noted that scrapie had been found in spleen and liver (presumably as well as in brain). He remarked on Gajdusek’s general interest in chronically active viral infections, including Visna, a neurological disease of sheep that showed some resemblance to Multiple Sclerosis and emphasised ‘the need for a reasonably sophisticated outlook on the immunological aspect of these diseases’⁴⁰. Whilst the diary does not say so, the scrapie to mice transmission studies were conducted by Morris, Gajdusek and Gibbs and the findings of the researchers were reported at the meeting held in Washington in 1964 (Morris, Gajdusek & Gibbs in Gajdusek, Gibbs & Alpers 1965: 195 and 273). Morris, as previously mentioned, is the scientist who explained the reason why some scientists were reluctant to work in the area of vaccine-associated problems following Eddy and Stewart’s findings on the dangers of SV40.

Schofield and Burnet also discussed the ‘possible relationships of kuru to scrapie and autoimmune conditions’. That scrapie was being discussed in 1962 is not surprising

⁴⁰ FMB Office Diary at WEHI 1962 May 21st
given that William Hadlow broached the relationship between kuru and scrapie in his 1959 *Lancet* article (Hadlow 1959). What is significant is that Hadlow reported on contaminants in poliomyelitis vaccine at an earlier period, and the transmission of scrapie was associated with the use of loping ill vaccine during the nineteen thirties, forties and fifties. These iatrogenic links and the fact that kuru was being discussed within the context of vaccine protocols in 1962 provide a number of reasons for serious consideration of the possibility that kuru might have been transmitted through this means.

In 1962, there were few new developments or ideas about kuru. There was however one new finding of relevance for prion research that provides a possible link between the kuru project and the pituitary hormone project. It was found that the plaques of kuru pathology appeared to contain gamma globulin and “leakage of secretory substance in the neurohypophysis”. The neurohypophysis is another name for the pituitary gland. Burnet stressed that the autoimmune side of the question should be studied and likened the situation to that of myasthenia gravis where the thymus gland was implicated. This demonstrated the importance of studying the thymus and its counterpart in birds, the Bursa of Fabricius. The diaries show that French and Burnet were involved in these studies as part of the CSL Commission. Burnet asked for thymus from children with kuru to be sent to him. In October, they were. It is difficult to disassociate the reference to pituitary gland in this context from the pituitary hormone program that began informally in Australia the same year (Allars 1994). Could it be that pituitary glands from kuru patients were also sent to Australia and used to produce hormone, not then knowing of the potential hazards?

The *Report on the Inquiry into the use of Pituitary-derived Hormones in Australia and Creutzfeld - Jacob Disease* (Allars 1994) indicates that the Commonwealth Department of Health granted CSL a licence to import glands from New Guinea in 1966, although there is no record of receipt in CSL files (1994: 49)⁴¹. Gajdusek sent pituitary glands from kuru patients to Smadel in September 1957⁴² and more were to

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⁴¹ The pathologist who had collected the glands from Port Moresby hospital could not remember whether the glands were sent to CSL, but a letter at the time indicates that they were to be sent.
be sent by Vin Zigas following Gajdusek’s departure in early 1958\textsuperscript{43}. There is the possibility therefore, that some glands might have been used in the production of pituitary hormone when the program began in America, and it is also possible that pituitary glands were sent to Australia before 1966. It is to be hoped that none of these affected glands from the kuru region were used to produce hormone when the early batches were made\textsuperscript{44}. Given the movement of tissue samples from 1957 onwards, I do not feel certain that this did not occur. If it were found to be so, it would mean that there is a direct link between the kuru project and the Human Pituitary Hormone Project. It is significant that the minutes of the preliminary meeting held in 1962 indicate that the NIH was interested not only in ‘looking for a scrapie-like agent’ but also in ‘further hormone and biochemical studies’ as well as ‘further therapeutic studies (e.g. F.S.H., progesterone and intensive modern steroids)’. Recommendation ‘H’ states, that on receipt of a reply from Dr. Masland, Chief of the National Institute of Neurological Diseases and Blindness, the Director of the Public Health Department in New Guinea should ‘request the Administrator to take the appropriate steps to contact N.I.H. and the U. S. Government above the technical level’\textsuperscript{45}.

The pituitary gland has other associations besides. In June 1956, when Gajdusek discussed with Smadel an article on Hemorrhagic Fever that was just published in a German journal, he noted that instead of a picture of a pituitary gland, an adrenal gland appeared (Gajdusek 1975:38). This highlights the significance of the pituitary gland in the pathology of hemorrhagic fever a disease likened to kuru in 1957 in that both diseases were equally problematic and difficult to understand. The pituitary gland is associated then, with HF pathology, kuru pathology and the pituitary hormone program, of which the latter resulted in deaths from iatrogenic CJD. That the wrong gland appeared in the publication is a sign that one can not always rely on published material.

The month after the preliminary meeting and the discussions that took place at the Institute in May 1962, Burnet travelled to Geneva for the WHO vaccine meeting, and

\textsuperscript{43} Letter dated January 12\textsuperscript{th} 1958 from Gajdusek to Smadel (Gajdusek 1975:384-388).
\textsuperscript{44} Between 1965 and 1985, more than 170,000 glands were collected by CSL from Australia, Mauritius, New Zealand and possibly New Guinea (Allars 1994:48).
\textsuperscript{45} Minutes p. 6. BP Series 10/6. FSH refers to follicle stimulating hormone, an anterior pituitary hormone.
to London for a meeting of the Glaxo Laboratories, where the problems associated with a number of vaccines were discussed in detail. When the diary entries and the minutes of the meeting on kuru are read together, they reveal the extent of the problems that the institutions faced and the way that vaccine protocols were conceived during the period in question. Add to this Brogan’s history of CSL and his rich source of information about Bazeley’s directorship and associated polio vaccine problems, there is little doubt that research during this period was marked by uncertainty.

I want to bring this chapter to a close by summarising some of the parallels between the study of kuru and the study of polio, as it was with a discussion of the problems of polio vaccine that the chapter began.

The parallels between kuru and polio research can not be denied. Not only did these two sets of events coincide in time they also shared an institutional relationship both in Australia and America. This chapter has concentrated on the Australian institutions involved, while the previous chapter outlined the role of the American National Foundation for Infantile Paralysis, to which both Bazeley and Gajdusek were affiliated. The first scientific report on kuru published in *The New England Journal of Medicine* in November 1957 lists Gajdusek as a Fellow of the NFIP while undertaking the kuru study. Despite this, earlier that year in a patrol officer report, he is described as being a representative of the American Department of Health. It is not clear whether these two affiliations were simultaneous or whether this is an inconsistency.

What is clear and another parallel between research on kuru and that conducted on polio vaccine is the ability of both Bazeley and Gajdusek to operate outside of normal channels in situations that were marked by complexity in terms of the institutional

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46 FMB Office Diary at WEHI 1962 Glaxo meeting June 12th – June 18th.
47 The financial assistance contributed by the NFIP was primarily to buy the necessary trade items such as axes, tobacco and beads with which to ‘purchase bodies (with autopsy permission) and food’ for the people dying from kuru. A normal US salary it was said, would allow a stay of ‘several more months’, or ‘at least, long enough to watch the evolution of a number of current cases to their fatal outcome’ (Gajdusek 1975:66).
context. The many letters that were sent between Rivers at NFIP, Smadel, Burnet, Gunther and Gajdusek in April/May 1957 coincide with the timing of the Cabinet review of CSL's plant and organisation due, in part, to the problems associated with conducting research on diseases of humans and animals in the same facility. Recognition of the need to upgrade plant at CSL and WEHI during the nineteen fifties was repeated during the nineteen eighties, when it was discovered that pituitary hormone was contaminated. Brogan's near silence about kuru deaths and about CJD deaths from the hormone, is the same in both cases. This, I suggest, is a serious omission in an otherwise wonderfully rich source of information about CSL's history, and it leads one to question why both episodes in Australian medical science history attracted so little coverage by Brogan. Allars (1994) findings that there were legal and ethical irregularities involved in the pituitary program's operations need to be considered, as one of the common threads linking kuru research and the pituitary hormone program is the role of the pituitary gland, which is also significant for research on hemorrhagic fever.

What is clear and a striking feature of the archival record for this period is a near absence of discussion about cannibalism as an explanation for kuru. In its place we find numerous discussions about the difficulties involved in manufacturing safe effective vaccine and sera. When considering the situation involved in the kuru investigation along with the institutional context it is difficult to conclude that kuru was not associated with these problems. Anthony Morris's comments regarding the precarious nature of being involved in this field of research help to explain why it might not have been possible to freely disseminate all of the information available.

In 1962, when Anderson was about to go England to work in the field of biological standards with Bangham, Bazeley was still the responsibility of the Public Health Department, who it seems did not quite know where to place him. In August 1963, Refshauge made a proposal to the Minister of Supply that Bazeley be considered for a position at the Defence Standards Laboratory, which at the time needed some medical research work carrying out (Brogan 1990:154). A position was created at the defence laboratories for Bazeley but he requested more leave, thus making him absent from duty without authority. He was ordered to report for duty no later than March 17th 1964 (Brogan 1990:155) but did not appear for work and resigned on medical grounds.
on May 21st that year. Gajdusek on the other hand, continued to work on kuru, scrapie, and later on iatrogenic CJD and won the Nobel Prize in 1976 for his efforts.
Chapter 5

Kuru and Iatrogenesis: too much evidence to ignore?

The problems associated with the manufacture of polio vaccine during the nineteen fifties and sixties have been identified and the simultaneous existence of the kuru investigation emphasised, as have the parallels between the two sets of events through an exploration of the institutions and researchers involved. Knowledge of these issues form parts of the circumstantial evidence for an iatrogenic cause of kuru. Through an examination of relevant meetings held on the subject of unwanted effects from vaccines this chapter aims to highlight the concerns of some of the scientists who were involved in the investigation of kuru who also spoke at the relevant meetings. Another aim is to show how kuru was categorised at the beginning of the investigation in 1957, that is, within the same context as Multiple Sclerosis and Parkinson's disease, both of which were considered to be diseases of unknown origin. A third aim is to bring back into view the relationship between relevant research on animal and human disease. The information provided adds to the circumstantial evidence in support of the idea that kuru might have a much closer relationship with a medically acquired disease than has hitherto been countenanced. Looking at the iatrogenic context that surrounded the kuru investigation provides a guide to the concerns of some of the scientists involved and enables a rather different picture of the study to emerge.

New Jersey 1956

On June 17th the Annual Meeting of the American Association of Neuropathologists took place in Atlantic City, New Jersey. This occurred at the same time as the negotiations between Smadel at the Walter Reed Army Medical School, Burnet at the Walter and Eliza Hall Institute and Morison at the Rockefeller Foundation in New
York were occurring. At the time of the meeting Burnet and Anderson and Gajdusek were getting ready to make a preliminary visit to New Guinea.  

One of the striking features of the meeting is the overlap between some of participants and the kuru investigation. Three names of particular relevance are Dr Tom Rivers, Dr Derek Denny-Brown and Dr Webb Haymaker.

At the meeting, two Japanese workers, H. M. Uchimura and H. Shiraki, presented a paper on the effects of rabies vaccine, which was published in the *Journal of Neuropathology and Experimental Neurology* the following year in April 1957, at precisely the time of the kuru investigation. Uchimura and Shiraki’s paper is a detailed account comprising sixty-nine pages of text and well marked photographs explaining the pathology of various diseases resulting from post-vaccinal encephalitis. The title of the paper, ‘A contribution to the classification and the pathogenesis of demyelinating encephalomyelitis’, hints at the difficulty workers experienced at this time in differentiating between a number of diseases of unknown cause. When kuru was first investigated it too, was grouped loosely within this category of diseases with an ‘unknown cause’ like Multiple Sclerosis and Parkinson’s disease. The first paper to be published in an Australian journal on kuru was given the title ‘Kuru: Clinical study of a new syndrome resembling paralysis agitans in natives of the eastern highlands of New Guinea’ (Zigas & Gajdusek 1957). Paralysis agitans is a form of Parkinsonism of unknown cause (Miller & Keane 1987:922).

Kuru is not seen as a demyelinating condition today, although demyelination can take place, but in 1957 it was placed within this category. Evidence of the close association between the encephalomyelitis story and the kuru story can be found in a letter received in January 1957 by Dr R. Scragg, the Acting Director of the Department of Public Health in New Guinea. The letter describes a new serum made in Russia, which, it was suggested, might be useful as a form of treatment for kuru. The letter comments on the large group of “demyelinating conditions” and recent positive findings in regards to the Russian serum. Not only does this letter place kuru within

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1 Details about the preliminary visit are outlined in Series 10/2 of the Burnet Papers and Gajdusek’s account of the visit is discussed in Gajdusek (1996).

the category of demyelinating conditions it is an indication that the administration was dealing with kuru before March 1957.

After the presentation by the Japanese workers at the meeting in June 1956, an interesting discussion took place that revolved around work conducted many years earlier by Dr Tom Rivers. The previous chapter outlined the role of the Medical Director of the National Foundation for Infantile Paralysis at the time of the kuru study, which coincided with the polio vaccine research studies. Rivers is also significant because of his association with loping ill research and filterable viruses in that he published papers on these subjects during the nineteen thirties at the same time as Burnet published his paper ‘Louping ill disease as a possible cause of X disease’ (Burnet 1934).

In 1935 Rivers, who was working at the Hospital of the Rockefeller Institute, New York, concluded a short paper he wrote with T. F. McNair Scott by stating that

Many filterable viruses naturally attack the central nervous system of man and lower animals, causing encephalitis and can be recovered from the brain or spinal cord. So far no virus has been shown to produce a clean-cut picture of meningitis in man. The new agent with which we are working seems to be able to produce such a picture and to appear in appreciable amounts in the spinal fluids of affected individuals. Whether this virus produces only a picture of meningitis in man and how great a role it plays in diseases of the central nervous system remains to be determined (Rivers & McNair Scott 1935:440 emphasis added).

Another paper co-authored by Rivers during the 1930s was discussed following the meeting. Dr A. Ferraro from New York, after congratulating Shiraki and Uchimura for their contribution to science, noted the familiarity all would have felt with

the controversy concerning the etiology of primary demyelinating diseases and with the early contributions of Rivers and his co-workers when they began to experiment on the effect of injections of heterologous and homologous brain tissue in monkeys (in Uchimura & Shiraki 1957:203).

Ferraro indicated that he, along with other workers, had confirmed Rivers’s findings,

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3 This term refers to the destruction of the myelin sheath around nerve cells, which are required for transmission of signals to the brain.
and suggested that,

... notwithstanding the great amount of experimental data, emphasizing a similarity between this acute experimental encephalomyelitis [in animals] and acute multiple sclerosis in man, other investigators did not accept our views concerning the similarity between animal experimentation and human pathology. This task in my estimation has now been facilitated by this paper which may be considered as experiments in vivo. We have in a sense resorted to human beings to find out what effect the injections of nervous tissue would have upon their central nervous system. The data reported by Dr. Shiraki seem to bear out the fact that we may be closer to the solution of at least one pathogenic mechanism of multiple sclerosis (in Uchimura & Shiraki 1957:203).

Evidently, there must have been controversy about whether animal models were appropriate for speculation about human disease transmission. The findings were significant precisely because they suggested that the use of animal models was appropriate for speculation on human conditions, contrary to what others had argued. Uchimura and Shiraki's studies had re-confirmed this point through their observations of brain specimens taken at autopsy following death after rabies vaccination in humans, to show that encephalomyelitic lesions and a demyelinating process could occur in humans as well as in animals, as a reaction to rabies vaccine. The thinking at this time was that it was probably not the rabies virus that was responsible for causing the lesions, but rather an allergic reaction to animal neural tissue leading to the production of experimental allergic encephalomyelitis.

It seems clear from the above passage that at least in some scientists' minds there was a direct link between an anti-rabies vaccine and the onset of multiple sclerosis. What is less clear and should not pass by without comment is the meaning of the phrases 'experiments in vivo' and having 'in a sense' to resort 'to human beings to find out what effects the injections' might have. Perhaps these are simply figures of speech used by scientists, but they make the text ambiguous in terms of how the material was obtained.

The authors of the paper point out that in recent years a number of authors in Japan had directed their attention to the effects of the vaccine, that the sequelae had been divided into three main types, optic, myelitic and encephalitic and that a fourth neuritic type could be added. From the reports that had been made most focussed on
the myelitic and optic types. Uchimura and Shiraki focussed their attention on the encephalitic type. The authors noted that they had 'gathered 40 clinical and 9 autopsied cases of this encephalitic type of rabies postvaccinal disease during the past 7 years' (Uchimura & Shiraki 1957:142). Later, they note that out of the nine fatal cases of disease in humans that they examined, eight of these were of an encephalitic nature; the other was myelitic (1957:207). At another point in the text, the authors say that over the past twenty years they found only two cases where the findings of histopathology 'closely resembled those of multiple sclerosis' (p. 139). Over the past seven years they had 'had the opportunity of examining 6 similar autopsied cases' to discover that they too each had a history of vaccination against rabies. This made the researchers re-examine the two former cases to find that they also had a history of vaccination (p. 139). Thus making eight encephalitic types and one myelitic. I will come to the differences in types a little later. The ambiguity however, brought about by the wording of the above quotation, will have to remain unresolved because of lack of information. Leaving this issue aside, there is no doubt from the discussion that followed the meeting that the findings were significant.

Dr A. Wolf from New York, described the work as 'closing the circle of the work begun by Rivers and Schwentker, who based their experimental plan on what had been reported on rabies postvaccinal encephalomyelitis in man' (in Uchimura & Shiraki 1957:205)\textsuperscript{4}. Wolf remarked that 'we now return to that condition as supporting the suspicion of a possible allergic etiology of human multiple sclerosis' (p 205). Dr. E. C. Alvord from Houston, Texas, was another participant at the meeting who believed that the research findings of Uchimura and Shiraki were very important. 'In the past', Alvord had 'been very skeptical of the attempts to correlate experimental diseases in animals with multiple sclerosis in man'. Now though, he thought that the disease in man was 'the reproduction of the diseases we have been playing with in experimental animals' (in Uchimura & Shiraki 1957:206). Uchimura and Shiraki pointed out that while there had been 'numerous and large scale research investigations' in relation to elucidating the mechanisms of multiple sclerosis, 'the clinical aspects and the pathogenesis of demyelinating diseases remain puzzling problems in neurology and psychiatry' (p 207). Now, with the results of their work,

\textsuperscript{4} In 1932-1933 Rivers and Schwentker co-authored a paper entitled 'Louping ill in man'.
they ‘considered that an important contribution to the classification and the
pathogenesis of demyelinating encephalomyelitis could be made’.

While kuru, scrapie and the various other transmissible prion diseases are not
considered to be demyelinating diseases *per se*, the similarities are apparent. Both
affect the brain and just as the two Japanese workers had demonstrated by comparing
the pathology of animals and humans, one possible route of transmission was vaccine,
which is similar to the situation regarding prion diseases like scrapie and mink
encephalopathy. Uchimura and Shiraki’s paper is useful therefore, in that it speaks
about the complexity of the research that began during the nineteen thirties with the
work of Rivers which continued throughout the next two decades.

Another relevant scientist from the point of view of kuru research who spoke
following the discussion in New Jersey in 1956 was Dr D. Denny-Brown. Denny-
Brown had previously been Gajdusek’s mentor at the Boston Children’s Hospital. In
late October 1957, Gajdusek was visited by a team of experts from Australia headed
by Professor J. C. Eccles, a leading neurophysiologist. Upon examination of the
people with kuru, Eccles suggested that Denny Brown should be made aware of the
situation. Gajdusek informed Eccles that he was already aware of the situation
through Smadel, who by this time was Assistant Director of the National Institute of
Health in the United States, having moved from Walter Reed Army Medical School
the previous year. At the meeting in 1956, Denny Brown spoke about conduction

5 By 1946, Hartsough observed a disease in blue mink shipped to the mid west of America (Gorham et
al 1965:279). As the losses increased, a number of what Gorham et al term ‘vaccine accidents’
ocurred. These were mainly vaccines prepared against distemper. At the time, the practice of making
vaccine involved taking a portion of infected material from an affected animal, in this case spleen, and
grinding the substance with saline containing 0.3 to 0.5% formalin’ (Gorham et al 1965:280). Then,
depending on the need, the mixture was incubated for a period of time, and injected into the mink. This
practice brought about widespread losses from Aleutian disease (AD). Gorham et al remarked:

In 1949 one rancher lost 500 mink to ad after vaccination for distemper with such a vaccine.
Floyd Marsh, a truly colourful mink raiser, said “they didn’t make baskets big enough to pack
out my dead mink.

A Connecticut mink rancher a few years later attempted to make his own vaccine against distemper
only to find that the results were ‘reminiscent of the scrapie outbreaks which followed the use of
looping ill vaccine’ (Gorham et al 1965:280). Nearly all of those mink vaccinated were dead of
Aleutian disease within six months.
problems of the nerve cell - how the length of the gap in the myelin sheath correlated inversely with the degree of remission possible.

Dr Webb Haymaker, an expert pathologist, was another researcher who contributed to the discussion following Uchimura and Shiraki's presentation in 1956, who was also involved in kuru research the following year. In 1957 Haymaker was shown a specimen of kuru that had been sent by Gajdusek to Dr E. G. Robertson, the Australian pathologist. The first pathology report written by Robertson in August that year included the opinion of Haymaker (in Gajdusek 1975:306). Haymaker published a paper on herpes virus transmission with Smadel in 1944 (see Margulis, Soloviev & Shabladze 1946), thus indicating not only his association with the kuru period but also earlier collaboration with Smadel. William Hadlow, who published a paper on polio vaccine contaminants the same year as the New Jersey meeting, also worked with Haymaker during the mid-nineteen fifties while at the Armed Forces Institute of Pathology (Hadlow 1992). This shows how post-vaccinal sequelae were of interest to a number of researchers who were involved in kuru research.

Just as polio was a problem during the nineteen fifties, so was kuru, along with a host of other diseases such as Parkinson's and multiple sclerosis, which were possibly associated with vaccine use. All of these diseases including kuru, attracted the heavyweights of medical science: Rivers, Smadel, Burnet, Eccles, Haymaker and Denny Brown. This leaves some nagging doubts about the cause of kuru and promotes a different reading of the kuru story from that which has been dominant. This was not the first time that a problem with vaccines had been identified. Uchimura and Shiraki mention Pasteur's first attempts at making anti-rabies vaccine, which led to neuro-paralysis. Others since have commented likewise on side effects from a number of vaccines: smallpox, yellow fever, TAB/TT inoculation (Miller & Schapira 1959b). Some of the problems associated with the manufacture of various polio vaccines were identified in the previous chapter. The significance of Uchimura's and Shiraki's work is that it focussed on diseases that were thought to be similar to kuru in 1957 as well as drawing attention to the complexities of vaccine research.
I want to look at one of the complexities for a moment, as it is important to understand just how complex the research could be. This relates to the way that the same agent could produce different symptoms in different people. The point can be used as an analogy with the agent/s of prion diseases in that they share a similar characteristic.

**Same agent, different manifestation**

This complicating factor is clearly shown in Uchimura’s and Shiraki’s paper. Out of the nine fatal cases of disease in humans that they examined, eight of these were of an encephalitic nature; the other was myelitic (1957:207). In addition, the disease could manifest in either a neuritic and/or an optic form. All were produced with the use of anti-rabies vaccine. In the eight cases with encephalitis the disease took a chronic course and the people died within one to six months after, in most cases, 18 inoculations. In the case of the patient with the myelitic form the course was acute and the person succumbed 6 days after the 10th vaccination (p. 140 & p. 207). In the opinion of Uchimura and Shiraki, the myelitic form resembled ‘acute disseminated encephalomyelitis’. The encephalitic form by contrast resembled multiple sclerosis, however these authors were of the opinion that it was very difficult to differentiate between multiple sclerosis, post vaccinal encephalitis, acute disseminated encephalomyelitis and experimental allergic encephalomyelitis as the similarities were perhaps more important. In addition, pathologically speaking, both encephalitic and myelitic characteristics could co-exist in the same specimen.

In light of these findings it is interesting that a recent paper published on strains of prion agent found that more than one strain could coexist in the same patient (Puoti et al 1999). There is a slight difference between the earlier work and prion research. In the former case it was a matter of the same agent causing different manifestations of disease, whereas in prion research it is a matter of different agents coexisting in the same person and thereby complicating the picture of the disease. The similarities however are enough to highlight as in both cases there was a problem in determining which disease was occurring in a given person and what had caused the problem.
The problem of differentiation was discussed a few years after the presentation of Uchimura’s and Shiraki’s paper in an article on the ‘Aetiological aspects of Multiple Sclerosis’ by Miller and Schapira, which was published in two parts in the *British Medical Journal* in 1959. These authors agreed with the views expressed by the Japanese researchers, notably that the controversy and debate about the differences between Multiple Sclerosis and Acute Disseminated Encephalomyelitis was misplaced and futile. They argued that it was not a matter of collecting more data. What was needed was a reinterpretation of the available data. Miller and Schapira remarked that the fundamentalists who argued that more knowledge was required about the structure and the properties of the agent were wrong. In the opinion of these authors, there was adequate knowledge available to draw some conclusions about at least one cause of multiple sclerosis. They pointed to the variability of the manifestations of both multiple sclerosis and acute disseminated encephalomyelitis, and how this made it very difficult to separate these diseases. They also pointed out that many cases of acute disseminated encephalomyelitis had been reported in the literature associated with a number of vaccines mentioned above.

The debate about the confused relationship between multiple sclerosis and acute disseminated encephalomyelitis is summarised by Miller and Schapira, who explain that

> if we were to synthesize a brief clinical definition of multiple sclerosis from current textbooks it would depict a chronic relapsing disease of the nervous system, of unknown aetiology, chiefly affecting young adults, initially intermittent but subsequently characterized by progressive disability, and terminating after a period of years in severe incapacity and death (Miller & Schapira 1959b: 811).

When it comes to the description of acute encephalomyelitis, this is

> generally regarded as an isolated self-limited episode, usually following a frank infection, more sudden and catastrophic in onset than the chronic disorder, and either immediately fatal or more often leading to rapid lasting recovery.

These authors posed the question: “How far do they correspond with the facts”? In

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6 March 21\textsuperscript{st} p737 and March 28\textsuperscript{th} p811.
their opinion, the ‘traditional position has been rendered more insecure by the clinical and pathological evidence of the last few years’, which had shown how variable the symptoms could be. This was particularly so in the case of the ‘definitive description of encephalomyelitis following inoculation against rabies’ (Miller & Schapira 1959b: 811).

An analogy can be drawn with descriptions of prion diseases including kuru, CJD, and the new variant form in humans related to BSE. One example of this relates to how kuru has been described with respect to dementia. While dementia is a sign of classical CJD7 it is not a marked feature of kuru or iatrogenic CJD. An early example of confusion surrounding dementia is suggested in the title of G. Jervis’s paper, ‘Sheep, minks, savages and presenile dementia’, published in Psychiatry Quarterly in 1968 (Vol 42 Supplement pp. 371-375). A more recent example can be found in Danny Penman’s book The Price of Meat (1996), written for a popular audience. Penman states that all spongiform diseases (Prion diseases) are classified together because of their similar symptoms, which are ‘progressive loss of brain function, manic dementia, coma and, finally death’ (p. 40). Prion diseases are often spoken about with reference to mania, hysteria, madness, and/or dementia and in Penman’s case mania and dementia are used together, which tends to give the reader the impression that these diseases are predominantly psychiatric disorders. And yet, according to Professor Colin Masters, ‘dementia is conspicuous by its absence’ in kuru (1995: 34). Likewise, Brown (1995: 6) notes that in the case of CJD acquired iatrogenically from human pituitary hormones ‘mental deterioration may occur very late and may not occur at all’. It is unfortunate that the discourse on kuru is confounded by discussion about dementia, but when dementia and cannibalism are considered together, the situation gives rise to even greater concern especially given that kuru has been likened to iatrogenic CJD with respect to an absence of dementia. Ridley and Baker have recently written that

A greater appreciation of the variety of clinical presentations, neuropathology, and age of onset of Creutzfeldt-Jakob disease in recent years ... should lead to greater ascertainment, particularly of those cases which are unusual8.

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7 Classical CJD occurs randomly in the world population and is evenly distributed at a rate of 1:100,000 per year per country.
Another parallel can be drawn with the perceived need to describe the agent/s responsible for prion diseases. There is a growing body of literature in this area but this does not seem to have helped to solve the problem. As I argued in the introduction, passaging various strains of scrapie agent through various species only multiplies the problem in that each time this is done a new strain of the agent can be produced. I turn for a moment to look at the discussions held at another meeting that focus on this issue.

**First International Conference on Vaccines Against Viral and Rickettsial Diseases of Man 1966**

Leonard Hayflick from the *Wistar Institute*, Philadelphia, emphasised ‘the effect of serial propagation from culture vessel to culture vessel on the biology of cells that divide in vitro’, in a presentation he made at this meeting.

In his presentation, ‘The primary problem with virus vaccines’, Hayflick noted that there was ‘such a large body of sophisticated research results on the viruses that compose modern vaccines and at the same time such an utter paucity of information about the cell substrates on which they are produced’ (Hayflick in WHO 1967:581). The disproportionate focus on the virus as opposed to its substrate that existed in the field of vaccine research found a parallel at the conference according to Hayflick, as many of the papers presented neglected to talk about the medium that had been used to prepare the vaccines. Hayflick spent some time discussing the transfer of SV40 from monkey to human via polio vaccine. He also pointed out that slow viruses could be a problem when using another species as substrate material (p 584). Hayflick’s comments coupled with the comment made in 1970 by Gajdusek that vaccines made from weakened (attenuated) viruses might actually contribute to the production of prion diseases, mentioned in the last chapter, cannot be ignored.

The complexity of the topic was also given special attention by C. H Stuart-Harris9,

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9 Stuart-Harris visited Burnet on August 3rd 1962. FMB Diary notes.
who noted in his Keynote Address entitled ‘Present accomplishments and future needs’, that:

... the very success of some of the weapons forged in the laboratory and applied in the field has served but to highlight the failures in other directions (Stuart-Harris in WHO 1967:xxv).

Stuart-Harris made the point that more work was required on ‘the immunizing materials themselves with a view to improving their purity and antigenic effectiveness’ (p27). That year, the WHO Scientific Group on Human Viral and Rickettsial Vaccines found that

... some virus vaccines honored by tradition fail to conform to the standards set for the more recently developed vaccines.

Many examples of problematic vaccines were given, ranging from rabies and smallpox to those used to protect against arboviruses (p26). Yellow fever and live influenza vaccines both needed to be ‘cleansed of accompaniments such as the fowl leukosis complex’; ‘Extraneous agents in living tissue and the possibility of further as yet unknown contaminants frighten[ed] many workers and greatly complicate[d] the problems of safety and control of live attenuated [weakened] vaccines’ (p27). Stuart-Harris argued for better surveillance of reactions in order to be able to assess the risks involved. This would have extended the existing form of control which focussed ‘primarily on the manufacture and test of the vaccine themselves’ (p28). It would seem from this, that knowledge of reactions to vaccine was missing.

I have shown how the problems faced by various institutions in the nineteen fifties extended beyond the problem of polio vaccine. This was but part of an ongoing debate about a general problem associated with vaccine manufacture, their use and differing effects. I turn now to examine how scrapie and loup ing ill were discussed at meetings during the 1940s and 1950s within an iatrogenic context. This will enable

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11 At a later meeting on the same subject, Cruickshank, R. (1971) also discussed the need to be aware of the problems involved in vaccine production. His paper is called ‘Factors to consider in the justification of vaccination programs’ (WHO Proceedings International Conference on the Application of Vaccines Against, Viral, Rickettsial, and Bacterial Diseases of Man 14-18 December 1970. Pan American Health Organization, Washington, D.C. pp 457-464).
me to highlight points of relevance for our understanding of kuru and to point out concurrent developments in scrapie research and kuru research.

