I was selected to be trained as an aid post orderly

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The administrator of the district, Mr John Colman, first employed me as a translator assisting the late Mr Muriso Warebu. I assisted him and in particular worked with the Gimi people and the people of Moraei. Then I was selected to be trained as an aid post orderly. After completing the course, I was sent back to Purosa to set up an aid post. With the help of the village people, the building was ready in less than two weeks.

During my working days, I visited and cared for more patients with kuru than with any other disease. I also assisted Dr Carleton Gajdusek in performing autopsies and stitching up the body and handing it back to the family for burial. Kuru had devastating social effects: I can remember one hamlet in the Purosa Valley where at one stage there were only five women with many more men and children.

Carleton was based in Agakamatasa village and I went out to help and treat patients in that area too. I also worked with Michael Alpers, Dr Hornabrook, John Mathews, Dick Sorenson and Paul Brown. I am particularly pleased that some of those I worked with in the past have been able to attend this meeting.

The medical scientists who went into Okapa District from abroad played a vital role in reducing the death rate of kuru disease. They helped to change the lifestyle of our people. We have lost many of our family members from kuru, which swept through entire villages taking innocent lives. However, we have recovered and today the population of the Purosa Valley is nearly 4000. Yet I still have one unanswered question: has kuru disease gone for good or will it return in the next generation?

The late 1970s: a lull in the action on kuru

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The late 1970s were a relatively quiescent period in kuru history. The disease's clinical course (Gajdusek & Zigas 1957; Zigas & Gajdusek 1957; Alpers 1964; Alpers & Rail 1971; Hornabrook 1975), geographical epidemiology (Alpers 1965, 1979) and dissemination by endocannibalism (Mathews et al. 1968) had been well described in the prior two decades. Seminal animal experiments had demonstrated the transmissibility of long-incubating neurological illness (Gajdusek et al. 1966; Gajdusek 1977). Whole cohorts of Fore children born after certain infection-spreading mortuary practices were abandoned in the late 1950s would, for the first time in possibly a century, survive to adulthood without dying of kuru (Hornabrook & King 1975; King 1975). Most tellingly, in each successive year, the median ages of new patients increased by approximately 1 year, a correlation consistent with interdicted transmission. The elusive infective entity appeared less likely to contain DNA or RNA even as molecular genetic techniques for finding nucleic acid improved (Gajdusek 1977; Manuelidis & Manuelidis 1979), but because the Fore no longer faced extermination by kuru, and because community-acquired spongiform encephalopathies were not emerging in other populations except for rare iatrogenic transmissions of Creutzfeldt–Jakob disease, investigative nihilism set into the field of ‘slow agent’ research.

I had an opportunity as a medical student to perform kuru fieldwork for five months in 1979 under the direction of Michael Alpers. I was charged with finding every person living with confirmed or putative kuru in the Eastern Highlands, using Alpers’ guidelines (Alpers 1964). Field assistants Anua Senavaiyo, Auyana

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Winagaiya and Igana Alesagu from the Papua New Guinea (PNG) Institute of Medical Research (IMR) in Goroka took me to each of the 23 kuru patients known to them in that interval. Our goal was to confirm or refute the epidemiological model that the kuru agent had been transmitted to children of both genders, and to girls and women older than approximately 7 years of age, probably owing to age–gender segregation at mortuary gatherings (figure 1). Each of these 23 patients was born before 1954, again confirming the transmission model. I also refined several family histories of intergenerational kuru, analysed childhood infection cohorts and identified an apparently longer incubation period in boys than in girls (Tarr 1980).

However, my chief effort was to interview the relatives and acquaintances of kuru decedents in the ‘Book’. The Book was the voluminous hard copy line listing of all kuru cases by village from 1957 onwards. Its major contributors in the early years were Patrol Officer Jack Baker (a particularly important contributor), Carleton Gajdusek, Vin Zigas and Alpers, who, in 1970, computerized these data at the NIH. Other contributors included Alex Nilsson, John Mathews, Richard Hornabrook and others from the IMR, kiaps (patrol officers), and many local and visiting doctors, students and missionaries. Steve Ono and Judy Farquhar entered the data from the field into the NIH mainframe computer and generated updated paper copies periodically (figure 2). This systematic collection of the estimated year of birth, symptom onset, death or ‘recovery’ of every definite, putative or refuted case of kuru (Alpers & Gajdusek 1965) represents ‘shoe leather’ epidemiology in every sense of the term. These data are as unusual as they are important. I know of no other epidemic where every case has been meticulously documented over a half century, or any disease disappearance so thoroughly chronicled. The MRC, to its credit, has supported continued kuru surveillance, analysis and data archiving.

