DISEASE AND DESCENT

An Anthropological Examination of the Bird Disease, Groote Eylandt, Northern Territory

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INTRODUCTION

Groote Eylandt is the largest island in an archipelago off the mid-east coast of Arnhem Land. The people of Groote Eylandt and neighbouring Bickerton island are a semi-isolated group sharing language and marriage laws. Their predecessors were socially linked with the Nunggubuyu people on the south side of Blue Mud Bay, on the mainland.

The history of the people of Groote Eylandt is peppered with episodes of cultural contact. In 1623 the Dutch explorers Carstensz and Van Colster passed through the area, and gave Groote Eylandt its name. (It means 'big island' in Dutch). The island has an area of 2260 sq.km.). At the same time, Macassans were coming to Groote Eylandt to collect sea-slugs (beche-de-mer, trepang) for world trade. This contact appears to have existed for about three hundred years (Worsley 1954:3) and took some of the Aboriginals from Groote Eylandt to Macassar (including Sulawesi and parts of Malaysia) and back, and gave others of them Macassan ancestors. It was not until 1921 that prolonged contact with white Australians began, when Anglican missionaries from the Roper river mission set up a home for half-caste Aboriginal children at Emerald river on the west side of the island.

From the early days of white contact on Groote Eylandt there appear to have been two distinct phases that

had substantial effects upon social life, missionisation in the 1940s, and the mining of manganese (in which the west side of the island is rich) in the 1960s. Now, a problem is occurring which may have its origins in both these events.

Since the late 1940s, when the first case is believed to have emerged, a mysterious disease has been affecting people on Groote Eylandt. It appears in two different forms; one characterised by congenital ligament laxity and muscle weakness, the other by a progressive neuro-muscular degeneration and ataxia occurring from the third or fourth decade of life. It has been referred to variously as the 'Bird Disease', the 'Manganese Nightmare' and the 'Groote Eylandt Syndrome'. Henceforth in this paper I shall call it the 'Bird Disease' as it seems to have been the name that Aboriginal people chose themselves; also, unlike the name 'Manganese Nightmare' it does not presuppose a cause. Altogether, seventeen people have suffered from the disease, three of whom have died of it. The disease thus has a prevalence rate in the Aboriginal population of Groote Eylandt of 1-2 percent. The total Aboriginal population of Groote Eylandt is approximately one thousand, three hundred at Umbakumba and seven hundred at Angurugu. Cole (1988) estimated that more than half the population at Angurugu in 1987 was under the age of sixteen. All the sufferers belong to two of the thirteen clans resident on the Island.

There are two primary Western medical explanations. One concerns poisoning by manganese, the other concerns genetic inheritance. Likewise there are two main indigenous explanations; one being poisoning by manganese dust, the other being centred on marriages that may have

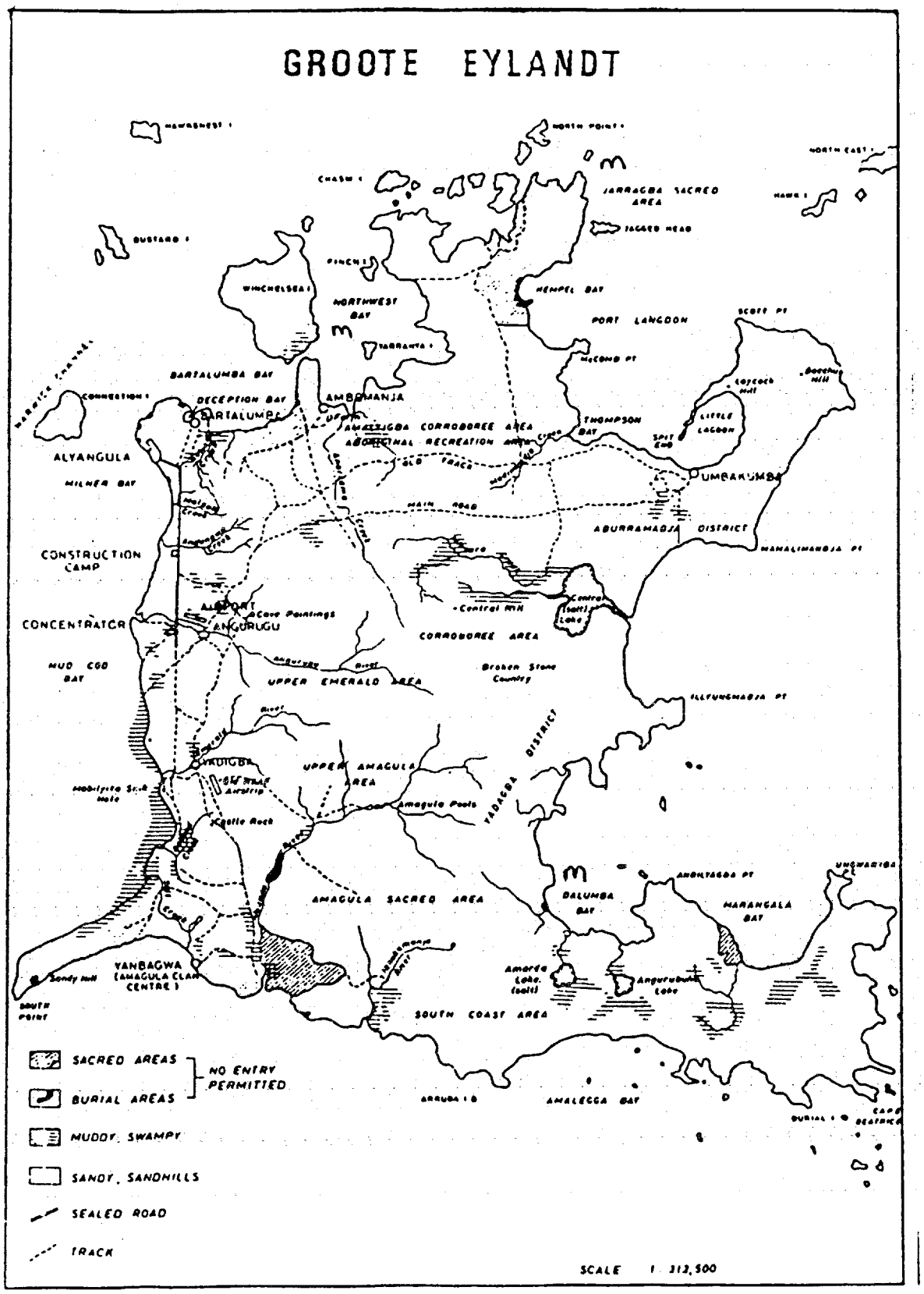
been considered 'too close'. Other explanations concerning cursing (Cawte 1984) have been put forward, as have those of sorcery or 'spiritual contamination' (Turner 1989:252-3). In this thesis I shall examine the evidence and propose a theory of the cause of the disease. This is not unprecedented- other researchers have also been trying to find a cause; however, their approaches from a Western framework of biomedical interest. My approach will entail a short discussion of medical exploration and examination of the case, but will primarily attempt to set that with a wider social framework, as I believe that it is within the social background that many of the causes are embedded. I shall also argue that it is important to regard the concerns of the sufferers, who experience illness in terms of symptoms and their effect on normal life, and have personal beliefs regarding the cause of the disease.