**Anglo/Australian/American meetings on scrapie and louping-ill research**

As Australia grappled with the problem of kuru in New Guinea in 1957, A. C. Palmer, a scrapie researcher, stated at a meeting in Britain that

> There is a mystery about scrapie that makes it an unpopular subject for research; its study can be a chastening experience. The condition has defied solution so persistently that perhaps it should be classified as a disease extraordinary (quotation from ‘Studies in Scrapie’, Palmer 1957: 1318).

W. S. Gordon, who opens the discussion following Palmer’s presentation, agrees; he likes ‘the two opening paragraphs of Mr. Palmer’s paper because they express, so effectively, the present position in the investigation of the enigmatic disease of sheep known as scrapie’ (Gordon in Palmer 1957:1324). Scrapie, as Gordon pointed out, was a most intriguing disease which did not ‘hold out hope of easy victory’ because the agent responsible for the disease did ‘not behave according to the accepted rules of pathology, virology or immunology’. These ideas apply equally to kuru.

The problem alluded to by Gordon in 1957 in relation to scrapie had been discussed more than a decade earlier at the National Veterinary Medical Association of Great Britain and Ireland: Annual Congress 1946. Gordon’s paper was the first to be read and discussed, thus pointing to the importance of the work. Later it was published as an article in The Veterinary Record entitled ‘Advances in Veterinary Research’ (1946). The sub-title of the article ‘Louping-ill, Tick-borne Fever and Scrapie’ is indicative of the close proximity of the three diseases in Gordon’s understanding.

As Gordon, who at the time was working at the Agricultural Research Council, Field Station, Compton, Berkshire, explained:
Between 1931 and 1934 I carried out experiments which resulted in the development of an effective vaccine for the production of loping-ill.\textsuperscript{12} This vaccine has been in general use since 1935 and in his annual report to the Animal Diseases Research Association this year [1946], Dr. Greig stated that about 227,000 doses of vaccine had been issued from Moredun alone (Gordon 1946:516).

Thus, a sense can be gained of the many thousands of loping ill vaccines produced and administered during this period, some of which were contaminated with the scrapie agent.

Immediately following Gordon’s research in the nineteen thirties he planned a second experiment, which showed that scrapie was a transmissible disease and could ‘be spread by cohabitation from affected to healthy sheep’ (Gordon in Palmer 1957:1324). The subject of whether scrapie can be spread by contact through infected pasture has been controversial. Nevertheless, as previously mentioned, one of the sheep thought not to have scrapie contracted through this means from the nineteen forties experiment ‘was the cause of the breakdown in the loping-ill vaccine that gave rise to cases in Australia and New Zealand’ (Zlotnik in Gajdusek, Gibbs & Alpers 1965:275)\textsuperscript{13}. At least in one person’s mind then, there was no doubt that loping ill vaccine was responsible for the outbreaks of scrapie during the early nineteen fifties in Australia and New Zealand.

Brash (1952), in his report of the sheep that had been imported into New Zealand during the early nineteen fifties, mentions loping ill vaccine, while not explicitly associating it with the cause of death. But when the comments of Brash and Zlotnik are read together they provide a continuity between Gordon’s work during the nineteen thirties, Greig’s research during the 1940s and the Australian and New Zealand outbreaks of scrapie during the early nineteen fifties. Here it is relevant that one of the researchers who worked on MVE in Australia noted that he had considerable experience working with loping ill virus (see chapter two). This shows

\textsuperscript{12} The reader is informed by an asterisk at this point in the text that ‘Dr. Gordon illustrated this portion of his paper by means of graphs and diagrams projected by the epidiascope’.

\textsuperscript{13} Zlotnik spoke at the Slow, Latent and Temperate Viruses meeting in Washington in 1964 following the presentation of a paper by Gajdusek and his colleagues Clarence Gibbs and Anthony Morris entitled, ‘Spread of Scrapie from Inoculated to Uninoculated Mice’ (1965).
how the research interests of scientists in England and in Australia coincided with each other.

By 1957, at the same time as the kuru investigation was taking place in New Guinea, research was being conducted on the genetic aspects of scrapie. One day in July that year, I. H. Pattison began a scrapie experiment devised by Gordon, known as the ‘24 breed experiment’. This involved the injection of 250 animals of differing breeds ‘intracerebrally with a suspension of scrapie sheep brain’ (Pattison 1988:662). Out of the 1027 sheep used in the experiment the remainder were injected through the subcutaneous route. It was shown that breed did indeed play a part in the disease. Some of the breeds succumbed far more readily than did others. After 1959, Gordon initiated a program to breed two distinct flocks of sheep for experimental purposes. Herdwick sheep were highly susceptible, whereas Dorset Downs were highly resistant. These two breeds were used in this later experiment, which was conducted between 1961 and 1973.

Going back to 1957 for a moment, one parallel between the investigation of scrapie and the study of kuru relates to the issues of eradication in the former case, and quarantine in the latter. In 1957, an eradication programme of scrapie ‘by means of a slaughter policy’ was in progress in the United States, where ‘valuable information on the epidemiology and spread of the disease’ was being obtained (Gordon 1957:1327). The scrapie eradication program is never explicitly linked to the kuru investigation, and yet, while this was occurring in the U.S.A., Gajdusek and Zigas were being asked in New Guinea if the kuru region should be quarantined to prevent ‘outbreeding’ of a suspected genetic mutation. The relevant paragraph in a letter dated August 6th 1957, states:

Administratively our colleagues are trying to get from Vin and myself advice as to whether to build an immense wall around the fore [the name given to the area most affected by kuru], and let them die in peace, or whether to encourage “out-breeding” and spread kuru throughout the world. Some decision is obviously needed soon, and I certainly cannot make any (Gajdusek 1975:174).

There is a sense of urgency in the letter and a perception that the genetic aspects of the problem were influential. This is not unlike the concerns of the scrapie researchers
in England who only one month before had inoculated 24 different breeds of sheep to
test the respective susceptibility of the different breeds. What is important to note is
the timing of the letter - August 1957. Other accounts place the timing of the
cordonning of the area, much later, around 1960\textsuperscript{14}. The idea was on the agenda much
earlier however at the same time as America was also grappling with the complexity
of scrapie eradication.

During the same period, H. B. Parry worked on an independent survey of sheep
disease at the Nuffield Institute for Medical Research in Oxford (Pattison 1988). Parry’s research indicated that scrapie was a myopathy and he published two papers
on this subject in 1956 and 1957. Gordon suggested at the scrapie meeting in Britain,
that while myopathy could coexist with scrapie it could not account for the
‘complicated neurological manifestations of scrapie’ (Gordon 1957:1325). The
archival record shows that a colleague of Parry’s was invited to a CIBA meeting on
kuru, held in London in 1964\textsuperscript{15}. More research is needed before an association could
be established between Parry’s research and the investigation of kuru. This would
need to consider the Nuffield Foundation support for Burnet’s visit to America and
England in 1956, at which time he attended the London CIBA Symposium on the
physics and chemistry of viruses in late March\textsuperscript{16}. Also to be considered would be the
joint Nuffield/University of Sydney project carried out in the Highlands of New
Guinea, which found that 100\% of the people tested showed antibody to herpes virus,
as noted in chapter three\textsuperscript{17}. But even without further research, it seems fairly safe to
say that the temporal relationship is strong between the problems experienced in
England, Australia and New Zealand relative to scrapie, and the problems
experienced in Australia/New Guinea relative to kuru. The main difference between
the two diseases is that when scrapie appears in sheep, the common links are heredity,
which predisposes the animal to the disease and louping ill vaccine, which readily
transmits it.

\textsuperscript{14} A SMH report, dated May 23\textsuperscript{rd} 1960 noted that ‘in an attempt to combat “laughing death” the Fore
area of the New Guinea Eastern Highlands will be turned into a vast “concentration camp”’.
\textsuperscript{15} Letter from Doll to Burnet dated June 3\textsuperscript{rd} 1964. BP. Series 10/11.
\textsuperscript{16} The Director’s Thirty-Seventh Annual Report to the Board of the Walter and Eliza Hall Institute of
Medical Research, July 1956 p 6.
\textsuperscript{17} Letter dated May 9\textsuperscript{th} 1956 from Burnet to Dr Hale, Singapore. Series 10/2.
Gordon stated at the scrapie meeting in Britain in 1957 that while scrapie ‘was a nuisance, it was not a major economic problem’ for Britain until 1952 when it developed in British sheep exported to Canada, New Zealand and Australia. This resulted in a ban being placed on the importation of sheep from Britain by these countries and by the United States (Gordon 1957:1326). In 1958, William Hadlow, who at this time was a veterinarian working at the Rocky Mountain Laboratories, was sent to Britain to liaise between the American and British efforts to control the disease. The United States Department of Agriculture assisted with finances for the project ‘under the terms of Public Law 480’ (Pattison 1988: 661-662). In 1959, Hadlow wrote his paper for Lancet, which linked kuru and scrapie. I turn now to some of the popular accounts of how this link was made as they focus on a visit Hadlow made to an exhibit on kuru that was held at the Wellcome Medical Museum in London in 1959. According to Goodfield and Rhodes, the exhibition on kuru that had been organised by Gajdusek and the National Institute of Health, forms the central focal point (Goodfield 1985:21. Rhodes 1997: 62). The aim is to highlight the inconsistencies in the accounts in order to show that while much has been written about how and where the link was made, the story is still far from clear.

First a little background information. In the years preceding his post to England, J. R. M. Innes had been Hadlow’s mentor. In 1957, Innes wrote that in 1955 America had 41 flocks of sheep affected by scrapie in 14 different States (Innes and Saunders 1957:156). When he wrote about scrapie Innes was working at the Pathology Branch of the Medical Laboratories of the Army Chemical Centre in Maryland and was simultaneously carrying out research at the Wellcome Research Laboratories, Tuckahoe, New York. He was aware of research on scrapie carried out in England in previous years and knew that the field history of the disease pointed to a slow viral infection. As to when Hadlow became aware of scrapie, that is a more difficult question to answer.

18 Hadlow, in his own recollections of the events surrounding how he came to make the connection between kuru and scrapie, notes that Innes was his mentor (Hadlow 1992).
According to June Goodfield (1985), Hadlow had already ‘spent years on the mysteries of scrapie’ before he went to England to help with Britain’s effort to try to reproduce scrapie experimentally in goats (Goodfield 1985:21-22). In the context of talking about the ban on British sheep to America, Goodfield writes that once America had initiated the ban, the Department of Agriculture ‘nevertheless, wanted to be helpful, and that is why Bill Hadlow had been sent to England’. Then, following a visit from a colleague from Rocky Mountain Laboratory (William Jellison), who mentioned the kuru exhibit to Hadlow, he visited the Wellcome Museum in early July 1959 and had feelings of *deja vu* ‘for he was seeing changes he had been observing for years’ (Goodfield 1985:24)\(^{19}\).

Hadlow, in his own account of how he came to make the connection writes, ‘I came to Compton knowing little about the disease; to me it was nothing more than a strange malady notably of British sheep that was allotted little space in veterinary textbooks of the time’ (Hadlow 1992:41).

Richard Rhodes (1997) confines Hadlow’s previous experience with scrapie to one year prior to 1958. He writes that Bill Jellison ‘casually mentioned’ the exhibit on kuru at dinner one night, ‘Hadlow was curious’, went to see the exhibition, and was ‘riveted by the colour photomicrographs of brain sections full of holes’. These reminded him of the spongiform degeneration he’d ‘been studying for the past year’ (1997:62). Why such a factual matter as Hadlow’s foreknowledge of scrapie changes over time is difficult to answer.

Due to interest in how Hadlow made the connection between kuru and scrapie, some although not all of the correspondence has been published in *Prion Diseases of Humans and Animals* (Prusiner, Collinge, Powell & Anderton 1992 Chapter Seven). Included in the published letters are Hadlow’s letter to *Lancet* (1959); a letter Hadlow wrote to Gajdusek at NIH dated July 21\(^{st}\), a letter from Marion Poms, Gajdusek’s secretary, written on July 28\(^{th}\) to Hadlow; and a response from Gajdusek dated August 6\(^{th}\) 1959. I turn now to this correspondence to try to gain clarity surrounding the events that led Hadlow to make his crucial discovery.

\(^{19}\) A photograph of Hadlow and Goodfield appears in her book, which indicates that she had met Hadlow.
The sequence of events according to the published letters indicates that the *Lancet* letter written by Hadlow was forwarded to the journal by Poms rather than by Hadlow. Poms’s letter begins: ‘I write to inform you that I have despatched a copy of your interesting letter to Dr. Gajdusek and to the LANCET with reference to SCRAPIE AND KURU.’ (in Prusiner et al 1992:51). This suggests that Gajdusek’s secretary might have administered the publication of Hadlow’s *Lancet* letter while he was working in England. This is consistent with Hadlow’s own account which notes that he has no records of any correspondence between himself and the publishers about the letter he sent to *Lancet* (Hadlow 1992:43). Poms’s letter goes on to detail Gajdusek’s whereabouts (New Guinea) and notes that he will contact Hadlow when he receives the information.

In his response written on August 6th, Gajdusek thanked Hadlow for the *Lancet* letter and informed him where he could find more information on the pathology of kuru. In total he sent four sources of information and wrote: ‘I note that you have probably not seen our extensive pathological descriptions of KURU which include some features which were little stressed in the report you have quoted’\(^\text{20}\). Two references of publications by Klatzo, Zigas and Gajdusek were enclosed\(^\text{21}\), along with the name of a conference on *Encephalitis* held in Antwerp in May 1959, the proceedings of which were soon to be published and of interest because much of the discussion at the conference revolved around kuru pathology\(^\text{22}\). But it is the fourth piece of information that is most relevant here, as it refers to the address of the Wellcome exhibition, which in other accounts Hadlow had already visited. The relevant passage reads:

> Until August 15 or so I believe the NIH Exhibit on Kuru will still be on display at the Welcome Museum of Medical Science, 183 Euston Road, London, N.W.1, England. This exhibition included a very large display of the neuropathology of the disease with enlarged color transparencies of photomicrographs of the peculiar plaque-like body of Kuru\(^\text{23}\).

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\(^{20}\) Hadlow cited in his 1959 publication the work conducted by the Australian pathologist’s Fowler and Robertson. He wrote, ‘the multilocular “soap bubble” vacuoles in the cytoplasm of nerve cells’, before citing the work of the Australian pathologists.

\(^{21}\) The papers were published in *Journal of Neuropathology and Experimental Neurology* (1959, 18:2, 335-336) and *Laboratory Investigations* (1959, 8:4, 799-847).

\(^{22}\) It was at this conference that the remarks about the neuronophagic lesions were made, as described in chapter one.

This indicates that either Hadlow was unaware of the Wellcome exhibit by August 6th, or Gajdusek was unaware of Hadlow’s visit because he had not been told about it. Hadlow acknowledges in his own recollections that it was Fowler and Robertson’s report that first aroused his ‘curiosity about the possible similarity of the two diseases’ (Hadlow 1992:49). The Lancet letter does not make any reference to the Wellcome exhibit. This tends to indicate that although the popular versions of the story focus much attention on Hadlow’s visit to the Wellcome Museum in July, the exhibition might not have been as crucial to making the discovery as the accounts suggest.

It is indeed difficult to establish the sequence of events given the amount of time that has elapsed. This could also account for another inconsistency in accounts which relates to what took place following a meeting held in Washington in November 1959 to allay fears about the scrapie eradication program24. Apparently, there has been discussion about what occurred at this meeting. After the meeting Gajdusek is said to have approached Hadlow, but Hadlow has ‘no recollection of what was said. We parted’, he said, while he felt sure that his letter to Lancet was discussed (Hadlow 1992:44). Goodfield, on the other hand, writes that following the meeting in Washington, ‘Hadlow corresponded sporadically with Gajdusek’(Goodfield 1985:25). Hadlow’s own account indicates that, ‘[i]f we remained in contact my [sic] mail, phone or telegraph during the months that followed my return to England, I have no record of it until March 28, 1961’ (Hadlow 1992:44).

What seems clearer is that in March 1961, Hadlow received a letter and a telegram from Gajdusek inviting him to be a part of the kuru transmission studies that were being prepared. Hadlow says that he was only ‘slightly interested’ but felt committed to stay with the USDA. Nevertheless, late in July 1961, after his ‘unintended return to Rocky Mountain Laboratory’, he did meet with Gajdusek and Smadel (Hadlow 1992:44). Hadlow declined the offer and by July, says he was ready to ‘tackle scrapie’ with Carl Eklund. The date that contact was made with Hadlow coincides with the culmination of Hadlow’s three year contract in England, which shows that Smadel was aware of the details of Hadlow’s contract by 1961. The question is whether he was aware of the contract earlier?

24 A meeting took place in Australia in December 1959 in relation to kuru.
In August 1957, within a year of Hadlow’s polio vaccine research, Smadel wrote to Gajdusek telling him that he was going to go to the Rocky Mountain Laboratories for a week-long visit when he returned from the polio conference in Geneva (in Farquhar & Gajdusek 1981:113). At this time, Igor Klatzo, the neuropathologist in America was making his examination of the first kuru brain specimens and E. G. Robertson was doing the same in Australia before going to visit Webb Haymaker and J.G. Greenfield in America. The purpose of Smadel’s visit is not clear from the letter. Only more research would show if the visit related to Hadlow’s past work and whether there might be a closer relationship between scrapie research and kuru research than previously acknowledged. If it is assumed for a moment that Smadel’s visit is related to Hadlow being sent to England in March 1958, it seems possible that Smadel was involved in Hadlow being sent to England. This would mean that Smadel was simultaneously involved in kuru and scrapie research, which might mean that the kuru project could be seen as a part of a larger project on scrapie, or alternatively a part of a larger project looking at the effects of polio or other vaccines.

In some of the narratives about kuru there seems to be a reluctance to acknowledge that any of the researchers working with scrapie had any contact with those working with kuru prior to 1959. Goodfield makes a clear distinction between the researchers who worked in the field of veterinary health and those who worked on human diseases when she writes about kuru transmission studies that were being planned in primates in 1961. She notes that it was difficult to persuade Smadel of the need for the long-term studies, given that no one had ever heard of Hadlow, a veterinarian who had suggested the need for long term study. This interpretation overlooks the relationship that existed between Smadel and Rocky Mountain Laboratories in 1957, although proof is still needed of the purpose of the visit. Nevertheless, on the question of the distinction made between researchers in different fields, dating back to the beginning of the century, the boundary between research on animal diseases and research on human disease has always been blurred. Many scientists grappled with the transmission of agents across the species. Howes et al’s research on MVE in 1952 indicates that he worked with both MVE and louping ill. Burnet also worked with animal and human diseases. Hadlow, himself, worked in the area of polio and scrapie. Goodfield’s distinction is also difficult to accept taking into consideration the discussions following Uchimura’s and Shiraki’s paper.
A monograph entitled, *Diseases Transmitted from Animals to Man*, first published in 1930, with subsequent editions published in 1941, 1947, 1955 and 1963, collated research in this area for those interested in the subject. Thomas G. Hull, in his foreword to the fifth edition, wrote in the hope that the work would act as a ‘common meeting ground’ for veterinarians, doctors, health officials and research workers (1963:vii). William Jellison, who is said to have initially prompted Hadlow’s curiosity about kuru in London in 1959, is given special thanks by Hull ‘for his continued interest in the work and for his numerous suggestions’ (1963:vii). This is another indication that knowledge was shared across the boundary of veterinary and human health. But this is not how the kuru story is written about by Goodfield and Rhodes, whose accounts centralise the role of the Wellcome exhibition in Hadlow’s discovery and leave the impression that the researchers working on animal diseases were quite separate from those working on human disease.

As well as suggesting that Hadlow’s discovery on the links between kuru and scrapie was the second crucial element involved in unravelling the mystery of kuru, June Goodfield in *Quest for the Killers* also remarks that Hadlow had ‘two advantages over all other investigators’. These were that he came fresh to the problem ‘with a mind neither clouded by previous thinking nor exhausted by repeated and futile excursions into etiological cul-de-sacs’, and he ‘was totally familiar with a disease of identical pattern’ (1985:25). Meaning scrapie. It is difficult to see how any researcher could have a mind unclouded by previous thinking. I suggest that along with his previous work on scrapie, Hadlow might also have had his mind on polio vaccine contaminants following his research in this area a few years earlier (Eklund, Bell & Hadlow 1956), and so his mind was not totally unclouded. His expertise lay in this area. The point is ignored by those writing about kuru, with the exception of Hadlow who does mention this research in passing (Hadlow 1992). When assessing iatrogenic possibilities, polio, scrapie and louping ill vaccine research need to be factored into the analysis rather than overlooked.

I want to turn now to say a brief word about the question of what Smadel knew about scrapie. Rhodes remarks that Smadel knew about scrapie at the time of the kuru investigation, but he makes a point of saying that Gajdusek did not because he ‘was a city boy’. According to Rhodes, the first time that Gajdusek heard of scrapie was
when he received a letter written on July 21st 1959 by Hadlow, which pointed out the similarities between kuru and scrapie (Rhodes 1997: 63-64). On face value Rhodes’s statement that only Smadel knew about scrapie, seems reasonable. What is puzzling, is why it was even necessary to make the statement. What difference could it make to the kuru story if Gajdusek did or did not know about scrapie in 1957 at the time of the kuru study? It seems reasonable to assume that if Smadel knew about scrapie he might have shared this information, but even without this assumption, it seems fairly safe to say that Smadel was aware of scrapie by 1957, if not before. And while Gajdusek might have been a ‘city boy’, as Rhodes suggests, he was familiar with the workings of the Department of Agriculture and with animal diseases like loupng ill, which serves to show that the division between knowledge about animal and human diseases is artificial. I want to conclude this chapter with a brief look at loupng ill vaccine research that was conducted during the nineteen fifties in order to show the role played by the Wellcome laboratories in England.

In 1954, Gajdusek and Dr Alexander Terzin in Sarejevo discussed loupng ill research, following an epidemic of loupng ill in a small group of people in Slovenia the previous year. Terzin showed the results of a series of neutralisation tests on loupng ill virus carried out in mice using the sera of four of the patients from the 1953 epidemic. The sera had been forwarded to Dr. Vesenjak in England by Terzin and the tests were conducted by ‘D. F. Edwards [sic] at Wellcome Research Laboratories’ in England (Gajdusek 1991: 294). Gajdusek asked that the lyophilised virus be sent to Smadel as soon as Dr Vesenjak returned from Germany.

On May 2nd 1954, Dr Pond wrote to Gajdusek with his thoughts on various strains including ‘RSSE, loupng ill, and the diphasic encephalitis’. The ‘entire picture’ of the disease appeared to be similar to one found by Smorodintsev, which was similar to ‘the new Diphasic Meningoencephalitis’. The disease was ‘indistinguishable from RSSE’. Great effort went into the identification of this group of viruses at this time, for here was a situation very much like the one that was discussed at the New Jersey meeting a few years later, in that the same agent seemed to be able to manifest in

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25 The letter was written aboard the *S. S. Lostovo*, a Yogoslav steamer in the Adriatic off the coast of Albania.
different ways. Pond questioned whether these were different viruses or simply differ-ent manifestations of the disease through different vectors in geographically differ-ent locations. Although the agents were antigenically similar, he wondered what could explain 'the fact that encephalitis doesn’t occur in Scotland in human beings while louping ill is occurring in the sheep’\textsuperscript{26}. Pond indicated that he had 'the virus of louping ill and EMC [encephalomyocarditis] as well as the immune sera all packed' in his ice box waiting for diplomatic courier to go to Terzin.

Terzin had asked that reprints of articles held at Walter Reed be forwarded to him. Gajdusek seems to have perceived difficulties in the future were this not to happen as he wrote, 'Since he is the man I intend to put pressure on for the Diphasic Meningo-encephalitis strains, it would be wise to see if our reprints cannot be sent to him'\textsuperscript{27}.

It is not clear whether this research was of a military nature. What is clear is that the tone of a letter Smadel sent on May 11\textsuperscript{th} was of such a military nature it made Gajdusek cringe\textsuperscript{28}. At this time, Gajdusek was on his way to work in Afghanistan and Iran on Contract No. DA-49-007-MD-77, Title: Field Studies on the Control of Infectious Diseases of Military Importance. T. E. Woodward was the ‘Responsible Investigator’\textsuperscript{29}. This contract was not formally closed until March 1956 (see Gajdusek 1975:30), by which time Gajdusek was working at Walter and Eliza Hall Institute in Melbourne with Burnet, and it was following this contract that the National Foundation for Infantile Paralysis grant was provided.

Given the context, it is important to say a little more about the ethics of scientific research, particularly when military institutions are involved and the national interest

\textsuperscript{26} Letter from Pond to Gajdusek dated May 2\textsuperscript{nd} 1954 in Gajdusek 1991:313.
\textsuperscript{27} Letter to Woodward, Smadel, Pond and Morris dated May 17\textsuperscript{th} 1954 in Gajdusek 1991:325-327.
\textsuperscript{28} (The exchange of specimens in kuru research, as opposed to louping ill research, is a subject explored in depth by Warwick Anderson in a recent paper, ‘The possession of kuru: medical science and biocolonial exchange’ (2000). Anderson takes kuru brains and other material ‘to think more generally about the creation of value and the circulation of goods in global science’ (p714), and argues that these specimens became part of Gajdusek’s wealth that could be transacted with colleagues thereby increasing Gajdusek’s scientific credit (p 734). I am suggesting that the military context needs to be taken into account, as in the particular case mentioned above, a distinct power relationship seems to have been involved in the exchange.)
\textsuperscript{29} Ibid p 325.
\textsuperscript{29} Letter Smadel to Woodward dated May 30\textsuperscript{th} 1954 in Gajdusek 1991:311.
is perceived to be at stake, which, in 1954, was the situation that existed following the Korean War.

Gajdusek expressed concerns about the use of involuntary subjects in research generally, in a letter written in March 1954. In contrast, in the same letter, he describes how Terzin is well trained and imbued with a cautious approach through his work at Boston (Gajdusek 1991:295). At this time Terzin was developing a virus laboratory in Sarajevo having moved out of Beograd and was ‘skeptical of the rashness of our geniuses’. It is significant that Burnet and Stanley are listed amongst the class of geniuses. While it is difficult to go on one account in such a sensitive matter as a perceived incautious approach to experimentation, these remarks should be considered. For it was Burnet who became involved in the kuru investigation in 1957 following the discussions and negotiations during the preceding years with Smadel on encephalitides like X disease/Murray Valley encephalitis, yellow fever and Japanese B encephalitis.

Burnet was not the only person to come up against criticism at this time. Daniel and Robbins in their collection of essays on Polio (1997), remark on Smadel’s approach to experimentation within the context of polio vaccine trials. These authors write that:

One of the areas of dissension was whether or not there were adequate data, particularly on the safety of the Salk vaccine, to warrant a major field trial in 1954. A more conservative group represented by John Enders and John Paul, felt that more data were desirable and, on scientific grounds, saw every reason for moving cautiously. They were not prepared to take any significant risks. Those who wished to proceed, of whom Joseph Smadel was probably the most vocal, argued that one could never avoid some risk, and only after a large field trial could one identify an uncommonly occurring risk (Robbins & Daniel 1997:17 in Daniel & Robbins)\(^30\).

In January 1952, Hilary Koprowski, who was working on polio vaccine, met with Smadel and Karl Hooper, the then director of the G.W. Hooper Foundation (Hooper 2000:413). Edward Hooper, who has written about many of the issues related to polio vaccine trials, reports (2000:413) that in his 1959 lecture on his achievements in the field of polio research, Koprowski spoke about a “fateful meeting” that took place in

1952. According to this account, Smadel was aware of Koprowski's work conducted with live poliomyelitis virus and was interested in developing a collaborative study of polio. This meeting is said to have led to "prolonged and fruitful collaboration". Smadel's involvement with Koprowski's polio vaccine trials during the nineteen fifties is interesting given Smadel's reported incautious approach to experimentation, because Koprowski's studies have become the subject of debate at the end of the twentieth century. Questions have been raised about both the ethics of some of the studies and a possible association with the origin of HIV in Africa. A Royal Society meeting was held in England in 2000 to begin a process whereby an examination of any remaining batches of Koprowski vaccine could be tested for signs of primate immuno-deficiency virus. In September 2000, a press conference was called and it was announced that no virus had been found. This, however, might not be the end of the subject as Hooper's work highlights ethical issues pertaining to this period of research that need to be considered.

It would be fair to say that experimental research by its very nature is a potentially dangerous activity. There are however matters of degree. Working out how cautious or incautious any of the workers were in the field up until the nineteen fifties, is difficult to gauge. What is important to remember is that there was a perceived imperative to produce vaccine (Keogh 1951) and kuru occurred and was investigated during this time frame. It is therefore necessary to entertain the possibility that a vaccine might have contributed to kuru. In light of what has been outlined, there is little doubt that both Smadel and Gajdusek were knowledgeable about louping ill prior to the investigation of kuru, and the Wellcome laboratories were central to this work.

Meeting of the Biology of Viruses of the Tick-borne Encephalitis Complex 1960

Research on louping ill vaccine was discussed at a meeting of the Czechoslovak Academy of Science in 1960. The proceedings of the conference were published in Volume 3: Biology of Viruses of the Tick-borne Encephalitis Complex, edited by Helena Libiková (1962). Gajdusek, who attended the meeting, was at this time listed
as a representative of the American Department of Preventive Medicine at the conference.

D G ff Edward presented a paper at the meeting with his colleague K. J. O’Reilly from the Wellcome Research Laboratories, Beckenham, Kent. Edward’s and O’Reilly’s paper, ‘Ten Years Experience of a Vaccine against Louping-Ill in Man’, is an excellent summary of the routine use of the vaccine and some of the problems associated with immunity.

Between 1950 and 1960, at least 103 people had been vaccinated at both the Wellcome Research Laboratories and The Wellcome Veterinary Research Station and the vaccine had had no untoward effect and reactions had not been troublesome (Edward & O’Reilly 1962:345). But while there were no cases of louping-ill recognised in an immunised person, it was still possible to become sick if unvaccinated when exposed to the virus. Edward provides an example of this in a 1954 incident where an unvaccinated assistant was ‘in error employed to assist in the inoculation of sheep and in consequence she suffered from an attack of louping ill’ (p 345).

The problem for researchers working with louping ill virus was recognised in 1948, when it was noted that laboratory workers engaged in ‘large-scale production’ of louping ill vaccine for the protection of sheep were acquiring the infection (see Edward D G ff, Brit. J. Exper. Path. 29:372, 1948). In his paper in 1948, Edward described 12 cases of louping ill in humans acquired by laboratory workers. All these ‘infections were mild and it would appear that this particular laboratory-adapted strain of louping ill virus’ was much safer to handle than, for instance, the virus of Russian spring summer encephalitis (RSSE) (Edward & O’Reilly 1962:344). Nevertheless, it was felt that workers should be protected from contracting the disease. Three volunteers were given an inactivated mouse brain vaccine and went on to develop antibodies. Thus, it was suggested that the vaccine might be used to protect those exposed to the virus.