In the late 1970s, the IMR had limited resources to address the many medical disorders in the newly independent PNG. Michael Alpers, who succeeded Richard Hornabrook as IMR Director in 1977, faced these challenges by seeking new funding, expanding malaria, filariasis and nutrition research, founding IMR branches in the Madang and Southern Highlands Provinces, and supporting extensions of Ian Riley’s groundbreaking work on lower respiratory tract infection in Huli children and adults in Tari (Riley et al. 1977, 1981; Riley 2002). However, Alpers was one of Gajdusek’s original collaborators in Okapa and Bethesda, and his family had maintained part-time residence in Waisa throughout the 1960s and 1970s. Alpers therefore knew the value of kuru surveillance, and how to do it. Most importantly, he recognized that without shouldering personal responsibility for the arduous and underfunded kuru fieldwork, a
scientifically precious and unique data collection would meet an untimely end. Michael’s telephone call to me in Seattle in 1985 from Goroka (where it was 1 o’clock in the morning at the time) requesting clarification on some field notes I had taken 6 years earlier typified his stewardship of these efforts. 

Post-1970s kuru science, well described in this issue of the Transactions, is highlighted by the perseverance of Stan Prusiner in his refinement of small animal models of scrapie (Baringer & Prusiner 1978; Westaway et al. 1987), meticulous chemical methodology (Prusiner 1982; Bockman et al. 1985) and identification of PrP (Basler et al. 1986), and the thorough correlations of John Collinge and his group (Mead et al. 2003; Wadsworth et al. 2004) between host genotype and prion disease expression, thereby explaining the epidemiology of kuru and other human transmissible encephalopathies. However, the people of the Eastern Highlands remain the underappreciated heroes of this saga. Even though not a single one would plausibly benefit from this research, patients and their relatives unfailingly cooperated with kuru investigators for over a half century. Had the people of the kuru-affected region been less tolerant of the repeated and no doubt baffling studies by outsiders, the concept of transmissible, long-incubating, human encephalopathies would have been accepted tardily, if at all, by clinicians, scientists, governments and industry. Such a delay would have further stalled recognition that food of bovine origin could transmit variant Creutzfeldt–Jakob disease (vCJD), and magnified the 1990s vCJD epidemic, since so many outsiders, the concept of transmissible, long-incubating, human encephalopathies would have been accepted tardily, if at all, by clinicians, scientists, governments and industry. Such a delay would have further stalled recognition that food of bovine origin could transmit variant Creutzfeldt–Jakob disease (vCJD), and magnified the 1990s vCJD epidemic, since so many people worldwide.

Kuru is, or will soon be, extinct (Alpers & Kuru Surveillance Team 2005). The South Fore and their neighbours in the Eastern Highlands remain and prolonged the 1990s vCJD epidemic, since so many others, the concept of transmissible, long-incubating, human encephalopathies would have been accepted tardily, if at all, by clinicians, scientists, governments and industry. Such a delay would have further stalled recognition that food of bovine origin could transmit variant Creutzfeldt–Jakob disease (vCJD), and magnified the 1990s vCJD epidemic, since so many people worldwide.

Kuru, is, or will soon be, extinct (Alpers & Kuru Surveillance Team 2005). The South Fore and their neighbours are not. This story has taught us much about scientific inquiry, and about citizenship.

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I was less than 10 years old when Dr Carleton Gajdusek came into the Purosa Valley to carry out his research on kuru disease. Michael Alpers came after him and I worked alongside them to patrol out into the villages that were affected by kuru. My job was mainly to accompany Carleton and assist in collecting kuru patients’ samples and diagnosing their disease. I also worked as a kuru surveillance officer and went on my own into the villages to locate kuru patients. Sometimes, I was given the task by Carleton of transporting brain tissue and other selected internal organs to Kainantu station by tractor or Landrover.

The people’s belief system was that kuru was caused by sorcery as a means of payback by other clan members within the village or a nearby village. Collecting human samples was very hard owing to the fear of sorcery: the people feared that we might misplace some of the samples and sorcerers might pick them up. Nevertheless, those of us who worked with western medical scientists were free to move around in the villages to assist kuru patients and their families.

The others that I worked with in the field include John Mathews, John Colman and Jack Baker. It is great to meet again some of those colleagues I once worked with in the South Fore area of Okapa District.

I have conducted fieldwork on kuru, with colleagues from the MRC Prion Unit and the Papua New Guinea Institute of Medical Research (PNGIMR), since 1996. During this time, the fieldwork has been dependent on the support of the PNGIMR, and I thank the staff and the past and present Directors, Michael Alpers, John Reeder and Peter Siba, for their support. Michael’s vast knowledge and experience of Papua New Guinea has been invaluable and inspirational. I also thank my Papua New Guinean friends whose counsel has guided me through the occasional turbulent time. In particular, I thank Dr Inoni Betuela, Dr Ken Boone and his wife Lisa, Samson Akunaii, Anderson Puwa and Henry Pako. The members of the Kuru Project, past and present, have made a significant contribution to the success of the Project, and though I will not name them...