The first chapter will set the social background from which the disease has emerged. The ethnographies of the island will be introduced and their findings discussed in relation to preconditions for disease. The issue of culture contact and change will be raised.

In the second chapter I shall present the medical details of the disease such as signs and symptoms, nutritional status and investigative tests, and discuss the medical theories of its cause, such as genetic inheritance, malnutrition, 'slow virus' and manganese poisoning.

The third chapter will be an analysis of the data presented in chapters one and two, referring to theories put forward by medical anthropologists such as Capra, Engel, Torrey and Kleinman. I will present my conclusion as to the

cause or causes of the bird disease and through that, make some predictions about the future of the syndrome, as a form of future evaluation of the theory. Some pragmatic suggestions will be made with the aim of reducing some of the predisposing factors.



Printshop, Angurugu m = areas visited by Macassans

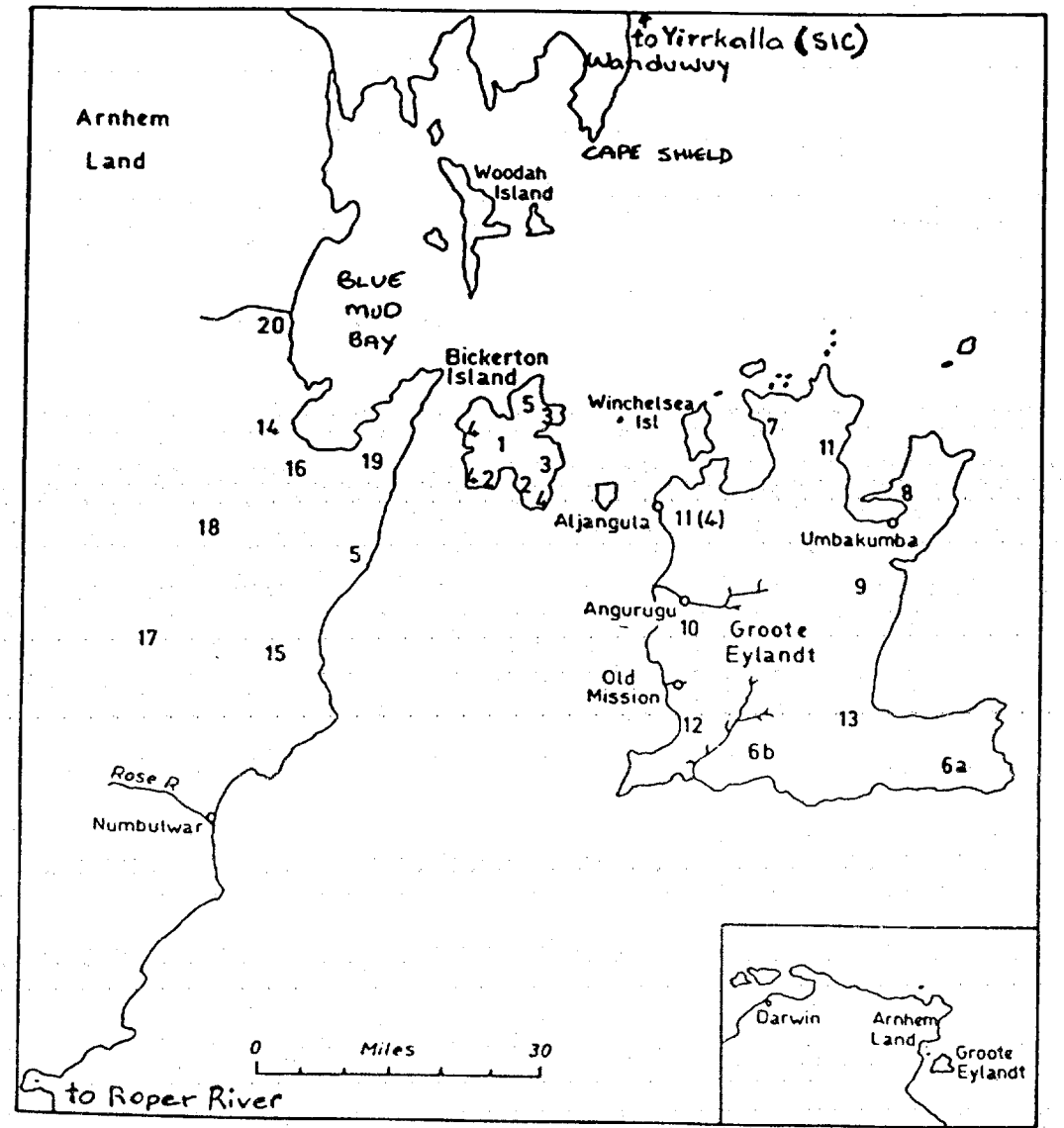


Figure 2 Map of Central-Eastern Arnhem Land showing territories of local groups in the area.