The vaccine was used in other laboratories in Britain and elsewhere (unspecified), but it was not 100% effective as Edward notes that one virologist, who was vaccinated,
suffered an attack of louping ill after an accidental needle stick injury with an infected needle. After the vaccination, no antibodies were found in his serum. It is possible that the vaccine was ineffective through inadequate storage and transit (p 345). This serves to highlight the difficulties encountered with louping ill vaccine during the ten years between 1950 and 1960.

The Wellcome Laboratory’s involvement in louping ill vaccine research needs to be assessed in terms of the investigation of kuru because of the problems associated with producing an adequate antibody response and finding a suitable substrate. Antibody response, or lack of it in kuru and scrapie, is an established problem. Developing suitable substrates was a problem of general interest during the period, as was the pathogenesis of tick borne viruses, that is, how the virus moves in the body to produce disease. Within a few years, similar questions in relation to kuru were being asked.

A question that requires further study is, are the louping ill studies conducted by the Wellcome Laboratories in England and the exhibition on kuru held at the Wellcome Medical Museum in June/August 1959 related? Given the historical relationship between scrapie/louping ill vaccine research, and the remarkable similarities between scrapie and kuru, which Hadlow is said to have made at the Medical Museum, it seems relevant to look more closely at where louping ill research, scrapie research and kuru research intersect. Another question that requires further research relates to the visit to Rocky Mountain Laboratories made by Smadel in August 1957 in terms of

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31 Vaccination is believed to stimulate ‘the formation of neutralizing antibodies which become reduced in amount or tend to disappear in the succeeding months’ (p 345). Thus the need for a booster dose. Initially a course of three inoculation of the vaccine were given. These were followed by yearly booster doses (p 344-5). Later the regime changed to ‘two inoculations, each of 1ml of vaccine, 4 weeks apart, followed by a booster dose of 1ml 9-12 months later’ (p 345). Edward points out that the ‘disadvantages of a vaccine prepared from mouse brain have been appreciated and, therefore, it was used only on those persons likely to be exposed to the virus’.

The situation regarding the effect of louping ill vaccine in the production of antibodies was unclear. It was noted that working with the vaccine and the agent led to a high titre of antibodies. This was noticed by comparing two workers, both of whom were vaccinated, but where only one was working with the vaccine, while the other was a veterinary surgeon (HS). HS had no detectable antibodies 3 years after vaccination, whereas, the other worker (GF), who was involved in the production of the louping-ill vaccine, showed a high level of antibodies in the following year (p 346). Edward suggests that ‘the probable explanation of the high levels of antibody noted in many of the vaccinated persons is that they resulted from exposure to virus’ (p 346). ‘Since no illness was noted it must be assumed that they had sub-clinical infections which boosted a basic immunity conferred by the vaccine’ (p 347). W. A. Fitzgerald made the vaccines involved in the Edward study and G. E. Scott gave technical assistance.

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whether it was related to Hadlow’s contract. In addition, more research might establish whether there were any discussions between Smadel and Innes and whether the louping ill research conducted in England between 1950 and 1960 at Wellcome was in any way related to the research conducted by Innes at the same institution in New York prior to 1957.

It is a pity that the accounts vary regarding who knew what about research on animal diseases at this time. For without definite knowledge it is not possible to put to rest some of the questions that have been raised, which prompted the publication of some of the letters in 1992. The suggestion seems to have been that had knowledge about scrapie been possessed this would in some way alter our understanding of kuru research. The inconsistencies in the popular accounts written about the kuru story only serve to complicate the issue. The seeming reluctance to acknowledge the sharing of knowledge between researchers on human and animal disease reminds me of Robertson’s remark made towards the end of 1957, at which time American laboratories were receiving most of the kuru specimens. He did not understand all the facets of kuru research therefore ‘the less said the better’.

In contrast, I am arguing that the more said the better. Rhodes argues that when the long term kuru transmission studies were suggested by Hadlow, non human primates were chosen because it was obvious that humans could not be used as experimental subjects, ‘Hadlow didn’t say what was obvious, that scientists couldn’t experiment on humans. He proposed the next best alternative (from the human point of view)’ (Rhodes 1997: 64). Despite what Rhodes says, I have tried to show that in experimental medicine, there is always a danger that the interests of science can override the rights of participants of any proposed study. While it is easy to make a judgement about the situation then, by employing concepts such as informed consent as this notion is understood today, it would be erroneous to do so. Unlike today, during this earlier period it was far more common for researchers to place the right to acquire scientific knowledge over the rights of individuals. I have tried to stress the perceived imperative to produce safe and effective vaccine during this earlier period. It is often forgotten that experimental medicine involves testing products on humans. For without such tests it would be impossible to test for efficacy and safety of
products. Today, informed consent as a concept is much more widely debated than hitherto. I am arguing that whether kuru has been the result of a trial or the result of a vaccine or product in general use that was contaminated, should remain an open question until further archival research can be carried out.
Chapter 6

Prion inquiries

The thesis up to this point has concentrated primarily on research that occurred during the first half of the twentieth century related to kuru, scrapie, louping ill, MVE and X disease. One of the aims has been to bring into sharp focus a picture of contamination problems associated with vaccines and medicines. Here, I focus on the inquiries that have been held on iatrogenic CJD and BSE during the second half of the century.

Prion diseases are extraordinary in many ways. They are paradoxical because many archival records exist simultaneously with a paucity of information in the public domain and to a lesser degree in the general scientific and medical community. In many cases, in the public domain knowledge does not become available until many years after the events and only then following an Inquiry. And when it is made available to the public and the wider scientific community, it is buried in weighty reports. The BSE Inquiry released in October 2000, known also as the Phillip’s Inquiry, runs to 16 volumes¹, condensed from many thousands of documents and transcripts of evidence, some of which I use in this chapter. The Report on the inquiry into the use of pituitary-derived hormones in Australia and Creutzfeld-Jacob Disease (Allars 1994) runs to over eight hundred pages. Kuru research similarly, brought about a mass of correspondence, much of which has been published, although not until long after 1957. In addition to the published information, Burnet kept nearly forty files on kuru research. Gunther also kept three files on kuru, although, generally, he ‘was not a diary writer or a keeper of government files’ according to Nelson (1996:190). Prion disease research seems to engender the keeping of personal files. Sir Donald Acheson, Chief Medical Officer (CMO) in Britain during the BSE crisis, kept personal files on BSE quite apart from his files related to his role as CMO. But despite this wealth of information on prion research, one official at the BSE Inquiry remarked that members of the Inquiry committee must have found an ‘absence of

¹ Mann (2000: 8)
footprints’, as they attempted to piece together a picture of events relating to the administration of BSE within the British government².

Judging by press reports and television news segments on BSE during the late nineteen nineties, which focus predominantly on the risk of eating beef, it could be argued that the absence of footprints in the BSE situation is related to food safety. I am not qualified to say whether this is the case for I have not focussed attention on this issue. What I can say, having researched the safety of medicines, is if there is a problem at all, it is precisely here that the greatest danger was thought to lie.

The aim here is to highlight how the dissemination of information about the safety of medicines in relation to prion diseases echoes the situation in relation to the transmission of information to the public domain about the safety of vaccines. The phrase ‘absence of footprints’ tends to suggest that, at least in the BSE situation, information about medicines might have been contained within a specialist group of researchers and officials.

Information in the area of vaccine research also stays within a closed circle judging by a comment made in 1967 by Sir Graham S. Wilson from the London School of Hygiene and Tropical Medicine, who stated that:

> The risks attendant on the use of vaccines and sera are not as well recognised as they should be. Indeed our knowledge of them is still too small, and the incomplete knowledge we have of them is not widely disseminated (Wilson 1967: 4).

Brogan in his history of CSL also comments about the lack of dissemination of information about pharmaceutical products, saying that this is because of the ‘ethical nature’ of the products. Thus information moves no further than health professionals and influential figures in science, industry and government (1990:241). It is this sort of thinking that I am seeking to challenge because when the information does become public knowledge, public trust is harmed. I will provide more detail about the secrecy that has been spoken about at the BSE inquiry surrounding the Medicines Commission in the next chapter. In this chapter my aim is to focus on specific events

² BSE Inquiry transcript Day 50 - Lister, Cunningham, Bridges.
over the past fifty years relating to the management of data in order to gain a picture of the complexities, uncertainties and tensions that have marked prion research and its administration. As Brian Wynne (1996) argues, these uncertainties, which are part of scientific practice, are rarely transmitted to the public domain when government assurances about safety are given. But in making these uncertainties explicit, it might be possible to learn something about the culture that seems to operate in order to change it. I stress that my interpretation of events is not aimed at alleging wrongdoing on the part of individuals who operate within the culture.

I want to introduce what I call a ‘culture of evasion’ to explain the situation, at least in part. Evasion in this sense is not to be understood as referring to an individual characteristic, although individuals live and work within cultural norms that constrain what can be spoken about openly and it is difficult to speak about the culture without showing how this operates on an individual level. In what follows, while I provide specific examples that do involve individual actions, it is important to keep in mind that I am talking about, what I see as a cultural phenomenon. One definition of evasion is to ‘pass-over’ (MED). This implies overlooking something and it is in this sense that I am using the term. I am referring to a systemic problem of a culture, which does not seem to be able to acknowledge problems when they arise, whether this is on a conscious or unconscious level.

The culture that seems to predominate, particularly as it relates to the CJD and BSE situation, can be described also as one that lacks transparency. It is one where the issues of iatrogenic reactions to medicines do not seem to be faced squarely, but instead are avoided until the situation becomes so untenable that an Inquiry is called for. It is as though the climate of secrecy that existed during the post-war period relative to disease and weapons research has continued in later decades but now it serves other interests, such as big corporations, pharmaceutical companies, reputations and profit. Attached to these concerns is employment for a large number of the population. It is therefore understandable that such interests are protected. But I would ask, at what cost? The system about which I’m speaking is pervasive, no doubt, in other areas of research, here though I concentrate only on prion research.
Two useful concepts that might help to explain the culture in which we live are division of labour and alienation. Marx pointed to the alienation experienced by workers resulting from the modern manufacture of products. That is, no one individual is responsible for the total manufacture of a product; different people carry out each segment of the process with little input into the work of others. This can lead to a dispersed responsibility for safety. As a particular example, when decisions were being taken in the 1970s about the safety of pituitary hormone in Australia, members from all of the committees with responsibility for the product met together and discussed the exclusion criteria required for safe collection of pituitary glands. No one person though, had overall responsibility for dissemination of the material to pathologists to ensure the manufacture of a safe product (Allars 1994). This highlights part of the problem of specialisation of knowledge and points to some of the difficulties surrounding division of labour within scientific disciplines.

Romanyslyn’s work, *Technology as Symptom and Dream* (1989), might also help to explain the situation. Citing the work of Joseph Weizenbaum, Romanyslyn writes about the psychological distance that is evident in scientific research culture. Distance, in this sense, is ‘not a matter of measure but of attitude’ (Romanyslyn 1989:21); it relates to the way one seeks to gain mastery over nature and to the way people are viewed as apart from nature rather than as a part of nature (p.28). As an example, when American scientists advised the Defense Department during the Vietnam War, Weizenbaum has remarked that

> These men were able to give the counsel they gave because they were operating at an enormous psychological distance from the people who would be maimed and killed by the weapons systems that would result from the ideas they communicated to their sponsors. The lesson, therefore, is that the scientist and technologist must, by acts of will and of the imagination, actively strive to reduce such psychological distances, to counter the forces that tend to remove him from the consequences of his actions (in Romanyslyn 1989:21).

Such a notion may be useful when applied more broadly to scientific research. Could it be that the lack of overall responsibility demonstrated in the pituitary program is related to this notion of psychological distance? It is worrying when statistical tables outlining the deficit in pituitary glands, take precedence over the dissemination of critical information required to produce a safe product, which is the situation that
exist in the nineteen seventies. This example highlights the tension involved between maintaining safety of a product and furthering the needs of science to help people.

Brian Wynne's (1996) analysis of public understanding of science is also useful to explain the culture, particularly as it relates to 'trust', 'risk' and the dominant model of scientific practice. Trust, as Wynne shows, is an ambiguous concept. It is something that calls for constant negotiation between the public and officials. It is not simply a matter of the public choosing which scientific pronouncements to agree with. And allied to the concept of trust is 'risk'. Wynne also shows that ideas about risk in the public's minds are formulated through their own experience of living with risks and with past experience in obtaining information from government officials and scientists, which in some cases is not forthcoming. This can lead to skepticism. The main thrust of Wynne's paper, which focussed on the perceptions of hill sheep farmers in Cumbria around the Windscale nuclear plant, is that members of the public, in Wynne's example farmers, have their own knowledges to draw on which often do not correspond with government pronouncements of safety. He argues that governments and scientists need to reassess the epistemological grounding of the concepts that they use to explain what risk is and how public trust is negotiated. While there is no doubt that government officials do not want to cause anxiety by informing the public of hazards, it might be wise to take on board Wynne's analysis. This means re-evaluating the model of what it is to be human, to shift from thinking of humans as purely rational actors and to incorporate the hermeneutic elements of relationship between the public, scientists and officials. In addition, this calls for a re-thinking of the model of science, which is formulated on control and prediction, as this model fails to incorporate the relational elements between humans. Wynne also shows that this model of science does not correspond with practice on the ground. As I will later show, this model of science also does not correspond with the reality in some situations that occurred in relation to the science and medical practice relating to the administration of kuru, the pituitary program and BSE.

While this chapter is primarily concerned with iatrogenic CJD and BSE, I want to begin with a brief survey of some of the tensions and uncertainties involved in the study of kuru around the time of the Committee of Inquiry held in 1959.
Kuru and the Committee of Inquiry

The Kuru Committee of Inquiry was held at the School of Public Health and Tropical Medicine at the University of Sydney in December 1959 in Australia to look at ways to control the spread of the disease and to discuss further research. At this time, considerable tension existed between some of the Australian and American researchers involved and the committee aimed to resolve these as well as determine how the research could be progressed with minimal conflict. There was a feeling by some that the research needed to be centrally controlled and co-ordinated in New Guinea and that a general lack of order and discipline was evident.

One source of tension came in May 1959 when an urgent telegram was sent to ensure that Dr Fortune did not leave for Europe with a patrol report of the south Fore region, but to no avail as a telegram three days later indicated. Just prior to the Committee of Inquiry on December 15th, Professor Robson from the University of Adelaide wrote to Dr Scragg, Director of Public Health in New Guinea, about his concerns. He suggested that ‘planning, order and discipline’ were necessary in any research work and these elements were ‘doubly necessary in the unusual circumstances of this problem’. In Robson’s view, none of the above had been in evidence in the kuru studies leading up to this period. A few months prior to the meeting on September 29th, Scragg expressed the view in a letter to Dr Zigas, that:

We must maintain some record at a central point and ensure that there is no breakdown as in the past by the disappearance of all information from the Territory.

Scragg’s concerns emanated from information he had been given that ‘some patients who received extensive medication during 1957’ were still alive and some of these patients received ‘more than one trial medication’. He believed that ‘the actual records of what these patients received’ were not available except to Zigas and Gajdusek, and then he thought that Zigas did not actually have the records. Scragg

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3 Series 10/6 BP.
stressed the need for data to be collected and stored of any trial treatments that were undertaken on the patients with kuru. He was not opposed to Gajdusek working in the area in 1959 but suggested that a summary of the trials should go to Okapa within one month of the date of discharge of the patient. Scragg suggested to Zigas that it was vital that all research workers cooperated with each other in the area.\(^8\) As a gauge to how the area was perceived at this time, Scragg noted that the laboratory was not ‘adjoining rooms in a building’ but rather ‘the whole of the Fore area’. It is a sign of Burnet’s influence in handling the committee that Smadel wrote to him in 1960 suggesting that if there was a New Guinea Peace Prize Burnet should be awarded it for negotiating two thirds of the area for Australian researchers, leaving one third for American investigators.\(^9\) This should not overlook however the considerable tension and uncertainty at this time related to the best way to carry the research forward. And while there was an effort made to contain and collate information about kuru in New Guinea, because of interest in the disease by other researchers this did not always prove possible. This highlights the messy situation that existed on the ground involving kuru research, which can be contrasted with a view of scientific practice as being tightly controlled.

The extent of the problems at this time were considerable and this is evident in a private letter that Professor R. Fisher wrote to Burnet in November, just prior to the Committee of Inquiry. Fisher expressed the view that the ‘politicians will need all the scientific guidance they can mobilize, if there is not to be something of a disaster’.\(^10\) He believed that the Australian Government had ‘officially full responsibility’ and he was concerned that if Gajdusek was in ‘any position to claim first hand knowledge the Administration will be held up to blame and contempt by the whole propaganda machine of the American (illegible) [press?]’. Fisher was puzzled how Gajdusek was able to gain support, when in his view little had materialised ‘to any impressive extent’. In fact, he was puzzled by the whole affair, for while he agreed with

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\(^8\) At this time, Zigas had just been made a member of the American Academy of Neurology - ‘an honour rarely bestowed outside America’, according to a SMH report dated September 18th 1959.


\(^10\) Letter from Fisher to Burnet dated November 17th 1959. BP. Series 10/16. Professor Ronald Fisher was a mathematical statistician who visited Okapa in August and was asked by Professor Bennett in Adelaide to attend the December kuru Inquiry meeting as consultant geneticist. Bennett requested Dr Gunther’s permission in December 1959. Gunther replied by reply paid telegram on December 7th, saying that Dr Scragg ‘advises Fishers presence’. Telegram from Gunther to Bennett dated December 7th 1959. Kuru file.
‘Bennett’s genetical theory’ current at the time, he remarked that there was ‘still a mystery in the background’ and some factors related to the population-genetics were ‘very peculiar’.

It is not clear from Fisher’s letter exactly what was meant by the comment that scientists and politicians needed to converse, but it resonates with one made by Horwitz at the First International Conference on Vaccines Against Viral and Rickettsial Diseases of Man in 1966. Horwitz suggested that ‘scientists and other important elements of the intellectual community’ needed to maintain an ‘active association with Governments, regardless of currents of political opinion’. He pointed out, that ‘as in any attempt to explain all phenomenon of the real world, the process is never ending’ (Horwitz in WHO 1967:xxi). Dialogue was necessary because of the complexities involved in vaccine research and because the health of the population was at stake; the ‘moral imperative’ to provide vaccines to the population, he said, needed to be balanced by knowledge of the problems.

Whether the problems experienced in kuru research were similar to those spoken about at the conference in 1966 is difficult to say. But to correlate these events with events described in the previous chapter, at the time of Fishers visit to New Guinea in August 1959, Gajdusek was corresponding with Hadlow in England about the relationship between kuru and scrapie. In November, one month prior to the Kuru Committee of Inquiry, the meeting was held in America to allay concerns that had resulted from the scrapie eradication policy. Following the Kuru Committee of Inquiry, a quarantine zone was declared around the Okapa patrol post restricting the movement of people in and out of the region.

This brief account of the kuru research project highlights some of the tensions involved. The main problem seems to have been that the research data was not coordinated centrally by Australian researchers, which resulted in critical information about the results of trial treatments being unavailable in New Guinea. Lack of coordination was also a problem during 1956 in relation to who would coordinate the research planned by Smadel and Burnet for New Guinea. However by 1962 Burnet and Gajdusek both agreed that coordinating the research was not as essential as disseminating freely what work was being conducted and the work proposed. So
while a lack of coordination was seen as problematic by some of the researchers involved during the early stages of the project, this view was not shared by all concerned in 1962.

The Pituitary Hormone Program and its Inquiry

On May 11th 1993, Associate Professor Margaret Allars was requested by the Minister for Human Resources and Health to examine the operation of the Australian Human Pituitary Hormone Program. The Report on the inquiry into the use of pituitary-derived hormones in Australia and Creutzfeld-Jacob Disease was released in June the following year (Allars 1994). Here, I examine two main areas of the findings of the inquiry relating firstly, to how information about side effects of the hormone was collected during the early stages of the program, and secondly, to show how information about the collection of pituitary glands was disseminated to pathologists. Through an examination of these two areas, which both have implications in terms of safety for recipients of the hormone, it will be possible to assess how well the program met its stated aims, which were:

To organise the collection of pituitary glands, the extraction and purification of pituitary hormones and the distribution of HGH and FSH. The HPAC [Human Pituitary Advisory Committee] was also charged with the duty of collecting and collating the data obtained regarding the treatment of dwarfism and infertility

The program gained official recognition from the Department of Health in Australia on August 11th 1967 after many informal and unofficial meetings that began in

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11 Cited by Allars (1994:20) as stated in an article published in the Medical Journal of Australia in 1969. In a letter to the Director General of Health, Professor Leslie Lazarus stated that the proposed role of the Advisory Committee was to include 'an opportunity and responsibility to collate the results of therapeutic trials and investigations (Allars 1994:521 citing letter of August 3, 1966, from Lazarus to Director General of Health). In Australia, the Fractionation Subcommittee, a subcommittees of HPAC, discussed trials of Follicle Stimulating Hormone (FSH is the fertility component of pituitary hormone as opposed to HGH, which refers to the growth component). The implementation of the trials was then referred to the Advisory Committee for discussion at the January 20 meeting of 1967, at which it was decided to distribute 600 ampoules of hormone to a Sydney and Melbourne team for a "comparative clinical trial". 300 ampoules were to go to each team (Allars 1994:521).
1962. The first official meeting of HPAC took place on Sept 15th 1967. Below are a few examples to show the different ways in which the program attempted to meet its primary objective.

Beginning in 1969, medical practitioners were required to submit a quarterly return showing unused stock of the hormone to the FSH Subcommittee, one of the subcommittees of the Advisory Committee. In early 1970, a computerised database was set up to record the use of pituitary fertility hormones in order that the data could be analysed. The Australian Health Department approved the introduction of the computer database, and the system was apparently, established by Professor Cox, the FSH Subcommittee and a departmental officer. Special forms were designed to illicit information on batch numbers, daily dosage, and the results of the recipient’s hormone levels. This shows that an attempt was made to collect data, but analysis was inhibited because, as Cox explained to the Inquiry, one of the computer manuals was lost which prevented some of the anticipated research and a detailed comparison of the relative success of hPG treatment regimes. Here, it seems that too much reliance was placed on a new technology. But this does not explain why a manual analysis of the data did not occur in the absence of the computer manual, because an attempt was made to collect this critical information. There were however other reasons why information was difficult to obtain.

One treating medical practitioner and a member of the FSH Subcommittee interviewed by the Allars inquiry, remarked that:

There were one or two workers that there may have been problems. I won’t mention names and we got sick and tired of the forms, this was when I was on the Committee, of them not being completed. We had no idea of what one or two workers were doing.... I remember that and we had no record of how much he had given the patient if anything at all. We never received a bona fide treatment sheet but he was well known for it (in Allars 1994:208).

According to the Allars Report, one doctor stated in a newspaper article in 1971 that

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16 Allars 1994:206. hPG refers to human pituitary gonadotrophin, one of the fertility hormones.
he saw the collection of data as departmental red tape and noted that he would give
the committee the figures at his convenience rather than at their demand\textsuperscript{17}. The
individual paid a price for non-compliance and was prevented access to the hormone
by the subcommittee at the time, but supplies recommenced in 1974 following a
decision made by the FSH Subcommittee, which the Advisory Committee accepted.
The price of not adequately monitoring the hormone supply and its effects might have
been much higher however for those who were the recipients of the hormone.

The iatrogenic effect of hyperstimulation of the ovaries was a major problem
associated with the use of pituitary hormone. The Allars Report notes ‘a concern on
the part of some obstetricians that ovulation induction be regulated because of risks of
hyperstimulation and multiple births’, but these concerns ‘... did not predominate’\textsuperscript{18}.
In 1971, one recipient of hPG delivered nine babies, none of whom survived. The
situation was recorded in the minutes of HPAC meeting on June 25\textsuperscript{th} that year, but the
situation was described euphemistically in a report to the Minister for Health, as ‘... just an unfortunate and bizarre occurrence’. The report did go on to note however that,
‘... it is also possible that unreliable hormone assays and unrecognised
overstimulation were responsible’\textsuperscript{19}. As Allars points out, ‘[i]n general the problem of
hyperstimulation was regarded as a matter for individual treating medical
practitioners’\textsuperscript{20}, and the development of appropriate treatment regimes was left to the
treating doctors to decide\textsuperscript{21}. It would seem from this statement that data on the
iatrogenic elements of the program during the early years, while available, was
scattered rather than coordinated, analysed and disseminated to ensure safe practice.

Use of the hormone by 1978 was no longer considered to be in a trial stage but rather
“routine treatment” as the main side effect of hPG was said to have been eliminated\textsuperscript{22}.
It is difficult though, to see how the transition from research trial to routine treatment
in Australia occurred, for it would have been hard to know what routine to follow,
without carefully analysed data on which to base a decision about treatment. Allars

\textsuperscript{17} Allars 1994:208.
\textsuperscript{18} Allars 1994:505.
\textsuperscript{20} Allars 1994:209.
\textsuperscript{21} Allars 1994:207.
\textsuperscript{22} Allars 1994:520 citing J. B. Brown’s paper, Pituitary Control of Ovarian Function- Concepts
Derived from Gonadotrophin Therapy (1978:47).
(1994) highlights the grey area between trial and treatment and stresses the need to delineate between the two areas. This brief examination of how data was collected and collated in the Australian program shows how difficult it was to meet the objectives of the program. The method that operated seems to be characterised more by trial and error\textsuperscript{23}, rather than the rigour that is said to underpin medical practice, and stands in contrast to a view of science and medical practice as being strictly controlled. When this information is revealed it does not engender a sense of trust.

Neither do some of the irregularities in the program’s operation that were reported on in the Executive Summary of the Allars Report. The program operated outside of normal channels in some respects. Allars (1994b) writes that ‘The pituitary hormones were dealt with in a manner inconsistent with the normal procedures for listing and testing of pharmaceutical benefits’. The National Biological Standards Laboratory (NBSL) failed to test the hormone for viral contamination. ‘The Director General of Health failed to insist that advice from NBSL on viral contamination be provided prior to making the recommendation to the Minister that the hormones be listed’ on the Pharmaceutical Benefits list. The Pharmaceutical Benefits Advisory Committee (PABC) made recommendation in 1964 and 1967 for listing of the hormones’, but the PABC ‘appears to have largely been circumvented as negotiations proceeded directly between members of HPAC and the Director-General of Health and the Minister’ (p.6). The hormones were listed in accordance with “special arrangements” under section 100 of the National Health Act 1953 (Cth). The Inquiry found that the listing of the hormones under section 100 ‘involved an abuse of power and was legally invalid’ (p.7). HPAC believed that they had the power to release hormone for research purposes under an Order in Council of the Governor General made in 1969 under section 9 of the National Health Act. The Inquiry concluded that the Order in Council was made ‘in excess of power and the research allocations of hormones was unlawful’ (p.7). ‘The Inquiry has formed the conclusion that glands were generally removed not for the purpose of post-mortem examination, but for the purpose of supply to CSL.

\textsuperscript{23} Regarding the comparative trials undertaken in Australia, one doctor interviewed at the inquiry said that the trial was initially an exercise in “clearance rate assessment rather than if treatment happened to work as a by-product” (Allars 1994:527). This seems to imply that clearance rate assessment was the priority not fertility promotion or growth promotion. The same doctor said that the normal control group were often people “sitting around in hospitals doing nothing”, they were people who "happened to be lying around in the ward" (cited by Allars 1994:527).
The use of the glands during the period of the human tissue legislation was therefore unlawful’ (p. 6). Uniform human tissue legislation was enacted following the Law reform Commission Report in 1977, and by the early 1980s each State had implemented the legislation.

Without drawing to close an analogy, this situation resonates with the one that existed between 1956 and 1961 when Bazeley was working on polio, and Gajdusek was working on kuru, at which time there were considerable issues related to manufacturing safe vaccine. This is not to suggest the same individuals were involved in the pituitary hormone situation but rather to highlight the common element in both time frames, namely, operating outside of normal administrative channels.

I turn now to the manufacture of pituitary hormone, a process where glands were required and decisions were necessary about which glands to include and which to exclude. Instructions about the exclusion criteria changed over time depending on new information becoming available, and these were provided to technicians and mortuary staff. Below are sets of exclusion criteria formulated between 1966 and 1985, sourced from the Allars Report (1994:57-65).

I draw attention to the term “neurological”, which first appeared in the 1971 criteria, for by this time knowledge had become available to show that prion diseases like scrapie in sheep, and kuru and CJD in humans could be transmitted experimentally through diseased neurological tissue. While the term appeared in the guidelines at this time, when the information in the 1971 guidelines was transmitted to pathologists, the term disappeared. Here I track the process through which the term gained recognition, was lost, and in the end completely disappeared from the guidelines provided to pathologists and technicians, thereby reducing the safety of the product.

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The 1966 exclusion criteria:
- known virus infections, particularly viral hepatitis
- disseminated malignant disease
- disseminated infections such as septicaemia and pyaemia
- obvious pathology of the pituitary gland

The 1971 exclusion criteria:
- active viral hepatitis
- neurological diseases of the central nervous system due, or possibly due to viral infection
- obvious pathology of the pituitary gland

The 1977 exclusion criteria:
- diseases of the pituitary gland
- infectious or serum hepatitis, and
- acute septicaemia

The 1982 exclusion criteria:
- diseases of the pituitary gland
- infectious or serum hepatitis
- acute septicaemia
- Presenile dementia (Creutzfeldt-Jakob disease)

The 1985 exclusion criteria:
- diseases of the pituitary gland
- infectious or serum hepatitis
- acute septicaemia
- Acquired immune deficiency syndrome (AIDS)
- Dementia of any type

In 1971, Dr. McGovern, a pathologist and a member of the Advisory Committee, gave two warnings about slow viruses\textsuperscript{25}. Seemingly unconvinced by the warnings, the committee sought the second opinion of the College of Pathologists, who responded in writing in agreement with McGovern\textsuperscript{26}. The letter from the College was discussed at a meeting of the Advisory Committee on June 25\textsuperscript{th} the following week and the exclusion criteria were amended to include the critical category "neurological" disease\textsuperscript{27}.

The new guidelines were published in the *Newsletter* of the College of Pathologists.

\textsuperscript{25} The first warning was given during a meeting of the committee, the minutes of which state: Dr McGovern ‘... informed the Committee that the pituitary glands obtained in the presence of certain chronic neurological disorders might be suspect’ (quote from HPAC Minutes of meeting, 19 February, 1971 item 2.7. cited by Allars 1994:60).
\textsuperscript{26} Allars 1994:60.
\textsuperscript{27} Allars 1994:61.
more than one year later in July 1972. Putting aside the seemingly tardy response, the layout of the exclusion criteria made recognition almost unnoticeable. The Allars Report describes the reasons for this in the following terms,

"The criteria were described in the midst of an article which commenced with a request from Dr McGovern for pathologists to cooperate in the collection of glands, and then provided a description of the procedure for collection, and the role of CSL and HPAC ....

"The paragraph containing the [1971] exclusion criteria commenced as the second paragraph in the first column of the Newsletter, but was interrupted by a table of statistics which ran across the entire page. These statistics showed the number of glands collected in each State and the deficit in each State from its expected total. As a result the text of the article diverted briefly to the top of the second column and then returned and continued down the first column.

"... the exclusion criteria (omitting the word "neurological"), appeared at the top of the second column and were likely to be missed altogether even by a careful reader of the Newsletter. None of the pathologists interviewed by the Inquiry recalled reading the criteria in the Newsletter (Allars 1994: 67-68 underline added).

The exclusion criteria were lost in messages with entirely other agendas like promoting the program and gland collection and so might not have assisted in the making of a safe product. Moreover, the critical term "neurological" was similarly lost.

When the guidelines were again re-formulated in 1977, the significant term was dropped altogether, while septicaemia, a category that had been inexplicably removed in 1971, reappeared\textsuperscript{28}. The Fractionation Subcommittee endorsed the Instructions prepared by CSL, as outlined in \textit{Draft Instructions for Collection of Pituitary Glands} (1977), and submitted them to the Advisory Committee for approval. The new instructions were briefly discussed at the November meeting in terms of how to improve the collection of pituitary glands, but no discussion about the content of the criteria, if it took place, was minuted.

The Allars Inquiry found that the Advisory Committee, the Commonwealth Serum Laboratory and the Fractionation Subcommittee were not able to account for the

\textsuperscript{28} Allars 1994:61-62.
decisions taken at this time. No record appears in the minutes of the committee meetings at the time to explain why the term had been dropped. One member interviewed by the Allars Inquiry expressed surprise that it had been. A member of the Advisory Committee said, "I haven't seen it before" when shown a copy of the 1977 exclusion criteria. Records provided by CSL to the Inquiry also reveal no explanation. Interviews with 'key personnel at CSL also failed to shed any light on the omission', and Professor Lazarus 'did not remember the change ever being put' to the Advisory Committee. It is as if the term was dropped on a blindspot.

However inadequate the previous attempts to disseminate this critical information had been, no attempt was made to publish the update in the pathologist's Newsletter when the exclusion criteria changed in 1983 and 1985, while new instructions were sent to CSL in 1985. The Allars Report came to the conclusion that no one person or institution had 'responsibility for ensuring that pathologists and mortuary attendants were made aware of the exclusion criteria'. This suggests again that a lack of coordination was the problem. But it is difficult to understand how some of the senior researchers, scientists and doctors involved in the program were so seemingly ignorant of the scientific knowledge available throughout the 1970s about the transmission of slow viruses through diseased neurological tissue.

One of the reasons for this apparent lack of knowledge, outlined in the Allars's Executive Summary, relates to the specialisation of knowledge. The summary of the report notes that knowledge about CJD was very specialised at this time and only resided within the field of neuropathology: '[n]europathologists, including Gajdusek, did not make a link between their knowledge of the transmissibility of CJD and the use of pituitaries in growth hormone programs' (1994b:2). Here it needs to be considered that Gajdusek and Burnet, in 1962, discussed how the pituitary gland was specifically implicated in the pathogenesis of kuru. So knowledge existed about the role of the pituitary gland in the pathogenesis of kuru when the program began informally in 1962 in Australia. By 1968, knowledge was available that CJD and kuru could be transmitted experimentally. And in 1971 McGovern suggested caution.

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30 Allars 1994:68.
regarding the potential dangers of transmitting slow viruses. Whatever the answer to this question, after 1976, at which time the committee process was reviewed and downsized by the government, McGovern was no longer a member, which left the committee without an expert neuropathologist altogether\textsuperscript{32}.

Another reason why the link between CJD and kuru might not have been made is because the fractionation subcommittee determined that "the only slow virus disease causally linked to human disease was kuru"\textsuperscript{33}. The Allars report notes that this 'groundless assertion can perhaps be explained in part by a perception that kuru was confined to certain native people of Papua New Guinea and members of the Australian community were not capable of contracting it'. It is not clear from the report whether the relationship between kuru and cannibalism hindered developments during this period. What is clear is that one pathologist interviewed by the inquiry noted that 'the story of kuru was of general interest to people who were not in the medical profession because of the cannibalism aspect of the story'\textsuperscript{34}. Questions remain therefore, about how much this might have also influenced members of the committees involved in the pituitary program. Whether this was the case or not, in conclusion, the fractionation committee seemed to be operating on 'outdated general knowledge' about what was possible in terms of iatrogenic transmission of slow viruses\textsuperscript{35}.

Also in need of consideration is the fact that by 1978 there were other means of transmission of CJD surfacing. Gajdusek and Burnet discussed iatrogenic transmission of CJD through the use of contaminated surgical instruments in January and March that year. Following the January conversation, Burnet made a note about 'the transfer of Kreutzfeld Jacob disease (KJD) to humans by surgical transfer from instruments previously used in neurosurgical manipulation in KJD patients'. The note continued, 'Beck seems to find quite regularly in squirrel monkeys infected with KJD virus that at an early stage of infection there is evidence of nuclear division? anabolic?

\textsuperscript{32} Allars 1994:351.
\textsuperscript{33} Allars 1994:357.
\textsuperscript{34} Allars 1994:315.
\textsuperscript{35} Allars 1994:358.
abnormal mitosis and only later the typical appearance of vacuolation spongiosis etc.\textsuperscript{36}

The note made after the March conversation provides information about the current hypothesis as regards iatrogenic transmission. The relevant passage reads:

"Long and to some extent very irregular incubation periods kuru eg from 2 to 30 years in man. No evidence of immunity or antibody in any of the diseases. A few groups of CJ cases are familial most are wholly sporadic in elderly people about 1 per $10^6$ per annum in most communities. Transmission seems to be wholly by iatrogenic actions involving penetration of brain or CSF cavity."

The note continued by summarising what is now well known: '"all the viruses are highly resistant to heat formalin UV and eth. dioxide sensitive to phenol and chlorine antiseptics or 120$^\circ$ autoclaving'; '"physically the activity is attached to minute fragments of membrane probably of endoplasmic reticulum', and '"unlike plant viroids not present in nucleus\textsuperscript{37}. Of special interest for the hypothesis then, and now, is the 'specific differences in infectivity species range and neuropathology of different strains, the capacity to change on species transfer, and the inability to produce any demonstrable immune responses\textsuperscript{38}."

This note is interesting because it is headed, 'Notes on kuru etc.', and because of the comment about transmission being wholly iatrogenic. The title implies that kuru and CJD were seen in the same mould and tends to give the impression that kuru

\textsuperscript{36} Burnet Papers Series 9/35.
\textsuperscript{37} Burnet Papers Series 9/35. The current hypothesis was that, "The agent is generated by informational error in control DNA resulting in the production of an anomalous intragenomic mRNA (or a protein derivative) which can pass from one cell to another and induce similar production of an excess gmrNA in other cells. This process may be of virtually no damaging effect and be shown only by an occasional doubling of neurons histologically and an accumulation of opportunities for further genetic error involving the affected process and influenced by various factors including the inherited quality of the genome and details of the initial error plus perhaps casual damage migrating nematodes[?] or viral infection in the CNS. This sequential error sequence gives rise then to the spread of a pathogenic agent through the CNS and other organs with destructive changes in appropriate parts of the CNS giving the standard disease picture. Once this stage has been reached entry of the agent into new individuals can induce the disease".

\textsuperscript{38} Thanks to staff at the University of Melbourne Archives for assisting me with numerous requests. Without their help, this document would not have been found as it is contained within Series 9, which contains Burnet's papers on molecular biology rather than Series 10, which contains the Kuru Papers.
transmission might also have been included in the iatrogenic category. The note also indicates that knowledge about iatrogenic transmission of CJD through surgical instruments was discussed in Australia at this time, at least outside of the deliberations of the committees involved in the human pituitary hormone program.

It was not until 1982 that the exclusion of pituitary glands from patients with CJD was added to the list of exclusion criteria, by which time the program was only a few years away from coming to an abrupt halt with the recognition that the hormones could produce CJD.

**Significant months in prion research: April/May 1985**

These months were critical in the development of prion research internationally, for while England, Australia and America grappled with the problem of iatrogenic CJD and contaminated pituitary hormone, England simultaneously grappled with the emergence of BSE. And while Australia was in contact with the National Pituitary Agency (NPA), the Food and Drug Administration (FDA) and the National Institute of Health (NIH) in America, the Director of the English Human Pituitary Program and members of the research establishment at Porton Down were communicating with their Australian counterparts. All were in need of each other’s advice and the situation was such that it was no longer possible to evade the issue of contamination.

Professor Leslie Lazarus, Chair of the Human Pituitary Hormone Advisory Committee received a call from the NPA on April 2nd informing him of a link between CJD and pituitary hormone. On April 11th Lazarus spoke with Clarence Gibbs at NIH about an American patient with CJD. Towards the end of the month (24th), the Director of CSL sent a letter to Mr Dodson, Head of the Therapeutics Division of Commonwealth Department of Health seeking information on legal

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40 Gibbs has been mentioned on previous occasions in relation to scrapie and kuru transmission studies carried out by himself and Gajdusek.
liability and indemnity arrangements. CSL telexed the FDA seeking their position the same day.

Concurrently, by May 7th, Dr Morgan at St Vincent’s Institute of Medical Research, Victoria, had received a letter from the Public Health Laboratories Service at Porton Down, who were seeking information about the Australian position on human growth hormone\(^{41}\). Two days later, the Chair of the Advisory committee telexed Porton regarding action that had been taken on April 24th\(^{42}\). On May 10th, Dr Bangham, Head of Britain’s Pituitary Hormone Program, telexed Lazarus and Dodson, informing them that the pituitary growth hormone program in the UK had officially been halted\(^{43}\). The same day, Porton telexed Lazarus and Wong regarding withdrawal of human growth hormone in the UK as of May 8th 1985. The program in Australia was officially suspended on May 29th, an urgent drug recall was issued, and a number of extraordinary meetings took place.

Without the Allars Inquiry, much of this information would not have been made available to the public. Many recipients of the hormone did not find out about the risk associated with the product until the press began to report on the issue in 1992, even though CSL had immediately sought advice on indemnity in 1985. In 1990, legislation was passed to enable the Commonwealth Serum Laboratories to be converted into a public company\(^{44}\) and the CSL Sales Act was passed in 1993 to allow for the sale. The situation is reminiscent of the Bazeley years at CSL when Cabinet reviewed the operations of the organisation and a Commission was appointed in 1961. Then too, it was proposed to sell off CSL, but the sale at that time did not go ahead\(^{45}\). Nevertheless, the similarity is worthy of note.

Here, it is also relevant to bring to mind the situation in 1962 when Burnet discussed finding a job in biological standards for Dr S. G. Anderson, at which time Bangham

\(^{41}\) The letter was passed on to Mr Wong, Acting Executive Secretary of HPAC.

\(^{42}\) That day, Wong sent a letter to the Department of Industry, Technology and Commerce requesting that the resolutions from the Fractionation Subcommittee be implemented. These related to biosynthetic growth hormone. As well, that day the government sent out the first letter to treating medical practitioners requesting that they advise patients of the risks of CJD, although this did not include patients who had received the hormone but who were not currently in treatment.

\(^{43}\) Allars 1994:560.


\(^{45}\) Brogan 1990.
was Head of the Medical Research Council. This shows one of the ways that research related to prion agents is related to research on non-prion agents over decades. Porton Down was also involved in prion research and research outside of this field, as from 1978 researchers were working on pituitary hormone (Allars 1994) and had previously conducted research on Japanese B encephalitis and louping ill virus during the period prior to 1967 (Gordon Smith 1967). In this last case, it is not clear if the same researchers were involved in both studies. What is clear, is that by April 1985 recognition of the hazards associated with prion research was such that it became impossible to overlook the issue any longer, for in England it was not simply a matter of grappling with iatrogenic CJD and pituitary hormone, BSE had also emerged. The connections between iatrogenic CJD research and BSE research are sometimes overlooked, although they did not escape the attention of Kevin Doyle, Australia’s Deputy Chief Veterinary Officer, who mentioned the links in 1996 at a meeting held in Sydney, Australia.\footnote{Kevin Doyle was the Deputy Chief Veterinary Officer, Department of Primary Industries and Energy and the Special Adviser, Quarantine, within the Australian Quarantine and Inspection Service (see in Davis p. 110). He said that the emergence of BSE came at a critical time; ‘when the Uruguay Round of Multilateral Trade Negotiations was commencing in Punta des Este, Uruguay’, when ‘investigations were being made into the spread of Creutzfeldt-Jakob Disease (CJD) among people as a result of the therapeutic use of material from cadavers’, when ‘the explosion of information technology which led to the availability of scientific information on the internet’, and when ‘the period in which human diseases, especially those associated with animals, were emerging in the public consciousness’ (Doyle in Davis 1996:112). The Proceedings of the Workshop of the Implications of International Disease Emergencies: BSE A Case Study October 30-31, 1996 were published in 1996 and edited by I.G.R. Davis. The report was put out by the Office of the Australian Chief Veterinary Officer and the meeting was sponsored by the Australian Animal Health Council Limited, the Australian Meat and Livestock Corporation and the Meat Research Corporation.}

**The BSE Inquiry**

In December 1997, the BSE Inquiry was announced and a report was released in October 2000, which is known as the Phillips Report, after the Chairman Lord Phillips of Worth Matravers. This section could well be called ‘another side of the story’, which is a phrase used by a member of the BSE Inquiry committee in reference to events surrounding BSE and the safety of medicines\footnote{\textit{Another side of the story} is a phrase used in the BSE Inquiry Transcript Day 84 – Curry.}. One of the aims of this final part of the chapter is to show that BSE was handled in the British Government during 1988 and 1989 as an iatrogenic issue as well as a food safety problem. With the help
of a few examples I aim to also show how difficult it has been for the inquiry committee to gather together evidence in this area - thus the relevance of the phrase the ‘absence of footprints’. But in spite of the absence of footprints, the BSE Inquiry has managed to piece together parts of the picture, which show the considerable amount of work that was carried out within government to address any potential problems in terms of maintaining safety. It was not simply a case of people doing nothing. I would argue that a distorted view of what actually took place occurs because parts of government departments were not open with the public about the issues and actions taken.

I turn to the situation in late 1988/early 1989, at which time, Medicines Division began to audit all pharmaceutical products for existence of BSE infected cattle parts. This involved 4000 letters being sent to pharmaceutical manufacturers along with guidelines to source bovine material from places other than Britain. Australia and New Zealand were the preferred options. The response rate to the letters by January 1990 was 94%, and in 1992 the British government announced that all products were free from BSE material. Dr Frances Rotblat, who worked at Medicines Division, played a critical role in the audit process from September 1988, and she is but one of the people who gave evidence at the BSE Inquiry of the situation that exists in relation to missing records. Rotblat noted in her witness statement that all of her ‘early papers on BSE were removed by a colleague and never returned’, and the colleague no longer worked at the Medicines Control Agency (MCA). The removal of the papers meant that Rotblat had to rely on papers made available to her by the Medicines Control Agency and the Department of Health.

Dr Gerald Jones, Head of Medicines Division in 1988, was also reliant ‘very heavily on the surviving correspondence, not all of which is complete’. In order to incorporate Jones’s assessment of the situation I want to describe events in May 1988 at which time a small group of government officials were discussing the implications

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48 BSE Transcript Day 79 Acheson.
49 Witness Statement No 422-Rotblat pt 32.
50 ibid pt 41
51 ibid. Medicines Control Agency is the name given to Medicines Division after 1989 when the division was renamed and restructured.
52 Transcript Jones G. 13/12/99.
of BSE for the safety of medicines. This will help to highlight how the implications of BSE were viewed.

On May 20\textsuperscript{th}, Dr David Jefferys, who worked in the New Drugs Branch of Medicines Division, received a note from Dr Hilary Pickles, the Scientific Secretary of the Department of Health\textsuperscript{53}. Jefferys replied on May 24\textsuperscript{th} saying that he was aware that a joint working group meeting had taken place between the Ministry of Agriculture Food and Fisheries (MAFF) and the Department of Health and Social Services (DHSS) and included the following preliminary thoughts to Pickles:

"... It also occurs to me that this is more of a long term issue and that it may well involve William Jenkins since this is rather more an ADR [Adverse Drug Reaction] problem than a New Drugs Group issue. I am therefore copying your minute to him. ..."\textsuperscript{54}.

Jefferys's expertise lay in the area of the safety of medicines, but he stressed at the Inquiry that he was responsible only for new medicines as opposed to existing ones, when BSE became an issue\textsuperscript{55}.

When asked at the Inquiry about the situation in 1988, Jefferys said that he thought that BSE would have been raised at a Divisional Management Group Meeting. Dr Gerald Jones, who was the Senior Principal Medical Officer Grade 3 of Medicines Division prior to April 1989\textsuperscript{56}, agreed, but he believed that the minutes 'may not be kept', as they would have been destroyed 'within about six months'. That the minutes of management group meetings should exist only for a period of six months say a lot about accountability in this part of the British government at this particular time. The significance of this should not be underestimated because, according to Jones, 'if there is an issue it is to do with the safety of biological medicinal products'\textsuperscript{57}.

\textsuperscript{53} Dr Pickles has been described as the 'key person' in the Department of Health relating to matters concerning BSE (Day 47). Previously, Pickles' work focussed on Aids related matters within the department.
\textsuperscript{54} In Transcript Jones G. 13/12/99.
\textsuperscript{55} Witness Statements Jefferys 419A-E Jefferys was the Principal Medical Officer in charge of the New Drugs Branch in 1986. Prior to this he had worked since 1984 as a Senior Medical Officer in the Medicines Division of the Department. During the period pre 1986, he was a member of the Committee of Review of Medicines. From 1986 he was also the Principal Medical Assessor to three committees: Committee on the Safety of Medicines (CSM); Biological Sub-committee of the CSM (CSM-B); and the Sub-committee on Safety, Efficacy and Adverse Reactions (SEAR).
\textsuperscript{56} Jones, G.
\textsuperscript{57} ibid
The concerns about the safety of medicinal products were underscored when Pickles wrote to Jones in June 1988, saying, "[w]e are clearly concerned that 'BSE-agent' may be transmitted in medicines". Thus Rotblat and a colleague, Dr Purves began to audit the situation by first conducting a computer search and then sending letters to the pharmaceutical companies.

In March 1989, the first report on BSE, known as the Southwood Report, was released. On February 9th 1989, just prior to the release of this Report, Sir Donald Acheson, Chief Medical Officer, wrote a minute concerning biological product safety in which he stressed that the Southwood Report made no explicit mention of the vaccine problem. Nowhere was there a reflection of concerns expressed by Pickles in a draft submission to the Secretary of State, in which was written, "we can't give any complete guarantee of safety for human medicines that use bovine materials in manufacture such as most vaccines". The relevant part of Acheson's minute of February 9th reads as follows:

... for some considerable time she has had serious concern about the safety of bovine-based vaccines in the light of the fact it has been discovered that contamination with placental material is a distinct possibility in the preparation of material for human vaccines derived from foetal serum.

This opinion gave Acheson 'sufficient cause for concern' and he asked the Minister to look into the matter with Medicines Division. The minute concluded by saying that he would amend the submission to the Secretary of State to the effect that the Southwood Report had not directly addressed the 'question of the safety of vaccines derived from bovine material' and he was 'making urgent enquiries' into the matter. Acheson acknowledged that he was trying to 'stir up more activity in the Medicines Division'.

Acheson believes that 'he broke Civil Service regulations by deciding to retain a personal file of papers relating to this condition'. But in early 1996, at which time he

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58 ibid
59 Transcript Day 79. Acheson & France. Sir Christopher France was the Permanent Secretary of the Department of Health and helped Sir Donald Acheson to understand 'how Whitehall works' when Acheson became a civil servant and CMO at the age of 57 years. France organised 11 joint meeting between Maff and DoH in 18 months.
60 ibid.
61 Witness Statement No 251 Acheson para 10.
was no longer CMO, he ‘called in at the CMO’s office’ to see his personal files and diaries and discovered that the diaries and ‘an unspecified amount of other material had been destroyed’ without reference to him\textsuperscript{62}. Acheson went to his former office because he wanted to refresh his memory following a report in 1995 of ‘a cluster of cases of CJD in young people’\textsuperscript{63}. He explains that the destruction of his ‘official diaries and some other papers’ was part of the official “weeding” process\textsuperscript{64}, and was done ‘in good faith in the interests of space’\textsuperscript{65}. While this is possible, a question remains as to whether the destruction of the Chief Medical Officer’s papers was related to his personal intervention in 1989 because of what he considered to be an inadequate response over the safety of medicines.

Acheson’s concerns began in March 1988, and at the inquiry he was referred to notes of a meeting held on March 17\textsuperscript{th}. The document outlines events that led to a meeting that was held at the National Institute of Biological Standards and Control on May 16\textsuperscript{th} 1988. Particular attention was drawn to paragraph 6, which reads,

“Biological products were produced of bovine origin and this applied to a significant proportion of insulin despite genetically manufactured sources. In addition cell cultures for many vaccines used a bovine serum medium. Dr Harris undertook to speak to the Director of NIBSC about biological products\textsuperscript{66}.

Acheson pointed out that ‘within that minute is coded if you like the fact that there was a potential problem’. In response to the question, “what is the coded message’, Acheson replied, ‘Well, the fact that he was going to speak to the NIBSC to get the advice as to how serious this was or not as the case may be’.

The exchange between Acheson and Counsellor Freeman over the issue of biological

\textsuperscript{62} ibid para 112.
\textsuperscript{63} ibid para 112. Having been away from the field since stepping down from office, Acheson could see by 1996 a distinct difference in the way scientists were talking about the BSE problem. The latent period and the BSE agents’ resistance to temperature, Acheson said, were different to scrapie. He notes that ‘the analogy with scrapie, which had been an important element in previous advice to the public was no longer entirely valid’ (para 113). Nevertheless, the situation in 1988-1989 needs to be assessed in terms of the thinking that existed then, which was that BSE would probably behave like scrapie.
\textsuperscript{64} ibid para 1.
\textsuperscript{65} ibid para 12.
\textsuperscript{66} see Day 79. see also Day 85 Newton & Moore.
product safety is telling in terms of the perceived urgency of the situation as of May 19th 1988. A minute written at the time notes a proposal put forward by Professor Southwood to “stop off the cattle feed source”; it “might be necessary to take steps to deal with any direct sources for humans”. “Minimalist steps” were mentioned “to cut off the various sources”. Meat inspection was given as an option. Of greater significance is the comment written at the bottom of Acheson’s handwritten note: “... we would need urgent advice on the question of the manufacture of biologicals from cattle material”. Freeman was interested to know if this was a concern Acheson ‘had had from the very beginning’, ‘You use the words here “urgent advice”; ‘How urgent was this to you?’. Acheson replied, ‘it was urgent’, before going on to suggest that he did not think it was any more urgent than taking action to remove the sick animals from the human food chain, but there was ‘no question that it was urgent’.

Here it is important to consider that other elements were at play, as Acheson went on to explain that he had of course to balance something here, that quite recently there had been a serious catastrophe due to incorrect information about whooping cough vaccine being publicised which led to a very substantial reduction in people who were prepared to offer their children the vaccination and a lot of illness as a result.

Since 1980, there had been ‘100 deaths from measles’ and Acheson thought about ‘50 deaths from whooping cough’. This situation seems to have affected the judgement of many involved in the BSE deliberations, as it rightly should have, but the question is, was the risk assessed as remote or moderately high? The urgency with which the matter was to be dealt suggests that it was not a remote risk under consideration. I will examine the perceived level of risk in various quarters in the next chapter. But here I want to show how difficult it is to obtain a clear picture of events given the time that has elapsed since the meetings that were held.

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67 Day 79.
68 ibid.
69 ibid.
70 ibid.
On February 1\textsuperscript{st} and 22\textsuperscript{nd} 1989, two meetings of the "Human and Veterinary Medicines Working Group" were held\textsuperscript{71}. The timing of the meetings is significant as they occurred just prior to the release of the Southwood Report, as Acheson was expressing concern to the Minister that Medicines Division was not adequately dealing with the issue of biological product safety. As the title of the group implies, efforts were made to coordinate actions taken with respect to both human and animal medicines and vaccines.

Rotblat attended the first meeting but had 'no recollection of what took place at this meeting'\textsuperscript{72}. It appeared 'from the documents' gathered by the inquiry that Dr Geoffrey Schild, Director of the National Institute of Biologic Standards and Control (NIBSC) attended the meetings. Schild also had 'no recollection of these meetings at all' and was 'not aware that a group of this name met on any other occasions'\textsuperscript{73}. The Head of Virology of NIBSC at the time, Dr Philip Minor, also remembered nothing about the meetings\textsuperscript{74}. Schild suggested that the passage of time had contributed to the inability to remember. There are 16 references to either, 'unable to recall', 'no recollection', 'do not recall', 'cannot remember' or 'cannot recall' in his statement relating to a number of meetings and correspondence. While much time had elapsed and the demands of both Schild's and Minor's positions were considerable, it is a pity that so little recall exists about what was discussed at these meetings, particularly when the role of the NIBSC is taken into account.

The role of the NIBSC is to ensure 'high standards of quality, safety, efficacy and consistency of biological substances used in medicines'\textsuperscript{75}. "Biological substances" are defined in Section 8 of the Biological Standards Act to mean 'substances whose purity or potency cannot, in the opinion of the Secretary of State, be adequately tested by chemical means alone'. In general, as Schild pointed out, 'these are substances prepared from complex biological materials which are incapable of being defined by chemical analysis alone'. What are required are 'specialised biological assays for their

\textsuperscript{71} Some of those in attendance at the February 22\textsuperscript{nd} meeting were Sir John Badenoch, Chair of the Joint Committee on vaccination and immunisation, Professor Asscher, Chair of the Committee on safety of medicines and the Chair of the Biological subcommittee. WS Rotblat pt 15.

\textsuperscript{72} WS Rotblat pt 11.

\textsuperscript{73} WS 575 Schild pt 45

\textsuperscript{74} WS 576 Minor pt 24.

\textsuperscript{75} WS 575 Schild pt 4.
testing and control. Included within this group are products derived from human blood, substances used in therapy like hormones and insulin, biotechnology products used in therapy like treatment for blood clotting disorders, interferons, interleukins and antibiotics. The brief of the NIBSC does not stop at the borders of Britain, as it acts as a World Health Organisation International Laboratory for Biological Standards. In this capacity it distributes a range of products including vaccines, ‘to enable manufacturers ... around the world to assay precisely the biological activity and potency’ of the products.

I turn for a moment to a report that emanated following a meeting held in May 1988 at NIBSC. In the report, Minor proposed that ‘studies should be set up with Wellcome Biotechnology’. It transpired that the studies were carried out independently by Wellcome. Schild did not recall seeing the results of the studies, although Minor did recall seeing them. It is not exactly clear from the transcripts and statements which studies are being referred to in this context, but in mid 1990 Pickles wrote to some of the manufacturers about whom there was still uncertainty regarding the source of bovine material. A statement from Wellcome that was copied to Pickles at this time confirmed that the company had been sourcing its bovine product outside of the UK for some time.

It would be wise to consider at this point, two reports that appeared in the press in October 2000, which indicate that in 1989, a Wellcome product (polio vaccine) was licensed and put on the market. In 1991, a company named Medeva bought the product from Wellcome. Medeva was then absorbed into Celltech in January 2000. This company was then sold to Oxford based Powderject pharmaceuticals in October 2000. Celltech are reported to have said that they relied on Wellcome for reassurances that the product was safe and had not been produced using BSE affected material. These events are significant because polio vaccine produced at Medeva was recalled in October 2000.

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76 *ibid* pt 4.
77 *ibid* pt 5.
78 *ibid* pt 7.
79 WS 576 - Minor pts 17 and 18.
80 WS 575 - Schild pt 41.
81 WS 115 - Pickles pt.5.4.
It is interesting that polio vaccine came into focus at the BSE Inquiry. I have attempted to show in earlier chapters that there have been concerns about polio virus throughout the century. Poliomyelitis virus and loping ill virus were both suspected of being the cause of X disease up until the nineteen thirties and polio vaccine posed problems during the nineteen fifties while kuru and scrapie research was being conducted. I have also shown how Hadlow not only made the link between kuru and scrapie but also had previously researched polio vaccine contaminants. Knowledge about these events should provide reasons for extreme caution in the future in relation to the safety of medicines.

Without the BSE Inquiry, the other side of the BSE story would not have been available to the public. But judging by comments made in a newspaper article since the release of the 16 volume Phillips Report, it would appear that the issue of medicines has not been fully addressed. James Meikle wrote the following comment in the Guardian Weekly on February 15-21 2001 about the British government, who

... promise[d] last week to review again the safety of vaccines that used material from cattle over the past 30 years, to consider opening up the secret licensing process for medicines, and to prepare contingency plans for the consequences of new BSE-like diseases.

This chapter and the next one might help to foreground some of the issues and point to relevant details, as we await the findings of the review.
Chapter 7

The safety of medicines and the prevention of prion diseases

The last chapter began to show some of the tensions involved in this complex field of prion research and administration. The main aim of this final chapter is to discuss vaccination policy both in England and Australia with respect to what is known about the potential to transmit prion diseases through vaccine and medicines. Judging by the following comment made by Norman Baker, a British Member of Parliament, there is good reason to do so. Baker suggested that,

“There has been a terrible averting of eyes on anything related to vaccines in the last 11 years. The whole reason seems to be that the vaccination programme must not be undermined....” (Baker in Meikle & Watt 2000: 8)\footnote{Norman Baker is the Liberal Democratic M.P. for Lewes, England.}

I argue that in its current form vaccination policy needs to move onto safer ground. In order to develop this argument the chapter examines policy in England and one of the factors that influenced policy decisions during the late nineteen eighties: a campaign for Measles, Mumps and Rubella vaccination that was implemented in England in October 1988\footnote{BSE Inquiry transcript Day 84. Currie.}. At the same time as decisions were being made about the safety of medicines relative to BSE, there was a move to improve the rate of uptake of vaccines. Hence the significance of the campaign in relation to policy decision-making. Evidence will then be presented about risk in relation to various routes of transmission regarding agents of prion disease in order to assess how likely it is that BSE has been transmitted to humans through eating meat or meat products. Later in the chapter, secrecy and the licensing process of medicines will be discussed, as the British government is about to again review the safety of medicines and to open up to public scrutiny the licensing process. Understanding how the Medicines Commission is structured and how the arrangement prevents dissemination of information to a wider audience because of commercial confidentiality is important in order to be able to argue that such an arrangement needs changing. There is nothing new about
secrecy surrounding medicines and vaccine. Through a comparison of the current situation in England and in Australia, and the situation that existed at the turn of the century it will be possible to highlight recurring patterns throughout the twentieth century. The chapter moves, then, from the main issue – vaccination policy – to one of the main factors affecting policy decisions, to the state of knowledge at relevant times, to some of the reasons why information is kept from the public.

What does all this have to do with kuru? It has been argued in the current work that loping ill vaccine and pituitary hormone are known iatrogenic routes of transmission for prion diseases. Kuru, I have argued, is the odd one out in this respect, when it comes to the question of transmission. The reason why the current chapter will be useful to assess kuru is because the evidence I present raises questions about how BSE has been discussed until recently in the public domain in news reports and television programs. As previously noted, the impression one gets from these reports is that the oral route of transmission is the main issue. While the oral route might be important in relation to BSE, other routes of transmission are more effective, yet there seems to be a less than frank discussion in the public domain about the possibility that vaccines and/or medicines might be an effective means of transmitting the disease. Likewise, in the case of kuru. Hence the similarity between the BSE and the kuru situation.

To contextualise kuru within this debate from the outset, I begin by bringing into the foreground another similarity: the administrative arrangements related to BSE and kuru research. It was noted earlier that kuru research was administered under the label of Child Growth and Development, and that in 1960 Gajdusek was a representative of the Department of Preventive Medicine at the National Institute of Health (Libiková 1962). In 1988, following the meeting held at the National Institute of Biological Standards and Control (NIBSC) on May 16th, the matter of BSE was given to Mr Roy Cunningham who headed the administrative arm of the Children, Maternity and Prevention Division of the Department of Health (CMP). Cunningham’s division dealt with immunization and vaccination policy³, and was

³ In Transcript Day 85. Newton & Moore
first alerted to BSE and its potential implications in March 1988 when a minute was sent about a new disease in cattle.