1. Wanungaunggeragba
2. Wuraliljanga
3. Wuranggiljangba
4. Wanungwadarbalangwa
5. Wanungamadada
6. Wanindiljaugwa
   (a) Wanindiljaugwa
   (b) Wanungamagula
7. Wanungwamalangwa
8. Wanungamagadjeragba
9. Wanungawurugwerigba
10. Wurramaminjamandja
11. Wanugwudjaragba
12. Wanungawurigba
13. Wanungmurungulja
14. Wanungargala (Minjawan)
15. Nunggumadjbar
16. Wanindilibala
17. Nemamurdudi
18. Nalmi
19. Murungun
20. Mangamangaraja (Wurramangar)

LOCATION MAP
(Turner 1974:7)

CHAPTER 1

ETHNOGRAPHIC EXPLORATION

In this chapter I shall present the ethnographic background of Groote Eylandt. Social conditions will be discussed, especially in terms of culture contact and change. It is possible that some of the preconditions of illness lie in the social background, or more specifically in the context of the social changes of the last fifty years.

Four ethnographies of Groote Eylandt have been written since Europeans' first contact with Aborigines on Groote Eylandt. The first was actually written at the time of contact in 1921, by N. Tindale, an entomologist of the South Australian Museum, and published in 1925. Later ethnographies are by F.G.G.Rose (Classification of Kin, Age Structure and Marriage amongst the Groote Eylandt Aborigines) published in 1960, P.M.Worsley (The Changing Social Structure of the Wanindiljaugwa) in 1954, and D.Turner (Tradition and Transformation) in 1974.

CULTURE CONTACT AND CHANGE

So far I have referred to three phases of history and social change on Groote Eylandt; briefly, Macassan visits for trade in beche-de-mer, missionisation, and the mining

of large amounts of high quality manganese by GEMCo at Angurugu.

MACASSAN VISITS

The Macassans, though their visits were intermittent, exploited the conception of payment for work (Worsley 1954:11), mostly in the form of goods such as pipes, gin, tobacco and metal implements. Less pleasantly they also, according to Worsley, punished by whipping those who they considered did not work fast enough. This work involved the collection and drying of beche-de-mer, which were ultimately sold by the British to China in return for tea and silk. In this way, Groote Eylandt Aborigines were marginally involved in world trade before the British even knew of their existence.

The Macassans did not, it appears, consciously set out to change facets of Groote Eylandt social life as Europeans did later. By and large, the two cultures worked together when the Macassans arrived, and basic conditions continued much as they had ever been. These visits only occurred at intervals in certain places (see map, p.v) and in particular seasons. They usually arrived with the north-west (monsoon) wind at the start of the wet season and departed with the south-east wind at the start of the dry season. Certain cooking implements and metal for spears were adopted by the Groote Eylandt Aborigines, and sails were made for canoes. Thus adoption of new technology and an interaction with an economic framework were the major

Sources ?

observable effects of Macassan contact. Another adaptation to Macassan contact may have been the strict seclusion of women. Tindale noted in 1925 in relation to the Macassan visits that:

> The Malays whenever possible took possession of native women and took them away on their homeward journeys. The Ingura native thus learned that the women should never be seen. (Tindale 1925:131)

On the whole, Groote Eylandt Aborigines apparently maintained their cultural integrity throughout Macassan interaction. According to Tindale, this protracted period of labour would not have disturbed normal migratory habits unduly as he says

> During the wet season, when food is plentiful and travel uncomfortable owing to the moist heat and cold rain, huts are built and occupied for several weeks or months at a time (1925:113).

The Macassans had to stop visiting Groote Eylandt after Australian Federation when foreign trade in the area was discouraged and Australian enterprise preferred (Worsley, 1954:18).

EUROPEAN CONTACT

When Groote Eylandt was first visited by white missionaries in 1916, missionisation of Groote Eylandt Aborigines was not their primary aim. The intention was, in fact, to find a suitable site for a mission to which 'half-caste' Aborigines from the mainland might be brought, away from their own people.

> It is important that the half-caste children be dealt with separately and distinct from Aborigines. There must be a separate establishment for half-caste children, and the teaching of trades be the most important part of the teaching and training next, of course, to religion. The tendency of the half-caste is to sink to the level of Aborigines...
> (Letter of the Bishop of Carpentaria to Church Missionary Society, Victoria 12.8.18 in Cole 1972:37.)

The mission for 'half-castes' was eventually established on Groote Eylandt at the Emerald River (Yadigba) in 1921 and continued in this role until 1943, when it was moved to Angurugu following the establishment of a wartime RAAF base at the mission's original airstrip at Emerald River. Shortly before that, its 'half-caste' population had been evacuated to the southern parts of the mainland also because of the war. When the mission moved to Angurugu, its focus was the conversion of Aborigines of full descent living on Groote Eylandt.

Although the primary purpose of the mission at Emerald River was education of 'half-caste' Aborigines, the presence of local Aborigines of full descent was not completely ignored. Right from the beginning a small amount of trading was set up, involving exchange of tobacco, metal and medical services for turtle shell, carvings and other artefacts, against a time when closer contact should be desirable. A certain amount of trust does seem to have been built up for, when the mission did move to Angurugu, the local Aborigines who were camping nearby went with them. It is possible that the move was prompted by incidents involving Aboriginal women and RAAF

personnel. It must be stated however that, while these camps were in the vicinity of the Emerald River mission, they were largely seasonal and normal migratory habits had to a large extent remained undisturbed. When the mission was set up at Angurugu, this situation changed somewhat. Short lessons were given, as well as rewards for attendance. A certain relationship of dependence was established. As Turner says

> ...Basically, the Aborigines at Angurugu found themselves in a situation where the people who possessed the goods they wanted - the missionaries - also disapproved of certain of their beliefs and practices, and would withhold their patronage if behaviour in these areas was not to some extent modified. (1974:189)

Modifications entailed postponement of marriage age, especially of women, more monogamous marriages (even today a notice in the church at Angurugu states "Be faithful to your own wife and give your love to her alone"), regulation of marriage classifications, participation in a cash economy, and general subordination of ritual activities to European activities. It must be noted here that although this appears to have been a conscious attempt to change Aboriginal culture, it was principally a result of prevailing European views of the time. The missionaries genuinely believed that what they were doing was right and the Government acquiesced, preferring a sedentary population. They believed that the Aborigines would be happier and better off as economically productive Christian members of society. (They did not know of the role that these people had already played in world

trade without radically change of their way of life). To this end, the missionaries 'taught' the Aborigines gardening, hygiene, cooking, reading and religion.