When asked at the Inquiry why his division had been informed about a cattle disease, Cunningham stated that he thought this was because his department was already handling CJD. The inquiry had difficulty seeing why this particular department was handling CJD when there did not seem to be an apparent connection between CJD and child or maternal health. Cunningham suggested that as well as being administratively responsible for the vaccination and immunisation policy, ‘also tagged on to that’ was anything to do with ‘slow viruses of which CJD is one’. He agreed with a remark made at the BSE Inquiry that the department was a sort of ‘catch-all’. Another plausible explanation for CJD being placed within the CMP which was not stated, is that in 1987 Cunningham’s department was not only responsible for maternity, child health and vaccination but also fertilisation, embryology, family planning and abortion. The distinction between sporadic CJD, which generally affects older people, and iatrogenic CJD is important here, because while the sporadic form of the disease does not affect child or maternal health, iatrogenic CJD is related to both women and children. Pituitary hormone was used to promote fertility in the former and growth in the latter group. Given that iatrogenic complications related to pituitary hormone were being dealt with in the Child, Maternal, Prevention Division, it seems that similar iatrogenic complication was also being considered relative to BSE. This is consistent with the memo sent by Dr Jefferys to Dr Pickles, which indicated that the matter seemed to be about adverse drug reactions, as mentioned in chapter six. Having drawn a parallel between the administrative arrangements in relation to kuru and BSE, I turn to examine vaccination policy.

**Vaccination policy in England**

There are risks and benefits with any vaccination policy. The perceived risk in transmitting BSE in England in 1988/1989 was seen as being far outweighed by the benefits. This perception was summarised in the witness statement of Professor

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4 Day 50 July 29th 1998. Lister, Cunningham, Bridges.
5 Witness Statement No 122 pt. 4 Cunningham
William Asscher, Chairperson of the Committee on the Safety of Medicines (CSM), who stated that the ‘risk-benefit analysis of exiting stocks of vaccines was comparatively easy’ for two reasons. One was that ‘... the risk posed by BSE to human health was ... always regarded as remote’, while the second reason was that vaccines were considered to be ‘very important to the protection of human health’. The CSM judged that the risks associated with interruption of the UK vaccination programme were far greater than the potential risk of BSE being transmitted.

Based on this risk assessment, rather than ordering ‘a withdrawal of stocks of efficacious products’, the CSM policy advised negotiation ‘with the manufacturers of such products so as to ensure they changed the sourcing of their bovine material as quickly as it was possible for them to do so’. Asscher stated that the idea that the risk was remote ‘was certainly the view of the Southwood Working Party in its report’. The policy then was aimed at not interrupting the vaccination program because the risk was seen as remote and vaccine use was highly prized.

At the beginning of 1989 as the Southwood Report was about to be released, the CSM and Veterinary Products Committee issued a joint statement to drug and vaccine manufacturers, which read:

“The CSM agrees with the Southwood Working Party that the risk to man of infection via medicinal products is remote. As a precautionary measure, and for the sole aim of seeking to guard against what is no more than a theoretical risk to man, the CSM and the Veterinary Products Committee (VPC) have agreed joint guidelines on good manufacturing practice for the manufacture of human and animal medicines who use bovine, or other animal, materials either as an ingredient or in the production process”.

This statement and the implied idea that the measures were only precautionary can be compared with the following comment, written in December 1988:

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6 WS No 441 pt 32 Asscher. Professor Asscher Chaired the Committee on the Safety of Medicines from 1987 to 1993 (pt 1.3).
7 ibid.
8 The Southwood Report was released in March 1989 following the work of the first expert group to consider BSE.
9 WS 441 pt.57 Asscher.
As I wrote in my earlier letter my colleagues are most anxious to ensure that existing products were identified and that manufacturers ensured that they conformed to the safety recommendations\textsuperscript{10}.

The term ‘most anxious’ in Asscher’s letter, which was written to Professor Southwood a few months prior to the joint CSM/VPC statement being issued, tends to suggest that using the precautionary principle might have understated the risk perceived by the CSM.

Contrary to media reports on the subject which suggest that eating meat and meat products is the main issue, the BSE Inquiry spent a considerable amount of time discussing the issue of medicines and their safety, and the reasons for the emergence of nvCJD is still an open question. The following interchange is useful as a gauge to the tenor of the discussion. Mr Kenneth Clarke, the Secretary of State post 1988 posed the following question,

You do not think we have any reason to think there is any risk from medicinal products now?

The then Sir Nicholas Phillips, Chair of the committee, interjected by saying,

the answer to that question is we do not know how the very small number of cases that to date have been diagnosed as having new variant of CJD have acquired the infection\textsuperscript{11}.

\textbf{The MMR Campaign}

It was established in the last chapter that vaccination rates were relatively poor in England during the late 1980s. Consequently, efforts were taken to increase the uptake rate of vaccines. The campaign against Measles, Mumps and Rubella (MMR) launched in October 1988 occurred at the same time as the joint guidelines were being revised by the CSM/VPC and the audit of pharmaceutical products was being

\textsuperscript{10} In Transcript Day 79. Acheson & France
\textsuperscript{11} Day 87. Clarke & Freeman.
conducted within Medicines Division. Specifically in relation to MMR, Cunningham, whose Division was responsible for the administration of the program, stated that ‘a lot of activity went into introducing this new vaccine’ and the division was involved ‘very much in encouraging the uptake of these vaccines’. Lord Newton, the Minister for Health at the time of the launch also pointed out that increasing the vaccination rate was a strategy outlined in the White Paper his department released during his time as Health Minister with Mrs Edwina Currie as Junior Minister. The question here is whether these forces, which were moving in the direction of increasing the rate of vaccination, played too greater part in the policy and adversely affected public safety.

During her evidence, Edwina Currie provided a hypothetical situation involving a planned vaccination campaign. We need to look carefully at the evidence because it might be the case that the situation was not as hypothetical as suggested given the line of questioning that the inquiry took with a focus on medicines and their safety. The hypothetical situation is as follows:

If, for example, we had planned a vaccination campaign, and we found at the last moment there was a problem with one of three suppliers, and we had to switch suppliers, and there might be a slight delay in delivering the vaccine to the north east of England, then Ministers would obviously have to be informed and would then be expected to deal with inquiries.

Because Currie was so specific in identifying a particular part of England, this tends to suggest that there might in fact have been a problem in providing MMR vaccine to this part of the country. A little later in the proceedings Phillips highlighted the other ‘counter-balancing cause for concern’; ‘the very danger people might stop asking for vaccinations and so on, before it was possible to re-source these’.

Relating to the issue of what was known and what to inform the public, Phillips posed the following hypothetical question to Clarke:

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12 Day 50. Lister, Cunningham, Bridges.
14 Day 84. Currie.
15 *ibid*.
16 *ibid*. 

Let us assume that the Committee had reached the conclusion that there was a significant although fairly remote risk that people might get infected ... from medicinal products sourced from a BSE infected cow. Having considered it very carefully, they reached the conclusion that the potential consequence of people abandoning vaccination because of the remote risk would be very much more serious than the risk itself. In those circumstances there might be two choices. One would be to say to the public: “There is a possibility, we do not think it should result in your refusing vaccines”, or they should say to the public: “Nothing to worry about”.

As Phillips pointed out, option one might give rise to a scare and to some people at least ‘adopting the course which had been decided to be undesirable’. In light of this, he went on to ask which approach should be adopted. Clarke remarked that one would need to be guided by the option that most matches the advice one is being given but admitted that the first approach would be ‘much more difficult’. He said, that in his experience the CSM faced this problem all the time in other less sensitive areas. There were always side effects to consider ‘in a tiny majority [sic] of patients who use it’, and the question is, ‘is the risk to this tiny group of people outweighed by the benefit to this group of people?’ The Secretary of State vouched for the cautionary approach taken by the Committee on the Safety of Medicines and his recollection and impression was that the risk of infection from vaccination was remote. He was not aware then, nor is he now in light of current knowledge, that ‘we are facing a situation where there actually was a bit of a risk but the benefits of the vaccine outweighed it’. That was why, as Phillips noted, he posed the question as a ‘theoretical situation’. He did not think the question had been answered nonetheless, ‘otherwise than saying it was a very difficult one’. The question, as Clarke suggested, is always:

Does the benefit to that substantial block of people justify the licensing of a medicine which will, we know, have harmful effects, possibly death on this tiny number of people.

Clarke did not believe that the situation under discussion fell into this category, at which point Mrs Bridgeman, a member of the Inquiry committee, interjected. Bridgeman reminded the Secretary of State that he mentioned that the decision would be based on the advice he received, but that he did not have any advice in this ‘particular dilemma on vaccines’. Clarke reiterated that the ‘advice we had was the

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17 Day 87. Clarke and Freeman
18 ibid.
19 ibid.
risk was remote’ and he ‘was unaware that such a dilemma existed in this particular case’. That the risk is remote from using vaccine or medicines produced with BSE infected cattle, is a mantra repeated throughout the inquiry hearings.

What do we know about transmission of prion diseases?

There are two areas that need to be considered: veterinary health and human health. Edwina Currie, the Junior Health Minister in 1988, noted that there was a lot of ‘guess work going on’ at the time of the MMR launch in relation to possible routes of transmission and the risk associated with them. But, she said,

we already knew from kuru, which was mentioned at the time, that it was possible by eating infected animal brains to acquire the disease or a version of the disease; we knew that.\textsuperscript{20}

It is difficult to determine whether this is a case of typographical error, and ‘animal’ should read ‘human’ brain, or whether Currie lacked very basic knowledge of the kuru literature. The significant point is that the oral route of transmission is the focus of the statement. This focus was quickly changed at the BSE Inquiry. Immediately following Currie’s statement her attention is drawn to paragraph 5.3.2 of the Southwood Report, which says that:

"Information from several spongiform encephalopathies suggests that parenteral inoculation is much more efficient in transmitting disease than oral or topical exposure ..."\textsuperscript{21}

Attention was also drawn to the fact that the Southwood Report indicated that the risk of acquiring BSE related to transmission routes from cattle to humans could be ordered: “The greatest risk in theory, would be from parenteral inoculation injection of material derived from bovine brain or lymphoid tissue”\textsuperscript{22}. This serves to show the distinction that was made between ingestion and injection.

\textsuperscript{20} Day 84. Currie.
\textsuperscript{21} \textit{Ibid}. Parenteral means through injection.
\textsuperscript{22} \textit{Ibid}.
While the distinction between inject and ingest might seem like a semantic triviality, the difference if significant in terms of what is known about how effective different routes of transmission are for prion agents. Understanding the differences will help to assess how likely is it that nvCJD has been acquired through eating meat, and how likely is it that kuru has been acquired through eating meat, be it animal or human.

Another important distinction that needs to be made is whether the risk was perceived to be remote as opposed to moderately high. Distinguishing between these terms will help to clarify whether the statements made in the vaccination policy and the Southwood report were consistent with what was known. If the risk associated with injecting potentially contaminated bovine material into humans was moderately high rather than remote, this meant that the pharmaceutical product manufacturers and the government that licensed the products for use had an enormous dilemma on their hands as stocks existed that were already potentially contaminated. Judging by the inquiry transcripts it seems possible that this was, in fact, the situation. The question was therefore whether the products should have been withdrawn.

Compare the idea that the risk was remote with the following comment made in July 1989 by Southwood. By this time, Dr D. A. J Tyrrell chaired a committee set up to look at research needs once the Southwood group had released its report, and the Tyrrell committee was writing an interim report23. Southwood wrote that he believed Tyrrell was

... absolutely right to point out gently how we are forced to argue from analogy with scrapie and one awaits, with some anxiety, the experimental confirmation of that assumption....

Personally, I would have thought the possibility of human infection was moderately high if some medicinal products were made from tissues of infected animals and injected into humans24.

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24 Day 87. Freeman & Clarke. NB. While this letter is spoken about as dated as July 5th 1989, the numbering code given to the letter by the Inquiry is 5.05, which means May 5th. It seems that this is a typographical error.
Southwood acknowledged that this was an extreme case, but his committee

... certainly had such anxieties very much in mind, and as you may know the whole series of recommendations, including the compulsory slaughter of animals.

It was suggested that if Tyrrell was thinking of making a few drafting changes, where at paragraph 2.2 on page 4 Tyrrell had written “[t]he Southwood Group was correct in their belief that this disease would not have implications for human health save through food”, Southwood said that ‘a more complete picture of our belief will be given if you added “provided various safeguards that were recommended were instituted”’.

The problem for the public is that the safeguards had not been implemented. One of the safeguards involved sourcing from other places. I mentioned in the last chapter that Australia and New Zealand were the preferred options. What is puzzling is that in 2000 it was reported that ‘Australian vaccines can be traced to British cattle, more than a decade after the mad cow disease threat was revealed’. Professor Richard Smallwood, Australia’s Chief Medical Officer, is reported to have said that Smith-Kline Beecham, the manufacturer of an oral polio vaccine in Australia, ‘discovered only last week that British calf serum originally sourced from British cattle was used in making the Australian product’. A spokeswoman for Smith Kline Beecham declined to comment on how the company had missed the British link for so long. A ‘fresh audit of the source of all vaccine material used in Australia’ was to commence. These events followed the withdrawal of the polio vaccine produced at Medeva in England in October 2000. Why Australia has been sourcing from England, when England throughout the 1990s is said to have sourced its bovine material from Australia is a mystery.

There is, however, nothing mysterious about Australia’s vaccination policy, which seems to be identical to the one in England. Quoting Metherell, Professor Smallwood said that

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25 Metherell 2000:4. One newspaper (Mann 2000:11) reported that the Australian product was made by Smith Kline Beecham and Commonwealth Serum Laboratories.
the benefit of immunising children outweighed the remote theoretical risk of vCJD, the human equivalent of mad cow disease. An expert panel was auditing the source of material used in the production of other vaccines, although it seemed unlikely that these would be involved.

According to this newspaper report, the Therapeutic Goods Administration in Australia is reported to have said that ‘the chances of other vaccines being affected were one in ten trillion’. A figure of one in a billion was given as the chance of acquiring nvCJD from polio vaccine. Asked whether there were concerns the vaccine risk factor might be greater, given emerging evidence of vCJD prevalence in Britain, Professor Smallwood is reported to have said

Australian experts’ assessment concurred with that of the Federal Drug Administration, which had cleared continued use of vaccines “grown” in the British Calf serum27.

Metherell’s article is brilliant at capturing the arguments I am attempting to outline, for while the words used in the article might not be a verbatim account of Professor Smallwood’s views, they no doubt convey what was said, as the argument is so similar to the one used in Britain. It is an argument used, I suggest, based on the belief that it is important to allay public anxiety in order to maintain the immunisation program.

We need to consider the message that is conveyed to the public when terms such as “remote” and “theoretical” are used. And the effect it might have on the public when figures such as one in ten trillion and one in a billion are given. On what evidence are these figures based and what do the terms mean?

The Collins English Dictionary defines “remote” as being synonymous with doubtful, implausible, small, slight or unlikely (Collins 1994:974). “Slight” means small in quantity, of small importance, insignificant, negligible, trifling, trivial and unimportant (Collins 1994:1086). A remote risk then could be interpreted as one that is unlikely but possible, and if this were to prove to be the case a small number of people would be affected. Collins defines theoretical as ‘lacking practical application

or actual existence; hypothetical’. Synonyms for the word are, conjectural, speculative, ideal, pure (1994:1202). Yet theory does not belong in some other world, completely removed from the real. A theory should have little credibility unless it is linked somehow to evidence. Even a speculation, which I would distinguish from theory, is usually based on some sort of observation. The problem with determining the risk in the case of prion diseases in a new species, as is the case regarding BSE and nvCJD is that calculations need to be made on assumptions. If the assumptions are incorrect, the figures can be affected greatly. In the case of the risk of transmitting prion agents through medical products and procedures there are well documented precedents in specialist circles which would warrant an extremely cautious approach as regards taking measures to prevent these diseases. When messages about this issue are provided to the public, the significance of the problem seems to be trivialised when figures like one in a billion and one in a trillion are used. Such figures offered up within the context of the problem being only theoretical might leave the impression in the minds of the populace that no one knows the potential risk and the chances of there being a problem is not worth worrying about. While I agree that at this stage it is not possible to know the actual risk, it is doubtful that any one with knowledge in this area would agree that the risk is not worth worrying about. But this is at odds with the messages of assurance given to the public. As of November 2nd 2000, despite knowing that the Australian product has used British bovine material, the CMO is reported to have said that the Australian product would not be withdrawn28. I have heard no announcement that this has changed.

In England 11 million doses of the Medeva vaccine have been administered29. One report puts the figure at ‘an estimated 35m doses, accounting for a third of all oral polio vaccine administered30. The two different accounts in terms of the figures might reflect an attempt to allay public anxiety in Australia as the higher figure is reported in The Guardian Weekly. Regardless of the actual figure, it would be wise to ask if the company that produced the third in this case is the same company that produced the third in Currie’s hypothetical example in relation to MMR vaccine? This question needs further research.

29 Mann 2000:11
30 Meikle & Watt 2000:8
When Southwood wrote to Tyrrell in 1989 in England about the perceived moderately high risk associated with injecting bovine material infected with BSE, and mentioned the safeguards that needed to be put in place, another one of the safeguards relates to the detection of sub-clinical animals. That is, animals not showing signs of BSE but who might have been incubating the disease nonetheless. The problem is that neither then, nor now, is it possible to test animals as no definitive test exists\(^{31}\). Southwood’s letter suggests that it would not be possible to say that ‘this disease would not have implications for human health save through food’ without transmission studies being conducted and no detrimental effects observed. It appears from his letter that finances influenced the studies that were undertaken, for he hoped that the Minister and others would, ‘notwithstanding the ridiculous attitude towards public expenditure, find the necessary funds to undertake the high priority research’. The man was ‘horrified to discover a little while ago that the former control study of possible vertical transmission had not yet been put in place’. It would be wrong to conclude from the letter however that no transmission studies were undertaken on BSE.

In 1990 a report was sent to the Chief Medical Officer, which outlined the state of knowledge about transmission routes. The document, called ‘Opinion on the public health implications of eating beef and the epidemic of BSE’, dated July 24\(^{th}\) 1990, at paragraph five states that,

> “Oral transmission of some spongiform encephalopathies undoubtedly occurs – although very large doses are needed because the oral route is very much less efficient than, say, intracerebral inoculation” \(^{32}\).

Section five of an annexure pertaining to the above document reads

> “The oral route is clearly capable of transmitting spongiform encephalopathies in the diseases of BSE and transmissible mink encephalopathy, and it is assumed to be an important natural route of transmission in scrapie and kuru”.

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\(^{31}\) In August (18\(^{th}\)) and September (1\(^{st}\)) 2001 there have been reports about a diagnostic test for BSE following the discovery that ‘the prion isoform [found] in urine may lend itself nicely to the development of a non-invasive diagnostic test for prion disease’ (New Scientist September 1, 2001 p. 55).

\(^{32}\) This document, which originated from the “MRC Common Cold Unit”, was addressed to the Chief Medical Officer Sir Donald Acheson (December 16\(^{th}\) 1990 See Transcript of Professor Will December 16\(^{th}\) 1999).
The document then went on to say that

"Experimental studies, however, show ingestion to be very inefficient, at least five orders of magnitude less efficient than intracerebral injection. In the transmission work done to date with BSE, the incubation period in mice was longer after large oral doses of BSE-infected cattle brain than after much smaller parenteral injection. In these as in other animal experiments, large doses, far in excess of what would be experienced in nature, appear to be needed for successful disease transmission”.

The conclusion drawn was that

"... the occasional low doses of BSE agent in human food are well below those capable of infecting humans, even if humans were specifically susceptible to the agent”.

This serves to highlight the point that oral transmission through meat, whether in the case of BSE or kuru, is the least effective means of transmitting prion diseases. It could be argued that oral polio vaccine is therefore of low risk when produced using BSE infected material. This ignores the fact though that in England, even it was considered to be of sufficient risk to withdraw it from sale. It is important to take into account the different means by which vaccines are produced in comparison with meat.

The issue of risk pertaining to route of administration was a subject of debate right throughout the late 1980s and 1990s both in scientific and government circles. On many occasions kuru is used as an example. Clarke, Secretary of State from 1989 stated that he remembered discussions taking place about CJD (meaning new variant CJD) and whether it had been acquired through ingestion after the first case of nvCJD became evident. His comments bear repeating. Like Currie, he also referred to kuru in a way inconsistent with the dominant literature, while speaking about nvCJD having been acquired by vegetarians. He stated that ingestion was not ruled out because:

... there was this question of kuru which I seem to remember is a disease that seemed to exist among the head hunters of Papua New Guinea, that they started to ingest the brains of their slaughtered animals. It did seem there was a possibility they had created a related disease by doing that."33

33 Day 87, Freeman and Clarke.
Putting aside for the moment the stereotypical depiction, while it is curious that two government officials spoke of animal brain as opposed to human brain, this should not detract from the knowledge that oral transmission was believed to be the least effective route. Southwood’s letter to Tyrrell gives the impression that the recommendation to remove contaminated meat from circulation was an action taken as much for the reason of preventing its use in medical products as it was to remove the meat from the food chain, although this was also important.

The irony is that the continued referral in the public domain to the idea that the risk was remote from eating beef seems to have been right based on the experience with scrapie. For scrapie has not been acquired by humans through eating meat during the past nearly two hundred years. But in the case of medicines, particularly those injected into the body made with brain, spleen or placenta, the risk was assessed as much greater based on previous experience with louping ill vaccine and pituitary hormone. Information about these risks associated with products administered through routes other than the oral route is rarely spoken about in the public domain whether in relation to BSE, nvCJD or kuru. The only case where the injectable route has been acknowledged relates to the hormone responsible for iatrogenic CJD. In the case of kuru, I argue that the discourse is confounded because of images of cannibalism, head-hunters, madness or dementia, which so often accompany any reportage of this disease, even its seems at inquiries into the disease and in government circles. The distorted images have been carried through into later reportage of BSE, which place an undue emphasis on mad cows as opposed to how the role of medicines or vaccines might have played a part in the emergence of BSE in the first place.

**Veterinary medicines during the 1980s**

There has been recognition of the need to avoid using ovine and bovine material in pharmaceutical products in veterinary medicine since 1983. According to Sir James Armour, Chair of the Veterinary Products Committee (VPC), the government issued guidelines at this time\(^\text{34}\). The VPC ‘were aware that scrapie had been disseminated *via* 

\(^{34}\) MAL 67 – MAFF guide cited in WS No 477 pt. 8b. Armour.
a loping ill vaccine ... in which brain and spleen tissue from scrapie-infected sheep had been inadvertently incorporated\(^{35}\). As well as knowing about loping ill vaccine, pituitary hormone was also known to be problematic in veterinary medicine, perhaps not surprisingly considering that during the same period the hormone was shown to produce CJD in humans.

In 1987 the Head of the Medicines Unit at the Central Veterinary Laboratories wrote a cautionary note for the journal *Veterinary Record* about the potential to transmit scrapie through pituitary hormone, in which he stated,

... the use of brain tissue of bovine or ovine origin, particularly the pituitary-derived follicle stimulating hormones, could, by analogy from previous work on scrapie, possibly cause contamination of the BSE agent and recommended that manufacturers should source these hormones from species other than cattle or sheep\(^{36}\).

This statement indicates two things. Pituitary hormone is associated not only with human prion disease in the form of iatrogenic CJD but is also associated with the transmission of scrapie. And two years after the problems associated with pituitary hormone in the human situation had been realised, it would appear that pituitary hormones made from potentially infected cows or sheep were still being used in veterinary medicine.

Dr James Rutter, Director of Veterinary Medicines and Chief Executive of the Veterinary Medicines Directorate, explains how in 1988,

... products in the hormone group were called up for review which required the licence holders to submit updated dossiers to the licensing authority. From this review it transpired that four products in the pituitary hormone group that may have contained bovine material were on the market. These products were not defended and the four licences had expired by April 1991\(^{37}\).

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\(^{35}\) Sir James Armour, now retired, held the position of Chairman of the Veterinary Products Committee (VPC) from 1987 to March 1996 and since 1985 had been a member of the VPC. Simultaneously, during the period 1986-1994 Armour was 'a member of the Scientific Advisory group to Merck Research Laboratories', and prior to this between 1977-1985 he also acted as consultant to Merck Sharp & Dohme Animal Health. All these 'interests were declared'. From 1953-1960 Armour worked as a 'Field Veterinary Officer and then Veterinary Research Officer, Colonial Service, Nigeria'. Later, from 1960-1963 he worked at the Cooper Technical Bureau (latterly Wellcome), in Berkhamstead, Hertfordshire (Witness Statement No 477, Armour).

\(^{36}\) WS 477 Armour. In reference to *Veterinary Record* 1987 Vol 123.

This statement indicates that products in this group were being used until 1991 in veterinary medicine, which should make us take note of the possibility that BSE might be attributable, at least in part, to the use of products in the pituitary hormone group. In April/May 1988, the VPC received a ‘request from the Medicines Unit, CVL [Central Veterinary Laboratories] to comment on draft guidelines for the review of veterinary products containing hormones which were being prepared for issue’. Members of the VPC were concerned about the ‘possible risk of BSE contamination emanating from the use of serum and glandular extracts from cattle or sheep where these species were the source of the hormones’. As a consequence of the concern a paragraph about sourcing outside of Britain was inserted into the guidelines for manufacturers in September and October 1988 and the revised guidelines were issued to pharmaceutical companies at the end of November\(^{38}\). Hence, the relevance of the joint CSM/VPC statement outlined earlier. The use of the precautionary principle in light of this knowledge does not seem to have been appropriate in terms of protecting the health and safety of both humans and animals, for the VPC were aware of pituitary hormone, louping ill vaccine and the associated iatrogenic transmission of prion diseases. This committee is charged with responsibility for the safety and efficacy of veterinary medicines as well as for products with implications for the safety of humans and the environment.

In December 1990, the Veterinary Medicines Division circulated a final report of the results of a questionnaire sent to its members, which indicated that ‘companies had been taking a range of measures to comply with the guidelines but that advice was required on a few issues’\(^{39}\). The Veterinary Products Committee made four points:

Use of any bovine brain material during manufacture must not take place

In relation to the SKP bulk viral vaccines made with filtered donor calf serum from UK sources, further information was required on the herd history of the source of the donor calf serum

Dr Taylor at MAFF should be consulted on whether the autoclave treatment of bovine soup stock of UK origin prepared from bones at EC-approved abattoirs was satisfactory prior to its use in culture media’, and

\(^{38}\) WS 477. Armour.

\(^{39}\) ibid.
... action taken in relation to biological products should conform to EC regulations on BSE\(^4\).

This demonstrates the uncertainty as regards the safety of some veterinary vaccines at the end of 1990. Add to this the concerns expressed about pituitary hormone a few years earlier and there is certainly a reason to consider the role that these products played in the production of prion disease.

The Chair of the Committee on the Safety of Medicines suggested that pharmaceutical manufacturers acted quickly to source bovine and ovine material used in their production processes from non-British stock. From the perspective of some individuals this might be so. But from the perspective of the populace it might be that the changeover was not carried out quickly enough. I doubt that the public would be impressed to discover that Directive 81/851/EEC, which came into force in November 1983, made provisions for the guidelines ‘to be applied progressively, within 10 years, to veterinary pharmaceutical products already on the market in member states’\(^4\). In 1991 it was reported that all manufacturers could ensure sourcing from non-British bovine material, and in 1992 the government declared that all medicinal products were safe. While it is commendable that the changeover occurred within the time of the provision, it is not so surprising that the announcement that vaccines were now safe was made in 1992. A question to ponder is, is it the case that BSE, at least in part, has been produced by veterinary vaccines or hormones and then material from these animals has been used in human medicines and vaccines and produced at least some of the cases of iatrogenic nvCJD?

Given the iatrogenic pathways known to produce prion diseases, it would be unwise to ignore the state of knowledge relating to both veterinary and human medicines. The fact that the Division of Children, Maternal and Prevention was given BSE to administer and that this department also administered vaccination policy would be consistent with this line of reasoning. As to the degree of influence the MMR campaign had on vaccination policy, this is difficult to gauge but given that CMP was

\(^{4}\) ibid.
\(^{4}\) WS No 499 pt. 20 Rutter.
working hard to improve the uptake of vaccines, as was the Minister for Health, it
does seem that not interrupting the campaign was the overriding concern. It is
possible that the Committee on Safety of Medicines did not know about the degree of
risk perceived by some individuals. This is unlikely however given that the CSM and
the VPC worked together to issue joint guidelines. Given this, the decision not to
withdraw existing stocks of potentially contaminated products seems inappropriate to
say the least, for there were other products discussed at the Inquiry made prior to 1992
besides MMR and polio vaccine that require evaluation.  

Distribution patterns have been traced since the release of the Phillips Report in
October 2000 and the polio vaccine being withdrawn. On March 3rd 2001, it was
reported in a radio report that four and a half thousand children in Ireland had been
exposed to polio vaccine with expired use by dates. The message given to the public
was that this would not cause a problem apart from the fact that the vaccines might
have been ineffective. I am not so sure. While there is to date no conclusive proof that
vaccinations have caused nvCJD, a policy of ‘do not interrupt the vaccination
program at all costs’ might well be in need of reassessment.

The position taken in Australia in relation to vaccination policy mimics the situation
in England and it appears that a culture similarly lacking in transparency operates,
judging by a television report in February 2001. Rebecca Smith from the Australian
Consumers Association is disappointed by the NHMRC expert Committee’s lack of
transparency and the fact that the meetings were held behind closed doors. The
public was informed on the program that most people in the UK have acquired vCJD
from eating meat and we do not know if there are other ways of transmitting the
disease. It is understandable that health departments do not want to alarm the public.
But in light of the information presented, it seems preferable that, in future, the public
is informed about what is known and blanket statements such as, we do not know
about other ways of transmitting BSE or similar diseases are avoided.

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42 See Appendix 7c.
43 ABC News Radio Report March 9th 2001. Rachael English speaking to Dr Cormack McNamara in
Dublin.
44 Insight (2001). SBS TV Inga Johansen reporting on BSE in Britain, February 22nd.
An attempt was made to distinguish between medicines in toto as opposed to a particular batch, at the BSE Inquiry. Freeman began by saying, while it is one thing, to remove that batch or that product from the market where you might have an easily, relatively easy means of sourcing the supply from elsewhere. Of course it is a much more difficult problem if it were the case that there was a substantial or significant risk in relation to vaccine or pharmaceutical products generally which may be derived from bovine products.\(^{45}\)

Here is the crux of the matter. If it is the case that it was not a question of a particular batch being contaminated but rather a problem of far greater import, it is not difficult to see why such information has not been widely disseminated.

**Medicines and secrecy**

It is apparent from Meikle’s comment quoted at the end of the last chapter, that by as late as February 2001, the licensing process for medicines was an area in need of review.\(^{46}\) Questions were raised at the BSE Inquiry about lines of accountability and secrecy surrounding the Medicines Commission. Here a brief outline will be useful of the structure and authority of the CSM and its sub-committees, which were known as Section 4 committees. These committees derive their authority from Section 4 of the Medicines Act 1968. As Currie’s statement attests, the CSM licenses products\(^{47}\) and the Medicines Act ‘actually gives power to these authorities to take decisions’.\(^{48}\) Appendix 7a provides a flow chart of information with respect to the licensing process. The important thing to note is that the Chief Medical Officer is not privy to the flow of information, and those working in Medicines Division (later the MCA), while accountable to the CMO were also accountable to the Medicines Commission (see Appendix 7b). Bridgeman confessed to being confused about the role of the Medicines Commission\(^{49}\) and described it in the following terms:

\(^{43}\) Day 87. Clarke & Freeman.
\(^{45}\) Meikle 2001.
\(^{46}\) Day 84. Currie.
\(^{47}\) *ibid.*
\(^{48}\) *ibid.*
\(^{49}\) *ibid.*
It is a curious sort of quango established under the Medicines Act and not quite the same sort of creature I have been accustomed to in other Departments, because it appeared to have certain decisions delegated to it which then had to be kept in secrecy from the Department, so it was rather peculiar, this business surrounding medicines\textsuperscript{50}.

Lord Moore, Secretary of State prior to the time that Clarke assumed this office, agreed: ‘You are right, there were some rather peculiar structures insofar as that was concerned’. What this meant in effect, was that officials in Medicines Division were prevented from disclosing commercially sensitive information to the CMO. Commercial confidentiality and built-in structural conflicts of interest, therefore, play an important role in preventing information flowing outside of the deliberations of the CSM and its subcommittees.

As to the question of the line of accountability, Bridgeman noted that the Chief Medical Officer had told the inquiry that ‘he did not feel he had a locus in relation to the Committee on Safety of Medicines’. Currie, when asked whom the CSM reported to, replied ‘I think each other would be the right answer’\textsuperscript{51}. She expressed the view, as did Clarke that the committee always acted with caution\textsuperscript{52}. In Currie’s case, her confidence came from the fact that if problems arose, Ministers were kept informed. Ministers were not always kept informed though judging by Currie’s testimony, as she went on to note that it would have been overwhelming, apart from being ‘administratively efficient’, had this been the case. Nevertheless, during her time in office (up to the beginning of 1989) the CSM was delegated to explore the issue and ‘ensure that the products were safe as a result of their actions and decision’. Then it would be up to Ministers to back any decision taken by the committee\textsuperscript{53}. It was not the task of a Junior Minister to ‘gain say their decision’\textsuperscript{54}. Notwithstanding the secrecy involved in these arrangements, it is ultimately the responsibility of the Secretary of State to ensure that any recommendations that come from the CSM are workable and supported by enough evidence to withstand legal challenge\textsuperscript{55}. As Moore

\textsuperscript{50} Day 85. Newton & Moore.
\textsuperscript{51} Day 84.
\textsuperscript{52} \textit{ibid.}
\textsuperscript{53} \textit{ibid.} Currie left the department just prior to the release of the Southwood Report in early 1989.
\textsuperscript{54} \textit{ibid.}
\textsuperscript{55} For a discussion on the dual lines of accountability in Medicines Division see Transcript of Day 79 (Acheson and France), and on the legal implications of sanctioning recommendations such as the Specified Offals Ban, transcript Day 87 (Freeman and Clarke), is useful.
stated, ‘... whatever you call the work of the Medicines Control Agency [to whom the CSM report], ultimately, the Secretary of State is responsible to Parliament for them’.56

Lord Freeman, the Minister for Health in 1988, pointed out to the Inquiry that ‘before Southwood finally reported, action had already been taken to destroy tissues of infected animals’.57 He made the point to demonstrate that he ‘quite understood that some medical products might have been made before the destruction of affected cattle came into operation, which is August 1988’. Clarke, Secretary of State in 1989, fielded a hypothetical question at the inquiry about what he would have done if faced with ‘a whole series of pharmaceutical products on the shelf which are derived from a bovine product’ which might be infected and carry a moderately high risk. Clarke suspected that

... the Secretary of State would probably wind up ordering withdrawal of all batches that might be affected if “moderately high” meant what you – I mean I agree it could well mean that a significant number of patients may be infected by this in that case one would probably withdraw unless the advice was some catastrophic consequences for some patients would ensue or with a large number of patients if you did that’.58

Were there not grave concerns about the safety of medicines and vaccines, it is difficult to see why the inquiry took the line of questioning that it did. The responses of some individuals in positions of influence at the time of the MMR campaign and in the years that followed, demonstrate one of the possible outcomes of vaccination policy that negotiates with manufacturers rather than withdrawing medicines known

56 Transcript Day 85. Newton & Moore. Events during the period 1986 to 1987 are covered in this statement. Lord Moore was the Minister of Health from 1986 to mid 1988 and then became the Secretary of State in June 1988. Moore had also been Secretary of State prior to 1986 when he had been involved in AIDs administration. He compared the open communication in the case of AIDs as opposed to the lack of openness involved in BSE. Lord Newton, as well as being the Minister for Health, was the parliamentary adviser to the Royal Pharmaceutical Society of Great Britain, which is the body concerned with the discipline of pharmacists, much like the General Medical Council. This body does not represent the pharmaceutical industry. Newton discusses the problem of making a decision about the vaccine dilemma at the time, but defers to higher authority like the Chief Medical Officer. As I have shown, the CMO was not privy to all of the information. Note: it is difficult from the transcripts to get an exact sense of who held the position of Secretary of State and Minister for Health during the period 1986-1989. It is apparent that Lord Moore, while he was appointed Secretary of State in June 1988 (see above) did not hold this position for long, as Mr Kenneth Clarke was the Secretary of State in 1989.
57 Day 87. Clarke and Freeman.
58 ibid.
to have incorporated bovine material infected with BSE. The situation described indicates that there might be a reluctance to acknowledge that any of the cases of \textit{nvCJD} that have occurred might be related to vaccines and medicines.

A culture insistent on not interrupting the vaccination program seems to be one in which unwanted effects would not be welcomed and might be minimised should they start to appear. This is not to imply that drug reactions are not documented in England at all, for as Dr Gerald Jones noted, the process of collecting information on Adverse Drug Reactions began in 1959 when there was a large reaction to a particular drug\textsuperscript{59}. Jones does not mention which drug, but this quite possibly relates to polio vaccine and/or loping ill vaccine which had been problematic in England throughout the nineteen fifties. The transmission of kuru should be considered within this context.

To summarise the situation and to conclude with a reflection on earlier times it will be possible to show that secrecy surrounding vaccine and medicines is not a new problem. In the 1988 situation, I have shown how information was prevented from reaching even the CMO let alone the public. In 1967, Professor Wilson remarked that information about vaccines is not widely disseminated (Wilson 1967). When the first international conference on vaccines was held in 1966, Horwitz expressed the hope that the lively and productive discussions would ‘be spread throughout the world’ (Horwitz in WHO 1967). My findings support the idea that such information is not being as widely disseminated as Horwitz might have hoped. Chapter four provided a possible reason for not openly discussing the hazards of immunization; the threat of losing one’s position seems to have acted as a powerful reinforcer that cautions researchers against speaking about worrying findings in research related to the manufacture of pharmaceutical products. This culture is characterised by not being as frank as one might hope with the public regarding the issue of iatrogenic reactions. Such a culture has existed for a long time.

In 1920, Chas M. Higgins, in an alarmingly entitled book, \textit{Horrors of Vaccination}

\textsuperscript{59} Transcript 13/12/9 G. Jones. Jones was called again to give evidence during the last week of the Inquiry’s sitting days, as were many individuals involved in ensuring the safety of medicines.
Exposed and Illustrated, argued that the real situation involving vaccination was either denied or concealed. In his opening statements, in which he made a public challenge to the Health Departments in the State of New York, Higgins wrote:

In order that there is no misunderstanding about the serious charge which I shall bring against vaccination, as being now actually more dangerous to public health than natural smallpox, and the equally serious charge which I make against vaccinating doctors – who now control our Departments of Health and Vital Statistics – of denying and concealing these facts from the people. ... If they [the Public Health departments] deny the truth of these charges I further solemnly challenge them to open their now concealed records to public examination and I will prove the truth of my charges from these records.... (Higgins 1920:vii).

Higgins’s main aim was to stop the compulsory implementation of vaccines to the armed forces in America and it was for this reason that he addressed his challenge to the then President, Woodrow Wilson, who as Commander-in-Chief of the Army and Navy was in a position of authority to alter the situation.

Higgins’s work documents many examples of vaccine reactions and deaths that were described to him by doctors and members of the public, along with accounts described in government bulletins, particularly from U.S Public Health Reports and the Department of Agriculture. There were many types of vaccination that concerned Higgins but his views on smallpox are the most relevant here, for in Higgins’s mind smallpox vaccine was associated with outbreaks of foot and mouth disease during the first decade of the twentieth century.

In support of his argument Higgins cites one U.S. Public Health report for September 2nd, 1910 (pp 95 and 99), which stated that Japanese vaccination was the source of the deadly epidemics of Cattle Plague, known as “Foot and Mouth Disease”. At this time, the problem for vaccine manufacturers in America was that they were using the Japanese seeding virus. The cattle plague led to the ‘slaughter of hundreds of thousands of animals, an unknown amount of human mortality, and a loss of millions of dollars to the Government and people’ of America (Higgins 1920:55-56). Given that outbreaks of foot and mouth disease have occurred in conjunction with BSE and

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60 This information is sourced to the supplement for the Year Book U.S. Department of Agriculture, 1914 page 21, and page 99
nvCJD at the beginning of the twenty first century, and that foot and mouth disease outbreaks in Europe were caused by vaccine against FMD during the early 1990s, as noted earlier in the thesis, it might serve us well to take note of Higgins’s text.

Australia has not had an outbreak of foot and mouth disease for one hundred and thirty years. As of March 1st 2001, Australia tightened its quarantine procedures to prevent the spread of this disease to Australian shores. While this might be the case, as shown in chapters two and three, foot and mouth disease has been discussed by scientists in relation to culture contamination since the nineteen thirties in both England and Australia. During the ‘thirties, foot and mouth, poliomyelitis virus and louping ill virus were all discussed simultaneous with the outbreaks of scrapie transmitted through louping ill vaccine. During the nineteen fifties, foot and mouth disease was discussed along with kuru at a working lunch, at which time poliomyelitis vaccine and louping ill vaccine were also problematic. In 1962 foot and mouth sera was being moved to the Animal Section of laboratories in Australia at a time when the hazards associated with working with human and animal studies in the same laboratory was being realised. Taking a broad perspective over the last century it is not difficult to see a pattern of similar problems emerging at different times.

Nearly a century after Higgins’s cautionary tale it might be wise to start disseminating information rather than relying on outdated vaccination policies and precautionary principles, which in some cases enable a lack of safety to be built into the system by minimising the dangers. As it would to review the lines of accountability relative to medicines and their safety and to open up the process to public scrutiny. Together these measures might provide a more transparent system where the need for inquiry after inquiry relating to prion disease would not exist.

During the early years of the twentieth century, any criticism that was made about vaccine was generally made by the public rather than by scientists. Wilson, in 1967, suggested that this situation had changed, as criticisms started to come from members of the scientific community as well as from the public (Wilson 1967). Such criticism is often based on empirical evidence of the effects a particular vaccine or medicine can produce; the risk is shouldered by all members of the community who receive medical products that have the capacity to transmit prion and other diseases.
This survey of what is known might help to move vaccination policy onto safer ground. It is not a plea to ban the use of vaccine but if vaccines, hormones and sera continue to be produced then it behoves us all to try and understand what can happen and to minimise the potential hazards, thereby reducing the consequent damage. On the basis of the evidence presented, I argue that there is a need for further archival research and for serious reconsideration of kuru and its causes before it is relegated to a footnote in medical history.
Chapter 7

The safety of medicines and the prevention of prion diseases

The last chapter began to show some of the tensions involved in this complex field of prion research and administration. The main aim of this final chapter is to discuss vaccination policy both in England and Australia with respect to what is known about the potential to transmit prion diseases through vaccine and medicines. Judging by the following comment made by Norman Baker, a British Member of Parliament, there is good reason to do so. Baker suggested that,

“...There has been a terrible averting of eyes on anything related to vaccines in the last 11 years. The whole reason seems to be that the vaccination programme must not be undermined...” (Baker in Meikle & Watt 2000: 8)\(^1\).

I argue that in its current form vaccination policy needs to move onto safer ground. In order to develop this argument the chapter examines policy in England and one of the factors that influenced policy decisions during the late nineteen eighties: a campaign for Measles, Mumps and Rubella vaccination that was implemented in England in October 1988\(^2\). At the same time as decisions were being made about the safety of medicines relative to BSE, there was a move to improve the rate of uptake of vaccines. Hence the significance of the campaign in relation to policy decision-making. Evidence will then be presented about risk in relation to various routes of transmission regarding agents of prion disease in order to assess how likely it is that BSE has been transmitted to humans through eating meat or meat products. Later in the chapter, secrecy and the licensing process of medicines will be discussed, as the British government is about to again review the safety of medicines and to open up to public scrutiny the licensing process. Understanding how the Medicines Commission is structured and how the arrangement prevents dissemination of information to a wider audience because of commercial confidentiality is important in order to be able to argue that such an arrangement needs changing. There is nothing new about

\(^1\) Norman Baker is the Liberal Democratic M.P. for Lewes, England.
\(^2\) BSE Inquiry transcript Day 84. Currie.
secrecy surrounding medicines and vaccine. Through a comparison of the current situation in England and in Australia, and the situation that existed at the turn of the century it will be possible to highlight recurring patterns throughout the twentieth century. The chapter moves, then, from the main issue – vaccination policy – to one of the main factors affecting policy decisions, to the state of knowledge at relevant times, to some of the reasons why information is kept from the public.

What does all this have to do with kuru? It has been argued in the current work that louping ill vaccine and pituitary hormone are known iatrogenic routes of transmission for prion diseases. Kuru, I have argued, is the odd one out in this respect, when it comes to the question of transmission. The reason why the current chapter will be useful to assess kuru is because the evidence I present raises questions about how BSE has been discussed until recently in the public domain in news reports and television programs. As previously noted, the impression one gets from these reports is that the oral route of transmission is the main issue. While the oral route might be important in relation to BSE, other routes of transmission are more effective, yet there seems to be a less than frank discussion in the public domain about the possibility that vaccines and/or medicines might be an effective means of transmitting the disease. Likewise, in the case of kuru. Hence the similarity between the BSE and the kuru situation.

To contextualise kuru within this debate from the outset, I begin by bringing into the foreground another similarity: the administrative arrangements related to BSE and kuru research. It was noted earlier that kuru research was administered under the label of Child Growth and Development, and that in 1960 Gajdusek was a representative of the Department of Preventive Medicine at the National Institute of Health (Libiková 1962). In 1988, following the meeting held at the National Institute of Biological Standards and Control (NIBSC) on May 16th, the matter of BSE was given to Mr Roy Cunningham who headed the administrative arm of the Children, Maternity and Prevention Division of the Department of Health (CMP). Cunningham’s division dealt with immunization and vaccination policy³, and was

³ In Transcript Day 85. Newton & Moore
first alerted to BSE and its potential implications in March 1988 when a minute was sent about a new disease in cattle.

When asked at the Inquiry why his division had been informed about a cattle disease, Cunningham stated that he thought this was because his department was already handling CJD. The inquiry had difficulty seeing why this particular department was handling CJD when there did not seem to be an apparent connection between CJD and child or maternal health. Cunningham suggested that as well as being administratively responsible for the vaccination and immunisation policy, 'also tagged on to that' was anything to do with 'slow viruses of which CJD is one'\(^4\). He agreed with a remark made at the BSE Inquiry that the department was a sort of 'catch-all'. Another plausible explanation for CJD being placed within the CMP which was not stated, is that in 1987 Cunningham's department was not only responsible for maternity, child health and vaccination but also fertilisation, embryology, family planning and abortion\(^5\). The distinction between sporadic CJD, which generally affects older people, and iatrogenic CJD is important here, because while the sporadic form of the disease does not affect child or maternal health, iatrogenic CJD is related to both women and children. Pituitary hormone was used to promote fertility in the former and growth in the latter group. Given that iatrogenic complications related to pituitary hormone were being dealt with in the Child, Maternal, Prevention Division, it seems that similar iatrogenic complication was also being considered relative to BSE. This is consistent with the memo sent by Dr Jefferys to Dr Pickles, which indicated that the matter seemed to be about adverse drug reactions, as mentioned in chapter six. Having drawn a parallel between the administrative arrangements in relation to kuru and BSE, I turn to examine vaccination policy.

**Vaccination policy in England**

There are risks and benefits with any vaccination policy. The perceived risk in transmitting BSE in England in 1988/1989 was seen as being far outweighed by the benefits. This perception was summarised in the witness statement of Professor

\(^4\) Day 50 July 29\(^{th}\) 1998, Lister, Cunningham, Bridges.  
\(^5\) Witness Statement No 122 pt. 4 Cunningham
William Asscher, Chairperson of the Committee on the Safety of Medicines (CSM), who stated that the ‘risk-benefit analysis of exiting stocks of vaccines was comparatively easy’ for two reasons. One was that ‘... the risk posed by BSE to human health was ... always regarded as remote’, while the second reason was that vaccines were considered to be ‘very important to the protection of human health’. The CSM judged that the

... risks associated with interruption of the UK vaccination programme were far greater than the potential risk of BSE being transmitted.

Based on this risk assessment, rather than ordering ‘a withdrawal of stocks of efficacious products’, the CSM policy advised negotiation ‘with the manufacturers of such products so as to ensure they changed the sourcing of their bovine material as quickly as it was possible for them to do so’. Asscher stated that the idea that the risk was remote ‘was certainly the view of the Southwood Working Party in its report’. The policy then was aimed at not interrupting the vaccination program because the risk was seen as remote and vaccine use was highly prized.

At the beginning of 1989 as the Southwood Report was about to be released, the CSM and Veterinary Products Committee issued a joint statement to drug and vaccine manufacturers, which read:

“The CSM agrees with the Southwood Working Party that the risk to man of infection via medicinal products is remote. As a precautionary measure, and for the sole aim of seeking to guard against what is no more than a theoretical risk to man, the CSM and the Veterinary Products Committee (VPC) have agreed joint guidelines on good manufacturing practice for the manufacture of human and animal medicines who use bovine, or other animal, materials either as an ingredient or in the production process”.

This statement and the implied idea that the measures were only precautionary can be compared with the following comment, written in December 1988:

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6 WS No 441 pt 32 Asscher. Professor Asscher Chaired the Committee on the Safety of Medicines from 1987 to 1993 (pt 1.3).
7 ibid.
8 The Southwood Report was released in March 1989 following the work of the first expert group to consider BSE.
9 WS 441 pt.57 Asscher.
As I wrote in my earlier letter my colleagues are most anxious to ensure that existing products were identified and that manufacturers ensured that they conformed to the safety recommendations\textsuperscript{10}.

The term ‘most anxious’ in Asscher’s letter, which was written to Professor Southwood a few months prior to the joint CSM/VPC statement being issued, tends to suggest that using the precautionary principle might have understated the risk perceived by the CSM.

Contrary to media reports on the subject which suggest that eating meat and meat products is the main issue, the BSE Inquiry spent a considerable amount of time discussing the issue of medicines and their safety, and the reasons for the emergence of nvCJD is still an open question. The following interchange is useful as a gauge to the tenor of the discussion. Mr Kenneth Clarke, the Secretary of State post 1988 posed the following question,

You do not think we have any reason to think there is any risk from medicinal products now?

The then Sir Nicholas Phillips, Chair of the committee, interjected by saying,

the answer to that question is we do not know how the very small number of cases that to date have been diagnosed as having new variant of CJD have acquired the infection\textsuperscript{11}.

\textbf{The MMR Campaign}

It was established in the last chapter that vaccination rates were relatively poor in England during the late 1980s. Consequently, efforts were taken to increase the uptake rate of vaccines. The campaign against Measles, Mumps and Rubella (MMR) launched in October 1988 occurred at the same time as the joint guidelines were being revised by the CSM/VPC and the audit of pharmaceutical products was being

\textsuperscript{10} In Transcript Day 79. Acheson & France
\textsuperscript{11} Day 87. Clarke & Freeman.
conducted within Medicines Division. Specifically in relation to MMR, Cunningham, whose Division was responsible for the administration of the program, stated that ‘a lot of activity went into introducing this new vaccine’ and the division was involved ‘very much in encouraging the uptake of these vaccines’\textsuperscript{12}. Lord Newton, the Minister for Health at the time of the launch also pointed out that increasing the vaccination rate was a strategy outlined in the White Paper his department released during his time as Health Minister with Mrs Edwina Currie as Junior Minister\textsuperscript{13}. The question here is whether these forces, which were moving in the direction of increasing the rate of vaccination, played too greater part in the policy and adversely affected public safety.

During her evidence, Edwina Currie provided a hypothetical situation involving a planned vaccination campaign\textsuperscript{14}. We need to look carefully at the evidence because it might be the case that the situation was not as hypothetical as suggested given the line of questioning that the inquiry took with a focus on medicines and their safety. The hypothetical situation is as follows:

If, for example, we had planned a vaccination campaign, and we found at the last moment there was a problem with one of three suppliers, and we had to switch suppliers, and there might be a slight delay in delivering the vaccine to the north east of England, then Ministers would obviously have to be informed and would then be expected to deal with inquiries\textsuperscript{15}.

Because Currie was so specific in identifying a particular part of England, this tends to suggest that there might in fact have been a problem in providing MMR vaccine to this part of the country. A little later in the proceedings Phillips highlighted the other ‘counter-balancing cause for concern’; ‘the very danger people might stop asking for vaccinations and so on, before it was possible to re-source these’\textsuperscript{16}.

Relating to the issue of what was known and what to inform the public, Phillips posed the following hypothetical question to Clarke:

\textsuperscript{12} Day 50. Lister, Cunningham, Bridges.
\textsuperscript{13} Day 85. Newton & Moore.
\textsuperscript{14} Day 84. Currie.
\textsuperscript{15} \textit{ibid}.
\textsuperscript{16} \textit{ibid}.
Let us assume that the Committee had reached the conclusion that there was a significant although fairly remote risk that people might get infected ... from medicinal products sourced from a BSE infected cow. Having considered it very carefully, they reached the conclusion that the potential consequence of people abandoning vaccination because of the remote risk would be very much more serious than the risk itself. In those circumstances there might be two choices. One would be to say to the public: “There is a possibility, we do not think it should result in your refusing vaccines”, or they should say to the public: “Nothing to worry about”17.

As Phillips pointed out, option one might give rise to a scare and to some people at least ‘adopting the course which had been decided to be undesirable’. In light of this, he went on to ask which approach should be adopted. Clarke remarked that one would need to be guided by the option that most matches the advice one is being given but admitted that the first approach would be ‘much more difficult’. He said, that in his experience the CSM faced this problem all the time in other less sensitive areas. There were always side effects to consider ‘in a tiny majority [sic] of patients who use it’, and the question is, ‘is the risk to this tiny group of people outweighed by the benefit to this group of people?’ The Secretary of State vouched for the cautionary approach taken by the Committee on the Safety of Medicines18 and his recollection and impression was that the risk of infection from vaccination was remote. He was not aware then, nor is he now in light of current knowledge, that ‘we are facing a situation where there actually was a bit of a risk but the benefits of the vaccine outweighed it’. That was why, as Phillips noted, he posed the question as a ‘theoretical situation’. He did not think the question had been answered nonetheless, ‘otherwise than saying it was a very difficult one’. The question, as Clarke suggested, is always:

Does the benefit to that substantial block of people justify the licensing of a medicine which will, we know, have harmful effects, possibly death on this tiny number of people19.

Clarke did not believe that the situation under discussion fell into this category, at which point Mrs Bridgeman, a member of the Inquiry committee, interjected. Bridgeman reminded the Secretary of State that he mentioned that the decision would be based on the advice he received, but that he did not have any advice in this ‘particular dilemma on vaccines’. Clarke reiterated that the ‘advice we had was the

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17 Day 87. Clarke and Freeman
18 ibid.
19 ibid.
risk was remote’ and he ‘was unaware that such a dilemma existed in this particular case’. That the risk is remote from using vaccine or medicines produced with BSE infected cattle, is a mantra repeated throughout the inquiry hearings.

**What do we know about transmission of prion diseases?**

There are two areas that need to be considered: veterinary health and human health. Edwina Currie, the Junior Health Minister in 1988, noted that there was a lot of ‘guess work going on’ at the time of the MMR launch in relation to possible routes of transmission and the risk associated with them. But, she said,

> we already knew from kuru, which was mentioned at the time, that it was possible by eating infected animal brains to acquire the disease or a version of the disease; we knew that.\(^{20}\)

It is difficult to determine whether this is a case of typographical error, and ‘animal’ should read ‘human’ brain, or whether Currie lacked very basic knowledge of the kuru literature. The significant point is that the oral route of transmission is the focus of the statement. This focus was quickly changed at the BSE Inquiry. Immediately following Currie’s statement her attention is drawn to paragraph 5.3.2 of the Southwood Report, which says that:

> “Information from several spongiform encephalopathies suggests that parenteral inoculation is much more efficient in transmitting disease than oral or topical exposure …”\(^{21}\)

Attention was also drawn to the fact that the Southwood Report indicated that the risk of acquiring BSE related to transmission routes from cattle to humans could be ordered: “The greatest risk in theory, would be from parenteral inoculation injection of material derived from bovine brain or lymphoid tissue.”\(^{22}\). This serves to show the distinction that was made between *ingestion* and *injection*.

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\(^{20}\) Day 84. Currie.


\(^{22}\) *Ibid.*
While the distinction between inject and ingest might seem like a semantic triviality, the difference if significant in terms of what is known about how effective different routes of transmission are for prion agents. Understanding the differences will help to assess how likely is it that nvCJD has been acquired through eating meat, and how likely is it that kuru has been acquired through eating meat, be it animal or human.

Another important distinction that needs to be made is whether the risk was perceived to be remote as opposed to moderately high. Distinguishing between these terms will help to clarify whether the statements made in the vaccination policy and the Southwood report were consistent with what was known. If the risk associated with injecting potentially contaminated bovine material into humans was moderately high rather than remote, this meant that the pharmaceutical product manufacturers and the government that licensed the products for use had an enormous dilemma on their hands as stocks existed that were already potentially contaminated. Judging by the inquiry transcripts it seems possible that this was, in fact, the situation. The question was therefore whether the products should have been withdrawn.

Compare the idea that the risk was remote with the following comment made in July 1989 by Southwood. By this time, Dr D. A. J Tyrrell chaired a committee set up to look at research needs once the Southwood group had released its report, and the Tyrrell committee was writing an interim report. Southwood wrote that he believed Tyrrell was

\[\ldots\] absolutely right to point out gently how we are forced to argue from analogy with scrapie and one awaits, with some anxiety, the experimental confirmation of that assumption.\[\ldots\]

Personally, I would have thought the possibility of human infection was moderately high if some medicinal products were made from tissues of infected animals and injected into humans.\[23\]

\[23\] Dr D. A. J. Tyrrell edited *Aspects of Slow and Persistent Virus Infections. Proceedings of the European Workshop* sponsored by the Commission of the European Communities on the advice of the Committee on Medical and Public Health Research, held in London (UK) April 5-6, 1979. The Commission of the European Communities: Martinus Nijhoff Publishers – The Hague/Boston/London Day 87. Freeman & Clarke. NB. While this letter is spoken about as dated as July 5th 1989, the numbering code given to the letter by the Inquiry is 5.05, which means May 5th. It seems that this is a typographical error.
Southwood acknowledged that this was an extreme case, but his committee

... certainly had such anxieties very much in mind, and as you may know the whole series of recommendations, including the compulsory slaughter of animals.

It was suggested that if Tyrrell was thinking of making a few drafting changes, where at paragraph 2.2 on page 4 Tyrrell had written "[t]he Southwood Group was correct in their belief that this disease would not have implications for human health save through food", Southwood said that 'a more complete picture of our belief will be given if you added "provided various safeguards that were recommended were instituted"'.

The problem for the public is that the safeguards had not been implemented. One of the safeguards involved sourcing from other places. I mentioned in the last chapter that Australia and New Zealand were the preferred options. What is puzzling is that in 2000 it was reported that 'Australian vaccines can be traced to British cattle, more than a decade after the mad cow disease threat was revealed'. Professor Richard Smallwood, Australia's Chief Medical Officer, is reported to have said that Smith-Kline Beecham, the manufacturer of an oral polio vaccine in Australia, 'discovered only last week that British calf serum originally sourced from British cattle was used in making the Australian product'. A spokeswoman for Smith Kline Beecham declined to comment on how the company had missed the British link for so long. A 'fresh audit of the source of all vaccine material used in Australia' was to commence. These events followed the withdrawal of the polio vaccine produced at Medeva in England in October 2000. Why Australia has been sourcing from England, when England throughout the 1990s is said to have sourced its bovine material from Australia is a mystery.

There is, however, nothing mysterious about Australia's vaccination policy, which seems to be identical to the one in England. Quoting Metherell, Professor Smallwood said that

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25 Metherell 2000:4. One newspaper (Mann 2000:11) reported that the Australian product was made by Smith'Kline Beecham and Commonwealth Serum Laboratories.
the benefit of immunising children outweighed the remote theoretical risk of vCJD, the human equivalent of mad cow disease. An expert panel was auditing the source of material used in the production of other vaccines, although it seemed unlikely that these would be involved.

According to this newspaper report, the Therapeutic Goods Administration in Australia is reported to have said that ‘the chances of other vaccines being affected were one in ten trillion’. A figure of one in a billion was given as the chance of acquiring nvCJD from polio vaccine. Asked whether there were concerns the vaccine risk factor might be greater, given emerging evidence of vCJD prevalence in Britain, Professor Smallwood is reported to have said

Australian experts’ assessment concurred with that of the Federal Drug Administration, which had cleared continued use of vaccines “grown” in the British Calf serum27.

Metherell’s article is brilliant at capturing the arguments I am attempting to outline, for while the words used in the article might not be a verbatim account of Professor Smallwood’s views, they no doubt convey what was said, as the argument is so similar to the one used in Britain. It is an argument used, I suggest, based on the belief that it is important to allay public anxiety in order to maintain the immunisation program.

We need to consider the message that is conveyed to the public when terms such as “remote” and “theoretical” are used. And the effect it might have on the public when figures such as one in ten trillion and one in a billion are given. On what evidence are these figures based and what do the terms mean?

The Collins English Dictionary defines “remote” as being synonymous with doubtful, implausible, small, slight or unlikely (Collins 1994:974). “Slight” means small in quantity, of small importance, insignificant, negligible, trifling, trivial and unimportant (Collins 1994:1086). A remote risk then could be interpreted as one that is unlikely but possible, and if this were to prove to be the case a small number of people would be affected. Collins defines theoretical as ‘lacking practical application

or actual existence; hypothetical’. Synonyms for the word are, conjectural, speculative, ideal, pure (1994:1202). Yet theory does not belong in some other world, completely removed from the real. A theory should have little credibility unless it is linked somehow to evidence. Even a speculation, which I would distinguish from theory, is usually based on some sort of observation. The problem with determining the risk in the case of prion diseases in a new species, as is the case regarding BSE and nvCJD is that calculations need to be made on assumptions. If the assumptions are incorrect, the figures can be affected greatly. In the case of the risk of transmitting prion agents through medical products and procedures there are well documented precedents in specialist circles which would warrant an extremely cautious approach as regards taking measures to prevent these diseases. When messages about this issue are provided to the public, the significance of the problem seems to be trivialised when figures like one in a billion and one in a trillion are used. Such figures offered up within the context of the problem being only theoretical might leave the impression in the minds of the populace that no one knows the potential risk and the chances of there being a problem is not worth worrying about. While I agree that at this stage it is not possible to know the actual risk, it is doubtful that any one with knowledge in this area would agree that the risk is not worth worrying about. But this is at odds with the messages of assurance given to the public. As of November 2nd 2000, despite knowing that the Australian product has used British bovine material, the CMO is reported to have said that the Australian product would not be withdrawn. I have heard no announcement that this has changed.