Thus the changes wrought by missionisation were radical and had no precedent in the history of Groote Eylandt Aborigines. Later changes brought by the advent of the mining were imposed on a culture already significantly altered by missionisation. The major modifications to Aboriginal life included the repression of gerontocracy and polygyny, attempts to impose a sedentary (non-migratory) settlement pattern, changes in diet, changes in health and the conception of saving money for personal use. A discussion of the nature and changes of marriage patterns on Groote Eylandt now follows.

It appears that the people of Groote Eylandt and Bickerton Island belonged to separate exogamous patrilineal clans which were grouped into two moieties. (Worsley 1954, Rose 1960). This pattern of marriage alliance was complex, based on marriage between a man and a woman of the category 'MMBDD'. (However, owing to a broad classificatory system 'mother' was any one ego called mother, and could include mother's sister -who may have a different father or mother from ego's biological mother- and 'mother's brother' could mean anyone whom 'mother' called 'brother' and so on). If a person made a 'wrong marriage', his or her relationship to people in the other moiety shifted accordingly (Worsley 1954:247). However Worsley also documents that, on the whole, marriages were originally fairly well regulated.

Marriage was highly polygynous, and one of the corollaries of this system was that a young girl might be promised to a husband much older than herself (Rose 1960, Turner 1974). A man might also inherit the wife or wives of a deceased clan relative (Rose 1960:79) and become polygynous in that way - some of these women may also be substantially younger than him. All these writers link this situation to a certain amount of tension between the older men, their wives and the younger men. Many incidents of wife 'stealing' by younger men occurred, many of whom stood in just as correct relationship to the women as the man to whom she was betrothed (Rose 1960:247-67:tabular data). Perhaps a corollary of this was the practice of wife segregation as mentioned by Tindale, unusual in the mainland areas. He noted that the women were kept strictly separate so that he only saw the women by accident (1925:72) and says also that the only people allowed to have contact with them were the older men (1925:71).

It would be difficult to prove which was the primary aim of keeping the women separate, although one might conjecture that the motives were somewhat mixed and that while a general rule of separation of women from the young men might have been an effect of the gerontocracy, segregation became much more strict in the presence of people of different cultures. Tension engendered by constant fear of wife-stealing reached a peak in the thirties with the murders of five Japanese and one Australian, all fishermen, who were accused of interfering with Aboriginal women on Groote Eylandt. The policeman sent

to apprehend the murderers was also killed. This contributed to some extent a reputation for aggression of Groote Eylandt Aboroigines which has not been dispelled.

The system of gerontocracy and concomitant polygyny was one of the casualties of missionisation and white moral colonisation (Rose 1990, Turner 1974). Between 1959-65 charges of carnal knowledge were brought by the police against older men with young wives, despite the fact they were by classification allowed to be with them, and some of these men were jailed for the 'offence' (Turner 1974:63).

Another early effect of colonisation was the labelling of the various totemic groups into 'families'. Rose's fieldwork in 1940 had entailed identification of distinct patrilineal clans, and given them names through their totems. Shortly after that, Fred Gray, (a white trepanger who managed the settlement of Umbakumba) and a missionary

> using this data, decided to take active steps to abolish or at least restrict polygyny and older men marrying young wives...the name I had given the patrilineal clans, which were their main totems, were adopted as the names of the various 'families'. (Rose, 1990 personal communication)

During the pre-white contact period, the basic settlement pattern was one of movement - to some extent within the clan territory, but at times over broad areas of the island. It would be extremely difficult to assign an

accurate acreage to each family but, in the interests of clarity, it should be said that the area of Groote Eylandt was 2260 square km, and the basic population was approximately three hundred, split into ten clans at the most. Each clan thus had a reasonable area about which to move. Worsley calculates that the ratio of people to land was in the region of one person to three square miles (five square kilometres), however I find this an arbitrary and misleading conception as clan members were probably dispersed between residence groups. These groups would have been fluid in composition according to season and in any case, not imprisoned within their designated territory. These territories may be seen in the location map on p.(v). The boundaries could be renegotiated and with few problems (disputes on the whole tended to be about women, not land). It is known for instance, and Turner documents the occasion, that at the turn of the century one family from Bickerton Island migrated to the north-west tip of Groote Eylandt following a run of poor wet seasons on Bickerton. The historical owners offered the newcomers land from their own territory and themselves moved to different areas of their territory (Turner 1974:10).

With the second World War and the coming of the RAAF, another such adjustment occurred, and not just at Angurugu. A QANTAS flying boat base was established at Little Lagoon in the north-east of the island, managed by a local white Trepanger named Fred Gray. It was also used by the RAAF during the war. Aboriginal labour was used to build the base. It appears that at about that time there

occurred a split in settlement locations on the island. Those clans that had originally occupied the eastern side of the island (Mamarika, Yantarrnga, Djaragba, Bara and Wuramarrba) went to live at Umbakumba, and those on the west side of the island and from Bickerton Island went to live at the mission (principally Wuramara, Lalara, Amagula and Wuragwagwa). Some clans were split in half by this arbitrary settlement pattern - the Bara, who lived at Bartalumba Bay, appear from census records to have lived both at the mission and at Umbakumba. Perhaps it was a function of who they were married to - those at Umbakumba appeared to have been married to Mamarika, those at Angurugu to Amagula. The basic effect though was that, from being a largely migratory people, they switched to semi-permanent settlements around European centres, engaging in European oriented forms of activity (cultivation, Christianity, health, housework...). It is not within the scope of this thesis to discuss how this was achieved; however it would be fruitful to ponder on the articulation of such a drastic outward change and the extent to which this change was inwardly absorbed.

The fact remains that Groote Eylandt Aborigines of full descent were living at the mission at Angurugu, interacting with the missionaries and eating locally cultivated foods and imported supplies from at least 1943. It appears that the mission garden was cultivated from that time until the mid-sixties, when the manganese mine began operations - a period of twenty years. Angurugu itself continued to be managed by the Church Missionary Society

until 1982. It is hardly surprising that changes should have occurred with such long and proselytising contact, especially in the area of diet, where they were not only eating new foods but also cultivating them.