In England 11 million doses of the Medeva vaccine have been administered. One report puts the figure at ‘an estimated 35m doses, accounting for a third of all oral polio vaccine administered’. The two different accounts in terms of the figures might reflect an attempt to allay public anxiety in Australia as the higher figure is reported in *The Guardian Weekly*. Regardless of the actual figure, it would be wise to ask if the company that produced the *third* in this case is the same company that produced the third in Currie’s hypothetical example in relation to MMR vaccine? This question needs further research.

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29 Mann 2000:11  
30 Meikle & Watt 2000:8
When Southwood wrote to Tyrrell in 1989 in England about the perceived moderately high risk associated with injecting bovine material infected with BSE, and mentioned the safeguards that needed to be put in place, another one of the safeguards relates to the detection of sub-clinical animals. That is, animals not showing signs of BSE but who might have been incubating the disease nonetheless. The problem is that neither then, nor now, is it possible to test animals as no definitive test exists. Southwood’s letter suggests that it would not be possible to say that ‘this disease would not have implications for human health save through food’ without transmission studies being conducted and no detrimental effects observed. It appears from his letter that finances influenced the studies that were undertaken, for he hoped that the Minister and others would, ‘notwithstanding the ridiculous attitude towards public expenditure, find the necessary funds to undertake the high priority research’. The man was ‘horrified to discover a little while ago that the former control study of possible vertical transmission had not yet been put in place’. It would be wrong to conclude from the letter however that no transmission studies were undertaken on BSE.

In 1990 a report was sent to the Chief Medical Officer, which outlined the state of knowledge about transmission routes. The document, called ‘Opinion on the public health implications of eating beef and the epidemic of BSE’, dated July 24th 1990, at paragraph five states that,

“Oral transmission of some spongiform encephalopathies undoubtedly occurs – although very large doses are needed because the oral route is very much less efficient than, say, intracerebral inoculation.”

Section five of an annexure pertaining to the above document reads

“The oral route is clearly capable of transmitting spongiform encephalopathies in the diseases of BSE and transmissible mink encephalopathy, and it is assumed to be an important natural route of transmission in scrapie and kuru”.

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31 In August (18th) and September (1st) 2001 there have been reports about a diagnostic test for BSE following the discovery that ‘the prion isofrom [found] in urine may lend itself nicely to the development of a non-invasive diagnostic test for prion disease’ (New Scientist September 1, 2001 p. 55).

32 This document, which originated from the “MRC Common Cold Unit”, was addressed to the Chief Medical Officer Sir Donald Acheson (December 16th 1990 See Transcript of Professor Will December 16th 1999).
The document then went on to say that

"Experimental studies, however, show ingestion to be very inefficient, at least five orders of magnitude less efficient than intracerebral injection. In the transmission work done to date with BSE, the incubation period in mice was longer after large oral doses of BSE-infected cattle brain than after much smaller parenteral injection. In these as in other animal experiments, large doses, far in excess of what would be experienced in nature, appear to be needed for successful disease transmission".

The conclusion drawn was that

"... the occasional low doses of BSE agent in human food are well below those capable of infecting humans, even if humans were specifically susceptible to the agent".

This serves to highlight the point that oral transmission through meat, whether in the case of BSE or kuru, is the least effective means of transmitting prion diseases. It could be argued that oral polio vaccine is therefore of low risk when produced using BSE infected material. This ignores the fact though that in England, even it was considered to be of sufficient risk to withdraw it from sale. It is important to take into account the different means by which vaccines are produced in comparison with meat.

The issue of risk pertaining to route of administration was a subject of debate right throughout the late 1980s and 1990s both in scientific and government circles. On many occasions kuru is used as an example. Clarke, Secretary of State from 1989 stated that he remembered discussions taking place about CJD (meaning new variant CJD) and whether it had been acquired through ingestion after the first case of nvCJD became evident. His comments bear repeating. Like Currie, he also referred to kuru in a way inconsistent with the dominant literature, while speaking about nvCJD having been acquired by vegetarians. He stated that ingestion was not ruled out because:

... there was this question of kuru which I seem to remember is a disease that seemed to exist among the head hunters of Papua New Guinea, that they started to ingest the brains of their slaughtered animals. It did seem there was a possibility they had created a related disease by doing that."33

33 Day 87. Freeman and Clarke.
Putting aside for the moment the stereotypical depiction, while it is curious that two
government officials spoke of animal brain as opposed to human brain, this should not
detract from the knowledge that oral transmission was believed to be the least
effective route. Southwood’s letter to Tyrrell gives the impression that the
recommendation to remove contaminated meat from circulation was an action taken
as much for the reason of preventing its use in medical products as it was to remove
the meat from the food chain, although this was also important.

The irony is that the continued referral in the public domain to the idea that the risk
was remote from eating beef seems to have been right based on the experience with
scrapie. For scrapie has not been acquired by humans through eating meat during the
past nearly two hundred years. But in the case of medicines, particularly those
injected into the body made with brain, spleen or placenta, the risk was assessed as
much greater based on previous experience with louping ill vaccine and pituitary
hormone. Information about these risks associated with products administered through
routes other than the oral route is rarely spoken about in the public domain whether in
relation to BSE, nvCJD or kuru. The only case where the injectable route has been
acknowledged relates to the hormone responsible for iatrogenic CJD. In the case of
kuru, I argue that the discourse is confounded because of images of cannibalism,
head-hunters, madness or dementia, which so often accompany any reportage of this
disease, even its seems at inquiries into the disease and in government circles. The
distorted images have been carried through into later reportage of BSE, which place
an undue emphasis on mad cows as opposed to how the role of medicines or vaccines
might have played a part in the emergence of BSE in the first place.

Veterinary medicines during the 1980s

There has been recognition of the need to avoid using ovine and bovine material in
pharmaceutical products in veterinary medicine since 1983. According to Sir James
Armour, Chair of the Veterinary Products Committee (VPC), the government issued
guidelines at this time\(^\text{34}\). The VPC ‘were aware that scrapie had been disseminated via

\(^{34}\) MAL 67 – MAFF guide cited in WS No 477 pt. 8b. Armour.
a loping ill vaccine ... in which brain and spleen tissue from scrapie-infected sheep had been inadvertently incorporated. As well as knowing about loping ill vaccine, pituitary hormone was also known to be problematic in veterinary medicine, perhaps not surprisingly considering that during the same period the hormone was shown to produce CJD in humans.

In 1987 the Head of the Medicines Unit at the Central Veterinary Laboratories wrote a cautionary note for the journal *Veterinary Record* about the potential to transmit scrapie through pituitary hormone, in which he stated,

... the use of brain tissue of bovine or ovine origin, particularly the pituitary-derived follicle stimulating hormones, could, by analogy from previous work on scrapie, possibly cause contamination of the BSE agent and recommended that manufacturers should source these hormones from species other than cattle or sheep.

This statement indicates two things. Pituitary hormone is associated not only with human prion disease in the form of iatrogenic CJD but is also associated with the transmission of scrapie. And two years after the problems associated with pituitary hormone in the human situation had been realised, it would appear that pituitary hormones made from potentially infected cows or sheep were still being used in veterinary medicine.

Dr James Rutter, Director of Veterinary Medicines and Chief Executive of the Veterinary Medicines Directorate, explains how in 1988,

... products in the hormone group were called up for review which required the licence holders to submit updated dossiers to the licensing authority. From this review it transpired that four products in the pituitary hormone group that may have contained bovine material were on the market. These products were not defended and the four licences had expired by April 1991.

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35 Sir James Armour, now retired, held the position of Chairman of the Veterinary Products Committee (VPC) from 1987 to March 1996 and since 1985 had been a member of the VPC. Simultaneously, during the period 1986-1994 Armour was 'a member of the Scientific Advisory group to Merck Research Laboratories', and prior to this between 1977-1985 he also acted as consultant to Merck Sharp & Dohme Animal Health. All these 'interests were declared'. From 1953-1960 Armour worked as a Field Veterinary Officer and then Veterinary Research Officer, Colonial Service, Nigeria'. Later, from 1960-1963 he worked at the Cooper Technical Bureau (latterly Wellcome), in Berkhamstead, Hertfordshire (Witness Statement No 477. Armour).


This statement indicates that products in this group were being used until 1991 in veterinary medicine, which should make us take note of the possibility that BSE might be attributable, at least in part, to the use of products in the pituitary hormone group. In April/May 1988, the VPC received a request from the Medicines Unit, CVL [Central Veterinary Laboratories] to comment on draft guidelines for the review of veterinary products containing hormones which were being prepared for issue. Members of the VPC were concerned about the possible risk of BSE contamination emanating from the use of serum and glandular extracts from cattle or sheep where these species were the source of the hormones. As a consequence of the concern a paragraph about sourcing outside of Britain was inserted into the guidelines for manufacturers in September and October 1988 and the revised guidelines were issued to pharmaceutical companies at the end of November.\(^{38}\) Hence, the relevance of the joint CSM/VPC statement outlined earlier. The use of the precautionary principle in light of this knowledge does not seem to have been appropriate in terms of protecting the health and safety of both humans and animals, for the VPC were aware of pituitary hormone, louping ill vaccine and the associated iatrogenic transmission of prion diseases. This committee is charged with responsibility for the safety and efficacy of veterinary medicines as well as for products with implications for the safety of humans and the environment.

In December 1990, the Veterinary Medicines Division circulated a final report of the results of a questionnaire sent to its members, which indicated that ‘companies had been taking a range of measures to comply with the guidelines but that advice was required on a few issues’.\(^{39}\) The Veterinary Products Committee made four points:

Use of any bovine brain material during manufacture must not take place

In relation to the SKP bulk viral vaccines made with filtered donor calf serum from UK sources, further information was required on the herd history of the source of the donor calf serum

Dr Taylor at MAFF should be consulted on whether the autoclave treatment of bovine soup stock of UK origin prepared from bones at EC-approved abattoirs was satisfactory prior to its use in culture media, and

\(^{38}\) WS 477. Armour.
\(^{39}\) ibid.
... action taken in relation to biological products should conform to EC regulations on BSE\textsuperscript{40}.

This demonstrates the uncertainty as regards the safety of some veterinary vaccines at the end of 1990. Add to this the concerns expressed about pituitary hormone a few years earlier and there is certainly a reason to consider the role that these products played in the production of prion disease.

The Chair of the Committee on the Safety of Medicines suggested that pharmaceutical manufacturers acted quickly to source bovine and ovine material used in their production processes from non-British stock. From the perspective of some individuals this might be so. But from the perspective of the populace it might be that the changeover was not carried out quickly enough. I doubt that the public would be impressed to discover that Directive 81/851/EEC, which came into force in November 1983, made provisions for the guidelines ‘to be applied progressively, within 10 years, to veterinary pharmaceutical products already on the market in member states’\textsuperscript{41}. In 1991 it was reported that all manufacturers could ensure sourcing from non-British bovine material, and in 1992 the government declared that all medicinal products were safe. While it is commendable that the changeover occurred within the time of the provision, it is not so surprising that the announcement that vaccines were now safe was made in 1992. A question to ponder is, is it the case that BSE, at least in part, has been produced by veterinary vaccines or hormones and then material from these animals has been used in human medicines and vaccines and produced at least some of the cases of iatrogenic nvCJD?

Given the iatrogenic pathways known to produce prion diseases, it would be unwise to ignore the state of knowledge relating to both veterinary and human medicines. The fact that the Division of Children, Maternal and Prevention was given BSE to administer and that this department also administered vaccination policy would be consistent with this line of reasoning. As to the degree of influence the MMR campaign had on vaccination policy, this is difficult to gauge but given that CMP was

\textsuperscript{40} ibid.
\textsuperscript{41} WS No 499 pt. 20 Rutter.
working hard to improve the uptake of vaccines, as was the Minister for Health, it
does seem that not interrupting the campaign was the overriding concern. It is
possible that the Committee on Safety of Medicines did not know about the degree of
risk perceived by some individuals. This is unlikely however given that the CSM and
the VPC worked together to issue joint guidelines. Given this, the decision not to
withdraw existing stocks of potentially contaminated products seems inappropriate to
say the least, for there were other products discussed at the Inquiry made prior to 1992
besides MMR and polio vaccine that require evaluation\footnote{See Appendix 7c.}

Distribution patterns have been traced since the release of the Phillips Report in
October 2000 and the polio vaccine being withdrawn. On March 3\textsuperscript{rd} 2001, it was
reported in a radio report that four and a half thousand children in Ireland had been
exposed to polio vaccine with expired use by dates\footnote{ABC News Radio Report March 9\textsuperscript{th} 2001. Rachael English speaking to Dr Cormack McNamara in
Dublin.}. The message given to the public
was that this would not cause a problem apart from the fact that the vaccines might
have been ineffective. I am not so sure. While there is to date no conclusive proof that
vaccinations have caused nvCJD, a policy of ‘do not interrupt the vaccination
program at all costs’ might well be in need of reassessment.

The position taken in Australia in relation to vaccination policy mimics the situation
in England and it appears that a culture similarly lacking in transparency operates,
judging by a television report in February 2001. Rebecca Smith from the Australian
Consumers Association is disappointed by the NHMRC expert Committee’s lack of
transparency and the fact that the meetings were held behind closed doors\footnote{Insight (2001). SBS TV Inga Johansen reporting on BSE in Britain, February 22\textsuperscript{nd}.}. The
public was informed on the program that most people in the UK have acquired vCJD
from eating meat and we do not know if there are other ways of transmitting the
disease. It is understandable that health departments do not want to alarm the public.
But in light of the information presented, it seems preferable that, in future, the public
is informed about what is known and blanket statements such as, we do not know
about other ways of transmitting BSE or similar diseases are avoided.
An attempt was made to distinguish between medicines in toto as opposed to a particular batch, at the BSE Inquiry. Freeman began by saying, while it is one thing, to remove that batch or that product from the market where you might have an easily, relatively easy means of sourcing the supply from elsewhere. Of course it is a much more difficult problem if it were the case that there was a substantial or significant risk in relation to vaccine or pharmaceutical products generally which may be derived from bovine products.45

Here is the crux of the matter. If it is the case that it was not a question of a particular batch being contaminated but rather a problem of far greater import, it is not difficult to see why such information has not been widely disseminated.

Medicines and secrecy

It is apparent from Meikle’s comment quoted at the end of the last chapter, that by as late as February 2001, the licensing process for medicines was an area in need of review.46 Questions were raised at the BSE Inquiry about lines of accountability and secrecy surrounding the Medicines Commission. Here a brief outline will be useful of the structure and authority of the CSM and its sub-committees, which were known as Section 4 committees. These committees derive their authority from Section 4 of the Medicines Act 1968. As Currie’s statement attests, the CSM licenses products47 and the Medicines Act ‘actually gives power to these authorities to take decisions’48. Appendix 7a provides a flow chart of information with respect to the licensing process. The important thing to note is that the Chief Medical Officer is not privy to the flow of information, and those working in Medicines Division (later the MCA), while accountable to the CMO were also accountable to the Medicines Commission (see Appendix 7b). Bridgeman confessed to being confused about the role of the Medicines Commission49 and described it in the following terms:

43 Day 87. Clarke & Freeman.
45 Day 84. Currie.
46 ibid.
47 ibid.
48 ibid.
It is a curious sort of quango established under the Medicines Act and not quite the same sort of creature I have been accustomed to in other Departments, because it appeared to have certain decisions delegated to it which then had to be kept in secrecy from the Department, so it was rather peculiar, this business surrounding medicines.\footnote{Day 85. Newton & Moore.}

Lord Moore, Secretary of State prior to the time that Clarke assumed this office, agreed: ‘You are right, there were some rather peculiar structures insofar as that was concerned’. What this meant in effect, was that officials in Medicines Division were prevented from disclosing commercially sensitive information to the CMO. Commercial confidentiality and built-in structural conflicts of interest, therefore, play an important role in preventing information flowing outside of the deliberations of the CSM and its subcommittees.

As to the question of the line of accountability, Bridgeman noted that the Chief Medical Officer had told the inquiry that ‘he did not feel he had a locus in relation to the Committee on Safety of Medicines’. Currie, when asked whom the CSM reported to, replied ‘I think each other would be the right answer’\footnote{Day 84.}. She expressed the view, as did Clarke that the committee always acted with caution.\footnote{\textit{Ibid}.} In Currie’s case, her confidence came from the fact that if problems arose, Ministers were kept informed. Ministers were not always kept informed though judging by Currie’s testimony, as she went on to note that it would have been overwhelming, apart from being ‘administratively efficient’, had this been the case. Nevertheless, during her time in office (up to the beginning of 1989) the CSM was delegated to explore the issue and ‘ensure that the products were safe as a result of their actions and decision’. Then it would be up to Ministers to back any decision taken by the committee.\footnote{\textit{Ibid}.} It was not the task of a Junior Minister to ‘gain say their decision’.\footnote{\textit{Ibid}.} Notwithstanding the secrecy involved in these arrangements, it is ultimately the responsibility of the Secretary of State to ensure that any recommendations that come from the CSM are workable and supported by enough evidence to withstand legal challenge\footnote{For a discussion on the dual lines of accountability in Medicines Division see Transcript of Day 79 (Acheson and France), and on the legal implications of sanctioning recommendations such as the Specified Offals Ban, transcript Day 87 (Freeman and Clarke), is useful.}. As Moore
stated, ‘... whatever you call the work of the Medicines Control Agency [to whom the CSM report], ultimately, the Secretary of State is responsible to Parliament for them’.\textsuperscript{56}

Lord Freeman, the Minister for Health in 1988, pointed out to the Inquiry that ‘before Southwood finally reported, action had already been taken to destroy tissues of infected animals’.\textsuperscript{57} He made the point to demonstrate that he ‘quite understood that some medical products might have been made before the destruction of affected cattle came into operation, which is August 1988’. Clarke, Secretary of State in 1989, fielded a hypothetical question at the inquiry about what he would have done if faced with 'a whole series of pharmaceutical products on the shelf which are derived from a bovine product' which might be infected and carry a moderately high risk. Clarke suspected that

... the Secretary of State would probably wind up ordering withdrawal of all batches that might be affected if "moderately high" meant what you – I mean I agree it could well mean that a significant number of patients may be infected by this in that case one would probably withdraw unless the advice was some catastrophic consequences for some patients would ensue or with a large number of patients if you did that.\textsuperscript{58}

Were there not grave concerns about the safety of medicines and vaccines, it is difficult to see why the inquiry took the line of questioning that it did. The responses of some individuals in positions of influence at the time of the MMR campaign and in the years that followed, demonstrate one of the possible outcomes of vaccination policy that negotiates with manufacturers rather than withdrawing medicines known

\textsuperscript{56} Transcript Day 85. Newton & Moore. Events during the period 1986 to 1987 are covered in this statement. Lord Moore was the Minister of Health from 1986 to mid 1988 and then became the Secretary of State in June 1988. Moore had also been Secretary of State prior to 1986 when he had been involved in AIDs administration. He compared the open communication in the case of AIDs as opposed to the lack of openness involved in BSE. Lord Newton, as well as being the Minister for Health, was the parliamentary adviser to the Royal Pharmaceutical Society of Great Britain, which is the body concerned with the discipline of pharmacists, much like the General Medical Council. This body does not represent the pharmaceutical industry. Newton discusses the problem of making a decision about the vaccine dilemma at the time, but defers to higher authority like the Chief Medical Officer. As I have shown, the CMO was not privy to all of the information. Note: it is difficult from the transcripts to get an exact sense of who held the position of Secretary of State and Minister for Health during the period 1986-1989. It is apparent that Lord Moore, while he was appointed Secretary of State in June 1988 (see above) did not hold this position for long, as Mr Kenneth Clarke was the Secretary of State in 1989.

\textsuperscript{57} Day 87. Clarke and Freeman.

\textsuperscript{58} \textit{ibid.}
to have incorporated bovine material infected with BSE. The situation described indicates that there might be a reluctance to acknowledge that any of the cases of nvCJD that have occurred might be related to vaccines and medicines.

A culture insistent on not interrupting the vaccination program seems to be one in which unwanted effects would not be welcomed and might be minimised should they start to appear. This is not to imply that drug reactions are not documented in England at all, for as Dr Gerald Jones noted, the process of collecting information on Adverse Drug Reactions began in 1959 when there was a large reaction to a particular drug\textsuperscript{59}. Jones does not mention which drug, but this quite possibly relates to polio vaccine and/or loping ill vaccine which had been problematic in England throughout the nineteen fifties. The transmission of kuru should be considered within this context.

To summarise the situation and to conclude with a reflection on earlier times it will be possible to show that secrecy surrounding vaccine and medicines is not a new problem. In the 1988 situation, I have shown how information was prevented from reaching even the CMO let alone the public. In 1967, Professor Wilson remarked that information about vaccines is not widely disseminated (Wilson 1967). When the first international conference on vaccines was held in 1966, Horwitz expressed the hope that the lively and productive discussions would ‘be spread throughout the world’ (Horwitz in WHO 1967). My findings support the idea that such information is not being as widely disseminated as Horwitz might have hoped. Chapter four provided a possible reason for not openly discussing the hazards of immunization; the threat of losing one’s position seems to have acted as a powerful reinforcer that cautions researchers against speaking about worrying findings in research related to the manufacture of pharmaceutical products. This culture is characterised by not being as frank as one might hope with the public regarding the issue of iatrogenic reactions. Such a culture has existed for a long time.

In 1920, Chas M. Higgins, in an alarmingly entitled book, \textit{Horrors of Vaccination}

\textsuperscript{59} Transcript 13/12/9 G. Jones. Jones was called again to give evidence during the last week of the Inquiry’s sitting days, as were many individuals involved in ensuring the safety of medicines.
Exposed and Illustrated, argued that the real situation involving vaccination was either denied or concealed. In his opening statements, in which he made a public challenge to the Health Departments in the State of New York, Higgins wrote:

In order that there is no misunderstanding about the serious charge which I shall bring against vaccination, as being now actually more dangerous to public health than natural smallpox, and the equally serious charge which I make against vaccinating doctors – who now control our Departments of Health and Vital Statistics – of denying and concealing these facts from the people. ... If they [the Public Health departments] deny the truth of these charges I further solemnly challenge them to open their now concealed records to public examination and I will prove the truth of my charges from these records.... (Higgins 1920:vi).

Higgins’s main aim was to stop the compulsory implementation of vaccines to the armed forces in America and it was for this reason that he addressed his challenge to the then President, Woodrow Wilson, who as Commander-in-Chief of the Army and Navy was in a position of authority to alter the situation.

Higgins’s work documents many examples of vaccine reactions and deaths that were described to him by doctors and members of the public, along with accounts described in government bulletins, particularly from U.S Public Health Reports and the Department of Agriculture. There were many types of vaccination that concerned Higgins but his views on smallpox are the most relevant here, for in Higgins’s mind smallpox vaccine was associated with outbreaks of foot and mouth disease during the first decade of the twentieth century.

In support of his argument Higgins cites one U.S. Public Health report for September 2nd, 1910 (pp 95 and 99), which stated that Japanese vaccination was the source of the deadly epidemics of Cattle Plague, known as “Foot and Mouth Disease”. At this time, the problem for vaccine manufacturers in America was that they were using the Japanese seeding virus. The cattle plague led to the ‘slaughter of hundreds of thousands of animals, an unknown amount of human mortality, and a loss of millions of dollars to the Government and people’ of America (Higgins 1920:55-56). Given that outbreaks of foot and mouth disease have occurred in conjunction with BSE and

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60 This information is sourced to the supplement for the Year Book U.S. Department of Agriculture, 1914 page 21, and page 99

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nvCJD at the beginning of the twenty first century, and that foot and mouth disease outbreaks in Europe were caused by vaccine against FMD during the early 1990s, as noted earlier in the thesis, it might serve us well to take note of Higgins’s text.

Australia has not had an outbreak of foot and mouth disease for one hundred and thirty years. As of March 1st 2001, Australia tightened its quarantine procedures to prevent the spread of this disease to Australian shores. While this might be the case, as shown in chapters two and three, foot and mouth disease has been discussed by scientists in relation to culture contamination since the nineteen thirties in both England and Australia. During the ‘thirties, foot and mouth, poliomyelitis virus and loping ill virus were all discussed simultaneous with the outbreaks of scrapie transmitted through loping ill vaccine. During the nineteen fifties, foot and mouth disease was discussed along with kuru at a working lunch, at which time poliomyelitis vaccine and loping ill vaccine were also problematic. In 1962 foot and mouth sera was being moved to the Animal Section of laboratories in Australia at a time when the hazards associated with working with human and animal studies in the same laboratory was being realised. Taking a broad perspective over the last century it is not difficult to see a pattern of similar problems emerging at different times.

Nearly a century after Higgins’s cautionary tale it might be wise to start disseminating information rather than relying on outdated vaccination policies and precautionary principles, which in some cases enable a lack of safety to be built into the system by minimising the dangers. As it would to review the lines of accountability relative to medicines and their safety and to open up the process to public scrutiny. Together these measures might provide a more transparent system where the need for inquiry after inquiry relating to prion disease would not exist.

During the early years of the twentieth century, any criticism that was made about vaccine was generally made by the public rather than by scientists. Wilson, in 1967, suggested that this situation had changed, as criticisms started to come from members of the scientific community as well as from the public (Wilson 1967). Such criticism is often based on empirical evidence of the effects a particular vaccine or medicine can produce; the risk is shouldered by all members of the community who receive medical products that have the capacity to transmit prion and other diseases.
This survey of what is known might help to move vaccination policy onto safer
ground. It is not a plea to ban the use of vaccine but if vaccines, hormones and sera
continue to be produced then it behoves us all to try and understand what can happen
and to minimise the potential hazards, thereby reducing the consequent damage. On
the basis of the evidence presented, I argue that there is a need for further archival
research and for serious reconsideration of kuru and its causes before it is relegated to
a footnote in medical history.
FRANCK MACPHERLANE BURNET - SERIES 10

AUSTRALIAN SCIENCE ARCHIVES PROJECT

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GUIDE TO THE RECORDS OF FRANCK MACPHERLANE BURNET
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Prepared by Gavan McCarthy, Oscar Manhal and Lisa O'Sullivan
with Tim Sherratt

Published December 1993 in hardcopy form.

The records of F.M. Burnet were deposited with the University of
Melbourne Archives in 1991. Enquiries about access should be directed
there.

Other enquiries about this GUIDE or orders of the complete hardcopy
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SERIES 10 X-DISEASE, KURU AND GAJDUSEK FILES
******************************************************************************

PART 1 X-DISEASE AND KURU, RESEARCH AND CORRESPONDENCE FILES

10/1 X-DISEASE - CORRESPONDENCE AND CLINICAL RECORDS
Inwards correspondence from: NSW Department of Agriculture; CSIR
Animal Health Division; R. Hughes; Ian (?); Austin Hospital; J.A.
Galloway; C. Duncombe; E.M. Pullar; Commonwealth Department of Health;
A. Mark and others. Includes: material on loping illness, post
vaccinal encephalitis and polio; clinical notes and records of
patients.
23 October 1923 - 21 October 1935, _5mm_

10/2 VISIT TO NEW GUINEA - PRELIMINARIES
Inwards and outwards correspondence with: F.M.C. Hasluck; J.T.
Gunter; Commonwealth Department of Territories; New Guinea Department
of Public Health; A. May; J.H. Hale; Rockefeller Foundation (R.S.
Morison); University of Malaya Department of Bacteriology. Includes
medical training pamphlet in pidgin and material on Murray Valley
encephalitis.
16 April 1956 - 5 July 1956, _4mm_

10/3 KURU 1 - GAJDUSEK LETTERS, 1957
Includes index to files Kuru 1 - Kuru 10. Mostly inwards
correspondence from: D.C. Gajdusek; R.W. Hornabrook and I.J. Wood;
13 May 1957 - 24 May 1963, _4mm_

10/4 KURU 2 - REPERCUSSIONS IN 1957
Inwards and outwards correspondence with: J.T. Gunther; A.L.G. Rees;
CSIRO Division of Industrial Chemistry; Medical Journal of
Australia, with typescript notes on Kuru; G. Anderson; R.M. Berndt;
R.F.R. Scragg; D.C. Gajdusek; I. Wood; T.M. Rivers; National
Foundation for Infantile Paralysis; New York; Commonwealth Department
of Territories; New Guinea Department of Health; New Guinea Crown Law
Office; Ian Burnet; F.M.C. Hasluck. Includes typescript notes on Kuru
and its aetiology, February 1957.
21 August 1956 - 6 December 1957, _6mm_

10/5 KURU 3 - ADELAIDE WORKERS
Inwards and outwards correspondence with: University of Adelaide; H.N.
Robson; J.H. Bennett; J.T. Gunther. Includes reprint and typescript
by Bennett, Rhodes and Robson, 'The Genetical Study of Kuru', no date
6 January 1959 - 11 September 1959, _3mm_

10/6 KURU 4 - SYDNEY MEETING OF DECEMBER 1959 AND SUBSEQUENT ACTIONS
Inwards and outwards correspondence with: N. McArthur; R.P.R. Scrugg; New Guinea Department of Public Health; J. Gunther; D. McCarthy; Department of Territories; J.E. Snedel; H.N. White; National Institutes of Health, USA; T.E. Lowe; Alfred Hospital; C.C. Curtain; J.H. Bennett; H.N. Robson; University of Adelaide; D.C. Gajdusek, with copies of D. Gajdusek to others. Mostly on formation of Committee on Control of Kuru. Includes: manuscript notes on Kuru; bibliography of papers by D.C. Gajdusek; copies of Committee Report, 21 December 1959; copy of 'Report on Kuru and Recommendations for its Further Investigation and Control' by J.T. Gunther, May 1960; copies of Minutes to Meeting. Preliminary meetings for Director, Public Health, Papua New Guinea, 18-19 May 1962; FMB typescript, 'Virology Lecture - Scrapie, aleutian disease, Kuru and NZB', 19 October 1963.
3 September 1959 - 25 November 1963, _10mm_

10/7 KURU 5 - 'CANNIBALISM IN THE KURU REGION'
Copy typescript of paper by R.M. Glasse. Includes additional copy typescript, 'The Social Life of Women in South Fore' by S. Glasse. No date (1960s?), _4mm_

10/8 KURU 6 - DEMOGRAPHY AND N. MCArTHUR
Copies of demographic records and reports, including: 'Report to Medical Research Advisory Committee (Papua New Guinea)', 28 August 1963; 'The Age Incidence of Kuru', no date (1960s). Also includes notes, and letter from N. McArthur.
August 1963 - January 1964, _6mm_

10/9 KURU 7 - REPLIES TO DRAFT PAPER ('THE PATHOGENESIS OF KURU')
Notes and correspondence. Includes inwards and outwards correspondence with: R.W. Hornabrook; R.P.R. Scrugg; M. Alpers; R.M. Glasse; R.J. Walsh; Red Cross Society; C.C. Curtain; D.C. Gajdusek; P.D. Schofield. Mostly replies and comments on 'The Pathogenesis of Kuru'. Includes summary of replies.
8 January 1964 - 11 March 1964, _3mm_

10/10 KURU 8 - P.R.J. BURCH AND J. MATHEWS
21 October 1963 - 17 March 1964, _5mm_

10/11 KURU 9 - LONDON MEETING, 15 JUNE 1964
Copies of reports for Meeting on Kuru at the CIBA Foundation, 15 June 1964. Includes meeting papers, including FMB's hypothesis. Also includes draft and copy typescripts by FMB: 'The Possibilities of Control', no date (1964); 'The Pathogenesis of Kuru: Speculations based on new Observational Material', no date (1963).
1963-1964, _11mm_