DIETARY BALANCE AND CHANGES

Tindale in 1921-22 noted carefully the constitution of the Groote Eylandt diet. Naturally, being an island, seafood formed a large part of the diet with turtle and dugong being the most highly regarded. On land the staple was burrawang (cycad) damper, yams and birds, as well as wallaby when available, and other small animals such as lizards and bandicoots (Tindale:78-80). 'Sugar bag' - the honeyed nest of the stingless bee- is still enjoyed above all else, and local fruits such as fig, 'cocky apple' and green plums (the world's richest source of vitamin C - Levitt 1981) were eaten. On the east side of the island, crocodile and turtle eggs were consumed. Pandanus (*P.Spiralis*), which grows profusely on the island, was also consumed. Its nuts were ground into flour, as were cycad nuts, but it was definitely considered 'second best', having a woody taste (Tindale 1925:77). The Aborigines of Groote Eylandt thus appear to have had a balanced diet including meat, fish, vegetables, eggs and fibre, and this was reflected in their physical form. As Tindale noted

> ...The food supply is plentiful on the island and this has a marked effect on the physical well-being of the natives.

> Usually they are well nourished, with a tendency to fatness in some of the men of middle age, but their legs always remain thin. (1925:76)

Worsley (1954) noted an intelligent awareness of food supply and natural resources on the island, and stated that the Aborigines "never merely wandered at random, but exploited the well-known and changing resources in a rational manner" (p.38). When I was on the island, the migratory interaction with diet was evoked by an Aboriginal informant telling me that in the old days they used to spend about a month in each place. When the particular food from that area was used, they would move to a different area and eat the different foods that were available there, and so on. Basically the pattern here was that of 'shifting exploitation' as opposed to shifting cultivation. It might also be noted here that the practice of burning off, as carried out in swidden agriculture, was also carried out on Groote Eylandt. Here it had the function of diminishing the intensity of the undergrowth, making it easier to travel through the bush, as well as of creating the heat needed to crack open the cycad and pandanus nuts for easier extraction of the kernel.

When Worsley was on the island, the same foods that Tindale noted were still available; however, access to them had changed somewhat. Settlement patterns had changed and most Aborigines were clustered around the mission at Angurugu or at Fred Gray's settlement at Umbakumba. Since most of the productive effort was now being put into gardening at both settlements, weekends and holidays were

the only times available to acquire the foods of their original diet. And as Worsley noted (1954:290), a weekend is not quite sufficient for serious hunting or gathering, so that what activities were carried out were conducted close to the mission. Settlement rations consisted mainly of tea, sugar, rice, wheat and flour, as well as vegetables grown in the gardens, both at Umbakumba and Angurugu. Worsley writes that

> ...Both settlements employ a large part of their labour force on the production of subsistence crops...large quantities of food can and have been produced from the gardens. (1954:281)

The gardens at Angurugu have not been cultivated for sometime, and all that remains now is a bare patch of ground bordered on its southern edge by an overgrown orchard. However, an Aboriginal woman, whom I saw retrieve a crowbar from a nearby patch of bush, told me she still got yams there and that the orchard still gave oranges, guavas and bananas. The fact that she needed a crowbar to extract the yams underlines Worsley's information (1954:286) that gardening was an unpopular activity both at Umbakumba and at Angurugu. At Umbakumba especially, it was characterised by hard work and poor return. Insect pests, droughts and cyclones, and the simple fact that it was hard work carried out under a hot sun with no shade, rendered the activity rather unpleasant. The garden at Angurugu was more successful however, as the riverine soil was more suitable for cultivation, and according to mission records, Angurugu was at times more than self-sufficient. By this

time, with introduced foods and restricted movement, the basic diet had become far less balanced. Not only had the focus of their diet shifted away from their original staples, but the new staples were characterised by irregular availability. Cultivation often did not produce enough food to last the whole year, and wheat flour, tea, sugar and rice - the new staples - had to be shipped in from the mainland; supply was thus dependent upon weather conditions. As Worsley noted, this led to frequent situations where the diet was nutritionally inadequate (1954;288) and that if it were not for the occasional availability of naturally occurring foods, the nutritional situation would have been a lot worse. An unfortunate precedent was set, and though a few improvements have been made [1] the nutritional situation remains the same today. The basic problem was that Europeans were ignorant of the available natural resources, and, in supplying hard rations of their own that were portable but less nutritious, they were ignoring the possibilities of the rich and varied diet that the Aborigines ate. What is more, they even succeeded in persuading the Aborigines to accept the comparatively poorer European supplies in its stead - for which, it might be added, they were encouraged to work.

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[1] A large supply of fresh-frozen meat and bread and cold-stored vegetables arrives every Tuesday, and the store is now more accessible to Aborigines, allowing for a little more control over supply.

CASH AND THE MISSION ECONOMY

Right from the start of the mission, the missionaries began educating the local Aborigines in the value of cash. The Macassans had, it is true, exploited the idea of remuneration for work, but the value of that work was never fixed (Worsley 1954:13). Leslie Perriman, one of the first resident missionaries on the island (along with A.J. Dyer) documents the system by which this was achieved.

> Our rule in trading was that we should not give anything for nothing, except medicine, nor take anything for nothing...Tobacco was the common currency on the mainland among Aborigines...Each stick of tobacco was cut into four, each piece representing a penny. We arranged a chart system so that when Aborigines wished to purchase articles it was possible for them to see their savings grow like a thermometer, with the parallel lines marked off in squares, each representing a penny. As the pennies in the squares were shaded much interest was created. (L. Perriman in Cole 1972:44)

A whole thesis could be written on the view of money thus engendered - the effects of encountering money as a resource to be possessed for the purpose of exchange for another item as soon as there was enough of it, and the views of the source of supply. Here however, the salient feature is that the introduction of a regulated cash economy was set up by the missionaries. This may have provided a certain amount of understanding about the receipt of cash rather than goods as a return for work; thereby creating opportunities for the Aborigines to work

in the manganese mine when it was opened. The missionaries also tried (and the church is still trying) to encourage the Aborigines to save money for the future and to reserve it for their own nuclear family (Turner 1974:179). However, in view of the conception of cash for use (which it appears the missionaries themselves set up), as well as a basic indigenous communal ethic, this has met with little success.

CONCEPTS OF HEALTH AND ILLNESS

With reference to Perriman's contention that nothing was given away free except medicines, it is very interesting to contemplate the view of western medical care thus engendered. It seems (Perriman in Cole 1972) that the Aborigines would come to the mission, even in the early days, for pills and embrocations - a band-aid approach. With their basically healthy lifestyle, and wide knowledge of plants for medicinal use (Levitt 1981), even this antidotal approach was not always necessary. The mission would initially have been seen as a last resort. The fact that medicine was given free, however, might have influenced its use - the collection of plants for medicinal preparation did involve a certain amount of effort. Even today, the delivery of health care appears to be seen as an antidotal resource by Aborigines on Groote Eylandt, however much effort is put into Western-style health care and disease prevention.

Unpublished data on the island suggests that Groote Eylandt Aborigines conceived of four states of health, the first being 'healthy', the second 'ill', the third 'wounded' and the fourth 'weak'. Healthy people didn't need care, ill or wounded people received treatment - by themselves or the mission. Weak people seem not to have received any special attention. This leads me to conclude that conceptions of health care and disease prevention largely emerge from the different cultural and physical experiences of different societies.

In the wider context of general Aboriginal health C. Berndt (in Reid 1982) addresses this contention in her essay on the articulation of Western and Aboriginal ideas of health in western Arnhem Land

> ...In the Australian European society that has been imposed on the western Arnhem Landers, as on other Aborigines, there has been and still is an emphasis on specific aspects (signs) of illness, disease' injury and the like- on specific physical manifestations, visible or inferred....But the views of the western Arnhem Landers...were much more comprehensive...The western Arnhem Lander's concern was with individual persons in a much wider context. (1982:136-137)

Berndt documents that disease prevention in the Aboriginal context included practices such as the mothers keeping their children away from the dangers posed by poisonous plants or sorcery, and in teaching their children to manage for themselves in these areas as they grew up.

Reid also states that the health of Aborigines is not seen by them as a matter of luck or the reflection of a good diet.

> It is the outcome of a complex interplay between the individual, his territory of conception and his spiritual integrity... his body, his land, and his spirit. (1982:xv)

Western medical intervention continues to produce newer and more effective corrective measures, but the gulf between the Indigenous and western European conception of health care continues to exist.

Worsley noted in 1954 that the Wanindiljaugwa were singularly free from major diseases (apart from leprosy) and that hookworm and malaria were virtually unknown. There are in fact anopheline mosquitos on the island, but the malaria Plasmodium is fortunately absent. The mode and time of the introduction of hookworm onto the island is not known, but now it is rampant. In fact, since Worsley's fieldwork, a wide range of conditions have become endemic, including hookworms, diabetes, middle ear infections, petrol sniffing and alcoholism. It would appear that the rapid influx of white people and western goods that has occurred since the opening of the manganese mine at Angurugu is associated with this state of affairs.

MANGANESE MINING

Unbeknownst to the missionaries, the site they had selected to move to from the Emerald River, and which they had carefully chosen for its abundance of fresh water, was also abundant in manganese. Groote Eylandt is unique in this respect as elsewhere in the world, manganese is found far underground and deep shafts need to be sunk. At Angurugu, there are manganese outcrops at the surface. Its presence was noted on the western coast of the island as early as 1907 (Cole 1988:19) during an exploratory expedition to the area, but the knowledge was not acted upon. Manganese was also noticed at Angurugu in 1955 but again no action was taken - it was not until 1960 that overt interest was shown by the Commonwealth Department of Mining. The Church Missionary Society then sought mining titles and started prospecting themselves (Cole 1988:23). Extensive outcrops were discovered in five areas around Angurugu. In fact, the western margin of Groote Eylandt has a barely concealed geological strata of manganese, ranging from one to eight metres thick. By the time BHP expressed an interest, the missionaries knew enough about the nature and extent of manganese on Groote Eylandt to negotiate successfully for substantial royalties to be paid to the Aborigines if mining was to go ahead. However ideologically questionable the process of missionisation is, it seems that within their own code these missionaries were strictly fair. The Groote Eylandt Mining Company (GEMCo) was formed as a

wholly owned subsidiary of BHP, and the first shipment of ore was taken out in 1966.

Certain facts about the leases GEMCo mine are relevant to this thesis. Firstly, the mining areas are close to Angurugu township (the stockpiles themselves are less than a kilometre away) and they allow for Aboriginal movement over leased land (Cole 1988:22). This means that the stockpiles are not fenced off. The metal dump at the edge of the stockpiles are a common place to see Aboriginal people 'recycling' items of metal discarded by the company. The whole town of Angurugu is steeped in manganese - the rich soil that made the vegetable garden a success was also particularly high in manganese (Cawte & Kilburn 1987:33) There is an old mine site about eighty to one hundred metres from the other side of the garden. The concentrator, a machine that crushes and blends the manganese ore is situated on the other side of the stockpile - about 1500 metres from Angurugu.

The coming of the mine brought new pressures to the communities on Groote Eylandt. The periodic payment[2] of large amounts of money, being the royalties from the mine, had great impact on a people who had previously had their cash administered by the missionaries. With the money also came the western goods on which to spend it - cars, sound systems and alcohol. The availability of employment, coming as it did simultaneously with the

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2 Every year on 2nd July a portion of the interest from the royalties of the mine is paid to Angurugu Aborigines. According to Turner (1989) this amounts to approximately 20 thousand dollars to each clan.

royalties, sat oddly in this situation. While receiving such large amounts of money, the motivation to work simply has less impact. Turner notes (1971:175) that in the subsistence economy production for immediate use was the norm, and demonstrated that employment patterns of Aborigines tended to reflect this state of being. Larger numbers of Aborigines than white people lose or 'abandon' their jobs and are re-hired, and there is higher absenteeism than with white employees, especially when family commitments clash with working times (Turner 1974:173). To some extent this interferes with training and consequently job prospects. Nevertheless, there are Aborigines on the island who have worked consistently for the mine over a lengthy period.

Before the setting up of the Council, employment was also possible with the mission. Since 1982, when the mission handed power over to a local Aboriginal Council, the Council has employed a certain number of Aborigines as well as white people. The local shop, which sells a wide range of goods, is another employer at Angurugu as is the Northern Territory Health Department which employs eight Aboriginal health workers at Angurugu and four at Umbakumba. Most of these jobs are also characterised by fluctuating attendance and numbers of staff, though the Council appears to be more stable. GEMCo also provides employment in Alyangula, the company town (about twelve km north of Angurugu), mostly connected with maintenance work and garbage collection.