10/12 KURU 10 - SCRAPIE REPRINTS
Annotated reprints by H.B. Parry, H. Jacob and others.
1962-1964, _7mm_

10/13 KURU RESEARCH CORRESPONDENCE
Inwards and outwards correspondence with: D.C. Gajdusek; M. Alpers; R.P.R. Scrugg; R.J. Walsh; R.M. Glasse. Includes: meeting notices for November 1965; journal extracts; bibliography on Kuru, by D.C. Gajdusek, 1963(?).
15 February 1965 - 23 August 1968, _3mm_

10/14 KURU CORRESPONDENCE AND GAJDUSEK'S NOBEL NOMINATION
14 January 1976 - 21 July 1978, _8mm_
10/15 GAJDUSEK CORRESPONDENCE
Correspondence from D.C. Gajdusek.
March 1980 - June 1980, _1mm_

PART 2 KURU CORRESPONDENCE - PHOTOCOPIES FROM WEHI

10/15 KURU CORRESPONDENCE, 1956-1963
Inwards and outwards correspondence with: D.C. Gajdusek; J.T. Gunther;
J.H. Hale; F.M.C. Hasluck; A. May; Department of Territories and
others.
9 May 1956 - 24 May 1963, _5mm_

10/17 KURU CORRESPONDENCE, 1957-1959
Inwards and outwards correspondence with: J.H. Bennett; J.T. Gunther;
H.N. Robson; R.S. Morison; F.M.C. Hasluck; R.F.R. Scragg; D.C.
Gajdusek and others.
8 May 1956 - 28 August 1959, _12mm_

10/18 KURU CORRESPONDENCE AND REPORTS, 1959-1963
Correspondence with: R.F.R. Scragg; J.H. Bennett; H.N. Robson; D.C.
Gajdusek; J. Smadel; J.T. Gunther and others. Includes Committee on
Kuru Control Reports.
1959-1963, _10mm_

PART 3 PUBLICATIONS - REPRINTS AND GAJDUSEK JOURNALS

10/19 REPRINTS - ASSORTED AUTHORS
Reprints on Kuru, Foamy Viruses, Creutzfeldt-Jakob disease,
encephalitis, dermatoglyphics and other topics. Includes articles by:
M. Alpers; H. Ward; R. Glasse; C.J. Gibbs and others.
1958-1977, _17mm_

10/20 REPRINTS - GAJDUSEK PUBLICATIONS
Articles on Kuru, Scapie, Creutzfeldt-Jakob Disease and other topics
by D.C. Gajdusek.
1957-1965, _22mm_

10/21 REPRINTS - GAJDUSEK PUBLICATIONS
Articles by D.C. Gajdusek. Includes _Paediatrics_, vol. 37, 1966,
Supplement.
1966-1969, _26mm_

10/22 REPRINTS - GAJDUSEK PUBLICATIONS
Articles by D.C. Gajdusek.
1970-1973, _28mm_

10/23 REPRINTS - GAJDUSEK PUBLICATIONS
Articles by D.C. Gajdusek.
1973-1975, _30mm_

10/24 REPRINTS - GAJDUSEK PUBLICATIONS
Articles by D.C. Gajdusek.
1976-1978, _28mm_

10/25 GAJDUSEK JOURNAL - CORRESPONDENCE WITH SMADEL
1955-1958, _24mm_

10/26 GAJDUSEK JOURNAL - SAHARA EXPEDITION
1960, _22mm_

10/27 GAJDUSEK JOURNAL - WESTERN CAROLINE ISLANDS
1961, _8mm_

10/28 GAJDUSEK JOURNAL - NEW GUINEA (1)
1961-1962, _23mm_

10/29 GAJDUSEK JOURNAL - NEW GUINEA (2)
1961-1962, _17mm_

10/30 GAJDUSEK JOURNAL - MELANESIA
1963, _13mm_

10/31 GAJDUSEK JOURNAL - WESTERN CAROLINE ISLANDS
10/32 GAJDUSEK JOURNAL - SOVIET UNION, AFRICA, INDONESIA AND NEW GUINEA
1969-1970, _43mm_

10/33 GAJDUSEK JOURNAL - COLOMBIA
1970, _5mm_

10/34 GAJDUSEK - BIBLIOGRAPHY OF KURU
July 1968, _10mm_

10/35 GAJDUSEK - BIBLIOGRAPHY OF KURU
Includes list of Gajdusek journals.
March 1975, _13mm_

10/36 GAJDUSEK - BIBLIOGRAPHY OF CREUTZFELDT-JAKOB DISEASE
April 1979, _10mm_

PART 4 LISTINGS OF RECORDS FROM OTHER COLLECTIONS

10/37 CORRESPONDENCE AND PAPERS HELD BY PAPUA NEW GUINEA INSTITUTE OF MEDICAL RESEARCH - LISTINGS
Copy typescript of listings of papers relevant to FMB, held by PNG Institute of Medical Research. Original listings prepared by R. Ince, 1978. See Appendix for a summary of files.
1978, _3mm_

10/38 CORRESPONDENCE AND PAPERS HELD BY AMERICAN PHILOSOPHICAL SOCIETY
List of correspondence and papers published as _Kuru_ 1956-1967
Correspondence, edited by J. Farquhar. Includes: summary of Burnet correspondence; notes on conversations. Also includes inwards and outwards correspondence with: Gunther; I. Burnet; Anderson; Wood; R. Scragg; D.C. Gajdusek; Rivers; Rees; Berndt; Winton.
12 February 1957 - 6 December 1957, _?mm_ --------------------------------------------------
end of file
Appendix 3

Notes on a rinderpest vaccination trial, Kenya 1945.

The following account of the trial comes from footnotes in the text *Unit 731* by Williams and Wallace (1989).

In 1945, a series of cables was sent between Sir Phillip Ewen Mitchell, Governor and Commander-in-Chief of Kenya Colonies and the Secretary of State for the Colonies, Mr Creasy. At 21.30 hrs on June 8th Mitchell received a cable from Creasy which reads:

"Following Top Secret and Personal from Creasy. Begins. Secretary of State’s Telegram no 361. Smith has already warned Daubney about these experiments in his secret letter of the 22nd March. For your own and Daubney’s information, experiments are connected with possibility that Japanese might attempt to introduce rinderpest into North America by means of balloons. US and Canadian authorities are most anxious that these experiments are being carried out. They particularly ask therefore that connection of work (a) with biological warfare and (b) with North America should be concealed. Suggestion is made in Smiths letter that in order to provide cover the two officers might be described as visitors studying work on diseases or stock being carried out in Kenya. If, however, you think that a better cover could be provided, authorities concerned here would be grateful for any suggestions to this end. No doubt you will be prepared to give laboratory facilities, etc. free of cost; any cost should be charged to Imperial Governments. Ends.  

Three days later on 11th June 1945, Mitchell responds:

FROM KENYA (Sir P Mitchell) TO Secretary of State, Colonies. R (No 414 Top Secret) Your top secret Telegram No 361 New Vaccine for rinderpest Your paragraph 2 noted. Your paragraph 3. Following in Daubney’s advice on the three points. Begins. (1) and (2). Can arrange with twenty animals in each case. (3) Will arrange additional test in paddocks with about forty animals. Field trial with 10,000 animals to follow after arrival of party and discussion with them. Daubney confirms that 10,000 (repeat 10,000) would be suitable number. Ends. 2. Your paragraph 5. Mid-July convenient. Given advance notice of seven days before arrival of vaccine, arrangements for immediate action will be made. 3. Your paragraph 6. Noted."

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1 Direct quotation from Williams and Wallace 1989: Chapter 10 Note 12.
2 Direct quotation from Williams & Wallace 1989: Chapter 10 Note 13
Another cable was sent the same day, which read:

FROM KENYA (Sir P Mitchell) TO Secretary of State, Colonies Received 11th June 1945 16.35 hrs Not numbered. Top Secret Following personal for Creasy. Begins. Your Top Secret and Personal telegram of 9th March. Rinderpest. Noted. Daubney suggests best cover would be US Agricultural, etc. Mission in Abyssinia. If party has own aircraft and came via Addis Ababa cover would be specially good. If not, one at least should go there and both should pretend to be engaged in the interests of the Abyssinian Government for the purpose of conversation with local technical staff and the general public. 2. One risk, since rinderpest is of such interest throughout East Africa, would be curiosity in other territories. I have, accordingly, told the Government of Tanganyika and Uganda and the Chief Secretary of the Governors’ Conference in strict personal secrecy. 3. Needless to say, the fact that the new (repeat new) vaccine is being tried must be kept secret. Object of visit would be to study the applications of existing vaccines to the problem of Abyssinia. New (repeat new) rinderpest vaccine would be headline news from here to Cape Town. Ends.

In October the same year, a further cable was sent from Kenya, which read:

FROM KENYA (Acting Governor) TO Secretary of State, Colonies Received 24th October 1945 09.20 hrs (No 875 Top Secret) Following from Daubney for General [Tommy] Kelser. Six controlled trials and two large field trials satisfactory to date. Leaving Kabets on or before 25th October.

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3 Direct quotation from Williams & Wallace 1989: Chapter 10 Note 14.
4 Direct quotation from Williams & Wallace 1989: Chapter 10 Note 15
Appendix 7A

A flow chart of information related to advice on the safety of medicines.

During the late nineteen eighties in Britain, advice on the safety of medicines flowed in the following direction. Note that the Chief Medical Officer is not included in the process.

NIBSC → CSM-B → CSM → MCA → MINISTER

(Formulated from BSE Inquiry WS 575-Schild)

Key:
NIBSC = National Institute of Biological Standards and Control
CSM-B = Biological Subcommittee of the Committee on the Safety of Medicines
(a Section 4 committee under the Medicines Act 1968).
CSM = Committee on the Safety of Medicines
MCA = Medicines Control Agency (formerly until 1989 Medicines Division)
Appendix 7B

A systemic conflict of interest involved in the process related to the licensing of medicines.

At the BSE Inquiry, Sir Donald Acheson, Chief Medical Officer (CMO), was asked if he was aware that guidelines ‘to be delivered to the pharmaceutical industry’ were being revised at the end of 1988. The CMO responded by saying that he did not think he was aware of this information at that time (Transcript Day 79). He explained that one of the possible reasons might have been because matters relating to medicines were dealt with in a slightly different way from other matters in the Department of Health (see Witness Statement 251 pt. 28 Acheson). The Medicines Division and its advisory Committees such as the Committee on Safety of Medicines under the provisions of the Medicines Act 1968 worked “to the Medicines Commission” due to the ‘conditions of strict commercial confidentiality required under the Act’. This arrangement prevented Dr Gerald Jones, as Medical Head of Medicines Division sharing information with the CMO. Hence, a conflict of interest was inherent within the system in that while the medical staff of Medicines Division were accountable to the CMO in theory, in practice, and ‘quite properly’, according to Acheson, the same staff had ‘also a strong line of accountability to the Medicines Commission’. Acheson suggested that in spite of this situation the medicines issue was dealt with ‘expeditiously’ once the Southwood Committee expressed concerns.

There are three main aspects to consider. One of these relates to actions taken to search for products already in circulation that had incorporated bovine material as an active component. The second aspect relates to actions taken to search for products in circulation that had incorporated bovine material in the substrate as opposed to being an active ingredient. And the third element revolves around products in circulation, or stock versus new products. New products required a license, which would only be issued when the criteria set out in the updated guidelines issued in February 1989 were met. See Appendix 7 c for information about some of the products that came to the attention of the BSE Inquiry.
Appendix 7c

Medicines and vaccines of possible relevance for nvCJD

In response to a letter written to them by Professor Southwood in December 1988, the Committee on the Safety of Medicines noted that:

We originally considered the problem of BSE in the light of the 43 products which our computer database showed to include bovine material as an active ingredient. We will now need to consider the possible hazard from the use of bovine material as an intermediate in the manufacture of products. This will include the use of bovine material in nutrient broths, foetal calf serum... (BSE Inquiry transcript Day 79 emphasis added. Letter dated January 26th 1989).

This points to the issue of substrates. The word now is significant because it indicates that while the matter had started to be addressed, this related only to stocks that used bovine material as an active component. Acheson indicated at the BSE Inquiry that he did not know that the situation had only reached this stage at this time and would have 'been a bit disappointed' had he known about these events at the time. The following month on February 9th he wrote a minute concerning biological product safety in which he stressed that the Southwood Report made no explicit mention of the vaccine problem. Acheson was sufficiently concerned that he asked the Minister to look into the matter with the Medicines Division. The minute concluded by saying, that he would amend the submission to the Secretary of State to the effect that the Southwood Report had not directly addressed the 'question of the safety of vaccines derived from bovine material' and he was 'making urgent enquiries' into the matter (Day 79).

The Department of Health was aware of BSE and its potential scrapie-like qualities by July 1987, when Mr John Sloggem a pharmaceutical officer, contacted Dr D Taylor at the Neuropathogenesis Unit in Edinburgh in July 1987 (Witness Statement, Little). Taylor wrote in response to Sloggem's inquiry on August 24th 1987, in which he 'confirmed a telephone discussion with Mr Sloggem about a disease newly reported in bovines which was “scrapie-like” (Little WS 331B. Referring to para 31 of Sloggem WS No 454).

A meeting was held between Dr Watson and Drs Dickenson, Kimberlin and Hope at the NPU [Neuropathogenesis Unit] on September 14th 1987, where a treatment with lecithin for neurologically brain injured patients was discussed (Little WS 331B). On September 16th Mr Bradley, the person responsible for liaising with the Department of Health, wrote in a memo “Medicines Committee Members DHSS asked Dickenson re danger. **** (unclear) BSE Treat bov brain lecithin? Oral mental patient” (Little WS 331B).

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1 Mr Bradley was appointed by Dr Watson as ‘CVL BSE R&D co-ordinator in June 1987’ (see Little statement). One of the ‘principal tasks’ of Mr Bradley, according to Dr Little, ‘was liaison on an almost daily basis with Government policy makers’. 
The question of risk relative to this product needs to be considered. While the oral route is considered to be less effective than an injectable medicine, when taken orally a treatment can be as risky as an injectable product if there is broken mucosa in the mouth and particularly, as in this case, if the product is manufactured from bovine brain.

Towards the end of 1988, when the survey was conducted to ascertain how widespread the problem of contaminated products might have been, one document, written in September 1988 discussed at the inquiry, indicates that Drs Rotblat and Purves, both of whom worked in Medicine’s Division at the time, wrote that

“The computer list shows 33 product licences extant for preparations of bovine origin and of these 42 are for insulin” (Transcript Day 79).

The document indicated that there were “no licenced products derived from bovine brain”.

Attention was drawn at the inquiry to what was described as a ‘rather odd’ document, which was also written in September 1988. This was an ‘extract from the MCA [Medicines Control Agency formerly Medicines Division] questionnaire summary’ which indicated that at least one product did contain calf brain. The company name is given as 01234 for reasons of confidentiality, and the drug is referred to as drug X (Day 79). It would appear that for some reason Rotblat and Purves were not able to record this drug in their report written the same month. It is not clear to which drug this refers. It might refer to the oral lecithin product mentioned above, although it might also refer to a pituitary hormone or other product.

By September 1988, manufacturers had begun to be notified of a potential problem and had begun sourcing their bovine material from places other than the UK. By March 1989 over 4000 letters had been sent to manufacturers (see Transcript Day 79), and guidelines had been issued jointly by the Committee on Safety of Medicines and the Veterinary Products Committee. The guidelines, worded as a “purely precautionary” measure, had the potential to minimise the importance of the actions that needed to be taken.

In July 1992 all vaccines in the United Kingdom complied with Joint CSM/VPC guidelines (WS 422-Rotblat pt 41), but questions have been raised at the BSE Inquiry regarding the safety of some medical products produced prior to this time. This applies to both veterinary and human vaccines.

I trace here some of the deliberations in order to demonstrate the concern expressed about safety of particular products.

On July 4th 1990 Dr Rotblat attended a meeting of the BSE Working Group, for which she had prepared a paper on the ‘current situation with regard to vaccine stocks for the meeting’ (WS 422 pt 42). The paper included information about ‘two individual companies’, their progress in relation to the manufacturing of new batches and the ‘extent of their vaccine stocks’. Also discussed at the meeting, was the state of responses from the questionnaire sent out previously to pharmaceutical
manufacturers; there were four ‘non-responders’, none of which held a ‘full product licence’. The Ministry of Agriculture Food and Fisheries (MAFF) informed the meeting that ‘most companies had changed the source of bovine material and were obtaining it from BSE-free areas for veterinary medicines’; ‘there were still stocks held of products that were associated with UK bovine material’ (WS 422 pt 43).

The first product mentioned at the meeting was a mixed measles, mumps and rubella vaccine commonly known as MMR. Rotblat stated that

> It was understood that the company which manufactured the first product (an MMR vaccine) under consideration had changed over to a New Zealand source of bovine material (WS 422 pt 44).

A decision was taken that ‘existing trial batches prepared with UK bovine material should not be used’ (WS 422 pt 44). This information needs to be contextualised within the MMR campaign from mid 1988 to October, at which time the British government was preparing for the campaign. It is unclear when this trial product was made, but if it was made after the campaign in 1988 this suggests that there was a reason to produce a new product, which indicates that there was concern about the older product. On the other hand, the trial product may have been the same one as used in the 1988 campaign. Whatever the case, at least one manufacturer of MMR vaccine in mid 1990 had trial products which were not to be used because of the risk of possible contamination with BSE, a scrapie-like agent (or one of its mutations).

Note: The transcript of Lister, Cunningham & Bridges (Day 50) indicates that the ‘rabies model’ was being used by 1994 rather than the scrapie model to understand how the agent manifested in the nervous system.

The second product discussed at the BSE Working Group meeting was a PPD vaccine. This product related to only one manufacturing company (WS 422 pt 45). PPD stands for purified protein derivative and is a constituent of the Mantoux test used to test for ‘cellular immune responses to *Mycobacterium bovis* (Tuberculin bacillus), incurred either, naturally or via BCG vaccine (Immunisation Procedure NHMRC 1991:110). Regarding the PPD product, the BSE Working Group decided

> ‘that the use of glycerol-beef broth was low on the list of potential infectivity and, since there were no alternative sources of the vaccine, the hazard from having no stocks available outweighed the potential risks from use’ (WS 422 pt 45).

The company was ‘changing to using peptone broth as quickly as possible’.

The third product discussed at the meeting was a measles vaccine. The company involved was changing to New Zealand sourcing. Any existing stocks were expected to be ‘deleted by September 1990’ (WS 422 pt 46).

The fourth product was a DTP vaccine. The manufacturer was a ‘majority supplier’. A decision was made at the meeting to speak with the manufacturer in order ‘to discuss its future plans’ and ‘the time by which all bovine components in the
manufacture of the vaccines would comply with the guidelines’ (WS 422 pt 47). DTP refers to a combined Diphtheria, Tetanus and Pertussis (whooping cough) vaccine.

In Australia, DTP in 1991 was administered at 2 months, 4 months, 6 months of age, and again at 18 months of age. (MMR is administered at 12 months of age) (Immunisation Procedure NHMRC 1991:22). This is a guide to the times of administration in the UK.

In September 1990, Rotblat was asked again to prepare a paper on existing stocks for the next BSE Working Group meeting (WS 422 pt 49). On September 11th the view was expressed that care needed to be taken in ‘selecting sources of bovine material for use in pharmaceutical products’ (Minute from Mr Meldrum to Mr Lowson) (WS 422 pt 50). This information had been under consideration for some time by the BSE Working Group, the Medicines Control Agency and its advisory committees. Rotblat stated that she had not seen the minute prior to recent times (late 1990s). The minute appears to indicate that either, the Veterinary division was tardy in getting this message out, or that they were again reminding others in the Department of Health of the potential problem. Either way, by the end of 1990 there was still a lot of concern about medicinal products and vaccines, and some products were still in use.

Rotblat noted that the measles vaccine (first product listed) was not ‘used much’. The second product is not a vaccine but a product used to test ‘whether it was necessary to use a vaccine’. This probably refers to a Mantoux. The product was used on teenagers rather than babies. The third product, the MMR vaccine, was never developed and ‘no product licence was ever issued’ for it. Nevertheless, it is curious why a trial vaccine was being developed in 1990 if the MMR vaccine given during the 1988 campaign was satisfactory. The final product was an ‘unabsorbed DTP vaccine’, ‘rarely used’, and only in adults (WS 422 pt 31).

According to Dr G. Schild, Director of NIBSC, bovine serum used at the time in vaccines and other medicinal products was then, and still is today, considered a low risk product (WS 575 –Schild pt 52). Schild remarks on the time required to phase out one vaccine and develop a replacement that meet revised guidelines:

The time needed in order to phase out UK sourced bovine materials completely from such products would be substantial. An unchanged and standardised manufacturing process is the key to ensuring a consistently high quality product; a change from bovine serum could potentially have had a significant effect on the quality and efficacy of a vaccine. Any such change could require research and development work and clinical studies to confirm the quality of the product (WS 575 pt 52).

Even a switch to the use of bovine materials sourced outside the UK could not have been made “overnight” without major disruption of the availability of vaccines. Typically, manufacturers make seed and bulk vaccine preparations which remain potentially useable for many years. Replacing vaccine stocks using a new seed which had not been in contact with any UK bovine materials would take time and effort on the part of manufacturers and was, in any event, undertaken expeditiously (pt 53).
Time was a critical factor. This does not deny the amount of time it took to actually contact manufacturers and for responses to be received. It is a matter of conjecture whether one accepts that the changeover was carried out expeditiously. Perhaps in terms of the processes described above, the changeover was expeditious. In terms of the humans who are subjected to the products, the time taken may not seem quick enough. As I began by saying, it was not until 1992 that all vaccines in the UK complied with Joint guidelines set down by the Committee on the Safety of Medicines and the Veterinary Products Committee in 1989.

The Director of NIBSC was a member of the BSE Working Party, which met five times between September 1st 1989 and July 8th 1992, but recalls attending only the first meeting and two of the subsequent meetings. He cannot recall why he missed the others, although his involvement with the Medical Research Council AIDS Research Programme could have prevented his attendance on the other occasions. He could 'remember nothing about the details of the meetings themselves’. Having considered the minutes, he could remember the balancing act it was necessary to perform between 'the risks posed by BSE and the benefits of the use of bovine material in medicines and vaccines' (WS 575 –Schild pts 50 and 51).

Pertaining to veterinary medicine.

Dr James Rutter, the Director of Veterinary Medicines and Chief Executive of the Veterinary Medicines Directorate, noted in his statement that:

Prior to the identification of BSE, Directive 81/851/EEC ..., which came into force in November 1983, required its provisions to be applied progressively, within 10 years, to veterinary pharmaceutical products already on the market in member states (WS 1999 pt. 20 Rutter).

In 1988 products in the hormone group were called up for review which required the licence holders to submit updated dossiers to the licensing authority. From this review it transpired that four products in the pituitary hormone group that may have contained bovine material were on the market. These products were not defended and the four licences had expired by April 1991 (pt. 20).

Questions about the safety of these four veterinary products in the pituitary hormone group remain along with the products outlined above, which were administered to humans.
Reference list


Arens, W. (1997) ‘Man is off the menu’ The Times Higher Education Supplement December 12th Perspective Section, p. 16


Boffey, P. M. (1976) ‘Vaccine imbroglio: The rise and fall of a scientists-critic’ Science December 3 pp. 1021-1025


Breinl, A. (1918b) ‘Clinical, pathological and experimental observations on the “mysterious disease”, a clinically aberrant form of acute poliomyelitis’ Medical Journal of Australia Volume 1 No 12 pp. 229-234


Brown, P., Preece, M., Brandel, J. P., Sato, T., McShane, L., Zerr, I., Fletcher, A.,
Will, R.G., Pocchiari, M., Cashman, N. R., d’Aignaux, J. H., Cervenáková, L.,
disease at the millenium’ Neurology Vol 55 No 8 pp. 1075-1981

Burger, D. & Hartsough, G. R. (1965) ‘Transmissible encephalopathy in mink’ in
Virus Infections, NINDB Monograph No. 2, U.S. Department of Health, Education,
and Welfare. Public Health Service, National Institute of Health, National Institute of
Neurological Diseases and Blindness. pp. 297-300

Volume 2 No 8 pp. 157-161

April 6th pp. 278-289

Burnet, F. M. (1934) ‘Louping ill virus as a possible cause of the X disease epidemics
of 1917-1918’ Medical Journal of Australia May 26, pp. 679-681

December, pp. 1519-1521


Burnet, F. M. (1968) Changing Patterns: An Atypical Autobiography. Melbourne,
London: William Heinemann

Caughey, B. (1997) ‘Chaperone-supervised conversion of prion protein to its
protease-resistant form’ Proceedings of the National Academy of Sciences December,
Volume 94 pp. 13938-13943

Cleland, J. B. & Bradley, B. (1917) ‘The mysterious disease’ Medical Journal of
Australia Volume 1 p. 499

House

Critchley, M. & Greenfield, J. G. (1937) ‘Jakob’s Syndrome (Senile Dementia with
Parkinsonism)’ Proceedings of the Royal Society of Medicine Vol 30 No 2 pp. 1100-
1101


Cruickshank, R. (1971) ‘Factors to consider in the justification of vaccination
programs’ in WHO Proceedings International Conference on the Application of
Vaccines Against, Viral, Rickettsial, and Bacterial Diseases of Man 14-18 December


Eddy, B. E., Stewart, S. E., Young, R. & Burroughs Mider, G. (1958) ‘Neoplasms in Hamsters induced by Mouse Tumour Agent passed in tissue culture’, *Journal of the National Cancer Institute*, Vol 20, No 4, April pp. 747-761


Ford, B. J. (1991) ‘Bovine Spongiform Encephalopathy’ a meeting held at the Linnean Society, Burlington House, Piccadilly on April 29th

http://www.sciences.demon.co.uk/wbsemtg1.htm 01/01/00 12.25


238


Gajdusek, D. C. (1953) *Acute Infectious Hemorrhagic Fevers and Mycotoxicoses in the Union of Soviet Socialist Republics*, Gajdusek, D. C., Captain M.C., US Army, Department of Virus and Rickettsial Diseases, Army Medical Services Graduate School, Washington, DC. May 1953


Gajdusek D. C. (1996) *Journal 1955-1957 Australia and New Guinea, Virology to autoimmunology, ethnopediatrics to kuru, enchantment by melanesians, politics to science (in two volumes)*. Laboratory of Central Nervous System Studies, National Institute of Health, Bethesda, Maryland, USA


Goodwin, B. (1998) Keen as Mustard: Britain's Horrific Chemical Warfare Experiments in Australia, Box 42, St. Lucia, Queensland, Australia: University of Queensland Press


Gunther, J. T. (1977) *Australia, Kuru and a Nobel Prize* (manuscript): a draft of a paper. ABN database 91-354227


Harris, S. (1995) ‘The art of scientific inference lies in deciding how many coincidences it is wise to accept’ *Skeptics* Vol 3 No 2 p. 72


Higgins, C. M. (1920) *Horrors of Vaccination Exposed and Illustrated: Petition to the President to Abolish Compulsory Vaccination in Army and Navy*. Brooklyn, New York: Chas. M. Higgins


Klatzo, I., Gajdusek, D. C. & Zigas, V. (1959) ‘Pathology of kuru’ Laboratory Investigation Volume 8, No 4, pp.799-847


Kuru: A Clinical Study (1958) a film made for the Department of Health, Territories of New Guinea by the Commonwealth Government Film Unit, Department of Information, Australia, MCMLV111. Held at the National Sound and Film Archive, Canberra. ABD000082
Lagan, B. (2001) ‘Cosmetics may be next on beef hit list’ Sydney Morning Herald January 8th


McGowan, J. P. (1925) ‘A further contribution to the subject of scrapie’ Scottish Journal of Agriculture Volume 8 No 2 pp. 190-195


Mellor, D. P. (1958) The Role of Science and Industry, Canberra: Australian War Memorial

Metherell, M. (2000) ‘Mad Cow link with vaccines’ SMH News Section November 2nd p.4


Monkey Business (1998) ‘Four Corners’ Television documentary, ABC TV, April 13th


‘Scientific’ (1918) *Medical Journal of Australia* Volume 1 April 6th pp. 292-294


Special Article (1951) ‘Murray Valley Encephalitis’ *Medical Journal of Australia* April 7 pp. 526-527


*Sun Herald* (1957) ‘Laughing Death’ October 6th
**Sun Herald** (1959) ‘More Die Laughing: Fresh Survey’ October 11th

**Sydney Morning Herald** (1959) ‘Laughing Death Haunts Tribe’ by Gavin Souter September 30th

**Sydney Morning Herald** (1959) ‘U. S. Honour for N. G. Doctor’, September 18th
A. A. P. – Reuter, Lae

**Sydney Morning Herald** (1960) ‘“Concentration Camp”: Attempts to Combat N. G. Disease’ May 23rd p 10, A. A. P. – Reuter, Port Moresby


ARCHIVAL MATERIAL

Papers of Frank MacFarlane Burnet held at the University of Melbourne Archives and collated as part of the Australian Science Archives Project (ASAP), now known as Australian Science and Technology Heritage Centre (AUSTEHC). Series 8. Series 9/35. Series 10. Series 12

Papers of Sir MacFarlane Burnet held at the Adolph Basser Library, Canberra, Australia.

‘Introduction to Kuala Lumpur talk on Kuru’ (1970) Ref 98/2/c8
‘WEHI FMB Office Diary 1961’ Ref 98/4/c
‘WEHI FMB Office Diary 1962’ Ref 98/4/c
Papers relating to allegations of germ warfare in 1952 Ref 98/2a/6
Papers of Dr John Gunther – Kuru File – held at the Australian National University with Professor Hank Nelson

Patrol Officer Reports: Eastern Highlands section of Box ‘Patrol Officer Reports 1943-1976’, Australian National University Menzies Library, Canberra

Jack Baker, Kainantu No 7, 1956-57. East Highland Sub-Division: microfilm 24 of 102
John Colman, Kainantu No 14, 1954-55. Kainantu sub-division of South-Fore Census area: microfilm 18 of 102
John Colman, Kainantu No 5 1956-57. South-Fore area: microfilm 23 of 102
John MacArthur, October 1954-55. microfilm 8 of 102

BSE INQUIRY Transcripts of evidence and witness statements:
WWW.BSE.ORG.UK
Acheson Witness Statement No 251 - Chief Medical Officer, British Government
Armour Witness Statement No 477 - Chair, Veterinary Products Committee
Asscher Witness Statement No 441 - Chair, Committee on Safety of Medicines
Jefferys Witness Statement No 419E - New Drugs Branch, Medicines Division, DoH
Little Witness Statement No 331A, 331B - Deputy Director, Central Veterinary Laboratory, (Ministry of Agriculture Food & Fisheries)
Minor Witness Statement No 576 - NIBSC Head of Virology
Pickles Witness Statement No 115 - Scientific Secretary, Department of Health
Rotblat Witness Statement No 422 - Medicines Division
Rutter Witness Statement No 499 - Director, Veterinary Medicines
Schild Witness Statement No 575 - NIBSC Director
Will Statement of information No 61c

Transcript Day 79 - Acheson, CMO
Transcript Day 84 - Currie, Deputy Minister of Health
Transcript Day 87 - Clarke & Freeman, Secretary of State & Minister for Health
Transcript Day of proceedings 13/12/99 Jones, G - Medicines Division D of H
Transcript Day 85 - Newton & Moore, Min. for Health & Secretary of State
Transcript Day 1 - Southwood, Chair of First BSE Working Group
Transcript December 16th 1999 – Will, Neuropathogenesis Unit, Edinburgh