Before the coming of the mine, there was little if any alcohol available. Now, owing to the horrendous impact of alcohol upon the population, Angurugu is again a dry area. Beer can be obtained at Umbakumba and from Bartalumba Bay. The Council at Angurugu has banned Aboriginal patronage of the club at Alyangula in an attempt to decrease alcoholism. There is, however, nothing to stop the Groote Eylandt Aborigines from visiting Darwin and consuming alcohol there. Alcohol, and its twin addiction on Groote Eylandt - petrol sniffing, has had a devastating impact on the population of Groote Eylandt and resulted in large numbers being either imprisoned or hospitalised in Darwin.

Local feelings about the mine are, understandably, mixed. On one hand manganese mining brings employment to the island; on the other hand, large numbers of social problems can be traced to the same event. One problem which is, rightly or wrongly, linked to the opening of the mine is the emergence of the Bird Disease. An extremely visible manifestation of the mining is the copious quantities of manganese dust that lie on any surface. The role that this dust plays in the emergence of this disease will now be discussed, as will other aspects of the disease's appearance and theories of its causation examined.

photograph 1.1: The mission garden at Angurugu.

photograph 1.2: The mission church at Angurugu.

CHAPTER 2

MEDICAL METHODS

In this chapter I will discuss the symptoms of the 'Bird Disease' and the causal processes with which they may be linked. I shall begin by presenting a general clinical picture of the syndrome.

SYMPTOMATOLOGY

The Bird Disease is a syndrome affecting the central nervous system (a syndrome is a combination of symptoms resulting from a single cause, or so commonly occurring together as to constitute a distinct clinical picture Miller & Keane 1983). In the Bird Disease the combination of symptoms forms two distinct clinical pictures, linked however, by their familial pattern of expression. These two forms were isolated and identified by Dr Charles Kilburn during his study of the Bird disease on Groote Eylandt. (Cawte and Kilburn 1987: 37).

One of these combinations is the ataxic variety, which is suffered by six people. The age of onset for this variety is during adulthood- on average at about thirty-five years, but may range to as early as nineteen years (see Table 2.1). The early signs and symptoms are; difficulty in balancing, especially on arising, and difficulty in walking. The sufferers develop a characteristic wide-based gait and pick up each foot

delicately and deliberately for the next step. This gait led to the naming of the disease, as it somewhat resembles that of wading birds. Other signs and symptoms include increased muscle tone and spasticity, tremor of the hands, nystagmus, and some paralysis of the ocular muscles. Late signs and symptoms include moderate to severe muscle wasting, paralysis, slurred speech, dementia (occasionally) and difficulty in swallowing. One person died of the disease before it came to medical attention. Her case has been labelled 'unclassified neurological disorder'. It seems likely from Kiloh et al's reconstruction of her symptoms (1980) however that she suffered from the ataxic form of the disease. At present, one man is bedridden from the disease and spends much of his time in a nursing home on the mainland. All the six people who have suffered the ataxic form are full and half brothers and sisters.

The other manifestation of the disease is its congenital form. This is a variety of Motor Neurone disease (known in the USA as Amyotrophic Lateral Sclerosis, or ALS) and is characterised by muscle weakness and ligament laxity (Cawte & Kilburn 1987). Classically, there is a medical history of delayed walking, and when walking is achieved it is laboured. 'Double joints' are a feature, for example, 100 degrees of movement may be possible when pushing the fingers over to the back of the hand. Other symptoms include spinal malformations (e.g. kyphosis, scoliosis), decreased reflexes, decreased muscle tone and flat feet. Eleven people have suffered from this manifestation of the disease and again, there are varying

degrees of severity. A seven year old child died from the disease in 1989, largely due to the consequences of her immobility.

Evidence of the exact type of brain damage occurring has been inconclusive, because it has not been possible to carry out any post mortems. A limited number of Computerised Tomography Scans have been done and show atrophy of the cerebellum, but cannot demonstrate the processes of that atrophy. Details of the age, sex and variety of Bird Disease suffered are set out below.

TABLE 2.1 DISEASE MANIFESTATION BY AGE AND SEX

CASE NO	DATE OF BIRTH	SEX	TYPE	AGE OF ONSET	SOURCE
1	1938	M	ALS	adol	K +Ki
2	1935	F	ataxic	30's'	K +Ki
3	1939(d.1971)	F	?ataxic	19	K +Ki
4	1940	M	ataxic	30's	K +Ki
5	1943	M	ataxic	30's	K +Ki
6	1947	M	ataxic	35	K +Ki
7	1945	M	ataxic	43	K +TB
8	1956	F	ALS	congenital	K +Ki
9	1958(d.1981)	M	ALS	congenital	K +Ki
10	1960	M	ALS	congenital	K +Ki
11	1961	F	ALS	congenital	K +Ki
12	1962	F	ALS	congenital	K +Ki
13	1967	F	ALS	congenital	K +Ki
14	1968	F	ALS	congenital	K +Ki
15	1972	M	ALS	congenital	K+ TB
16	1975	F	ALS	congenital	K +TB
17	1982(d. 1989)	F	ALS	congenital	K +TB

(Ki= Kiloh 1980, K= Kilburn 1987, TB= Tim Burt 1990)

According to Kiloh et al (1980), the earliest case to emerge was that of a male, born in 1938. He was noted in

1951 to have a kyphosis and an abnormal gait. His case was mild and has been progressing slowly since then. Kiloh does not document the cases born after 1968 of which there are four, all suffering from the ALS form, though he does note three cases at Wanduwuy,on the mainland [3] (see location map). Since Kiloh's article was written one more person on Groote Eylandt has been diagnosed with the ataxic form. He is one of the six cases mentioned above. Correlating Kilburn's and Kiloh's case histories with my own data and that in the genealogies thus proved a detailed task involving identification of each case.

SOURCES AND METHODS

Identification of the cases involved checking the data provided by Kiloh *et al* (1980) and Kilburn (1987). Kilburn also provided genealogies of each family. The data provided by Kiloh *et al* did not precisely match Kilburn's data; firstly because Kiloh documented the cases on the mainland, and secondly, because some cases were born, or their symptoms became apparent only after his paper was written. Kilburn does not document any cases on the mainland. The genealogies provided by Kilburn were useful, though they required amplification. During my field visit to the island therefore, I cross-checked mission census and genealogical data with the tabular data provided by Rose, and with information given to me by local Aborigines, in an

[3] Burt and Currie (pers. comm) noted cases of the ataxic form of Bird disease on the mainland, apparently genetically inherited in an Autosomal dominant pattern

attempt to find out the identity of both parents of each affected person, rather than the single parent documented by Kilburn. This was successful, and the results will be discussed in the section on genetic inheritance. Overall, I felt that the genealogical data needed to be explored, for it seemed that with the Aborigines tracing marriages through the mothers, and mission-based census data tracing genealogies through the fathers, a picture of the whole might reveal more information. The genealogical conclusions I make are, of course, based on the assumption that the census data is biologically correct. Information given to me on the island suggests that on the whole the census data is correct, as the terms 'same father/same mother' can be used to mean the identities of the biological parents. The genealogies did prove misleading in one instance- they did not provide correct information about the mother of the father of case 13. I discussed the genealogy with case 13's father, and he corrected it for me. The correction is seen in the genealogy on p29a.

It was necessary to establish a coding system in order to maintain patient confidentiality and still present a clear diagrammatic picture. Thus, each family has a number, odd if the family is in moiety one, even if the family is in moiety two. The two families whose genealogies are presented here are Family One and Family Three. However, owing to the fact that surnames given by the missionaries are passed patrilineally, the surnames of the cases are not limited to these two family names. It is thus necessary to be familiar with the genealogies of these

two families in order to see that the cases do belong to two particular families. These genealogies are presented in Figure 2.1 (over page). It is here that one of the Western explanations must be noted- that of genetic inheritance- for it is obvious from my reworked genealogies that all cases may be traced back to two men and that three of these cases (of which two are siblings) may be traced back to both of these men who are not, however, known to be related to each other. The original genealogies provided by Kilburn can be seen in Appendix One.

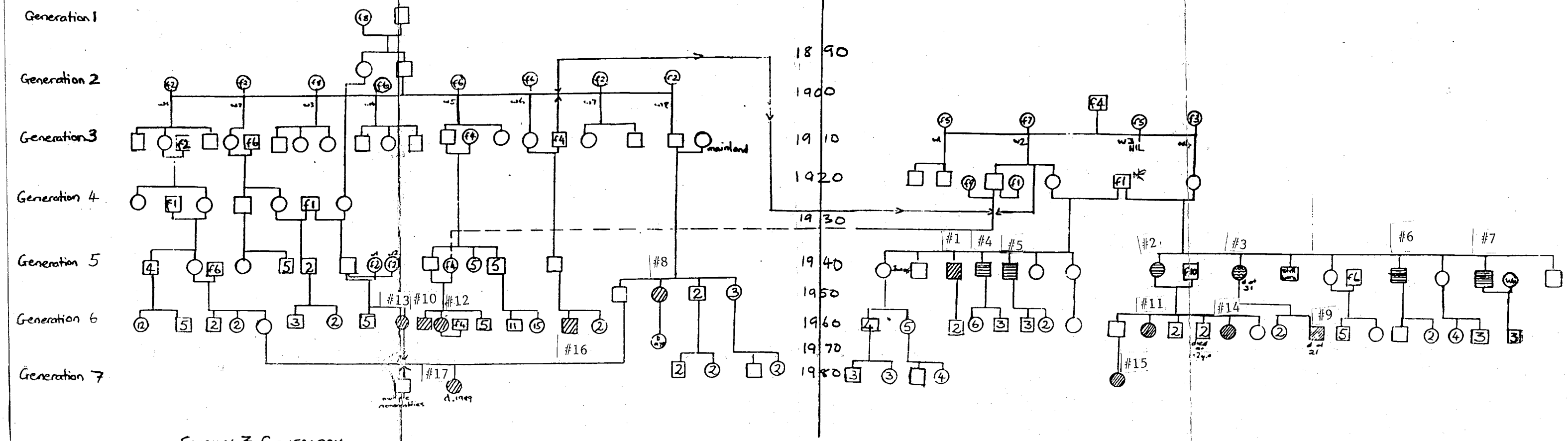
The familial link has not gone unnoticed by the Aborigines. One theory put forward by them to explain the Bird Disease involves the possibility that some 'wrong marriages were made, and that some partners were too closely related. Turner studied this possibility in 1987, and could not find any evidence that this was the case (Cawte & Kilburn 1987:55). However, from conversations I had with local Aborigines, it appears that this idea still has currency. Two variations on this theme are, firstly, that some marriages took place between Aborigines and Europeans (either mining or RAAF personnel) which threw the marriage system out of balance and caused some 'wrong marriages', and secondly, that the interference of missionaries caused some people to marry spouses whom they should not have.

A counter theory held by the Aborigines is that the Bird Disease is caused by manganese dust from the mine. The presence of the stockpiles so close to the town gives

graphic credibility to this explanation. There is a great deal of dissent between the proponents of these theories.

Other explanations given by Aborigines are concerned with sorcery and spiritual contamination. These explanations were not communicated to me, one does not confide spiritual or religious fears to a two-week visitor. They were documented by Cawte (1984) and Turner (1989). Cawte states that some Aborigines on Groote Eylandt attribute the disease to sorcery, and more specifically to disfigurement of a dreaming place by 'strangers'. The fact that agency is attributed to 'strangers' is perhaps appropriate, as contact with other people, especially of other cultures, has had its darker side. Turner, on a recent visit to Groote Eylandt, discussed the Bird Disease with Aborigines, and reported that spiritual contamination might be a possible cause. He disagrees with Cawte (though not in so many words) and says that no-one believes that the Bird Disease is caused by sorcery. Turner's explanation of spiritual contamination is delicately worded and must be quoted in full to be understood clearly.

> ... In pre-mission times bodies were raised on platforms until the flesh decayed off the bones, at which time the bones were wrapped in bark containers and buried in caves in the deceased's country. The body-juices though, were scooped up with baler shells as they fell to the ground and later evaporated over a pile of hot stones.... The danger was that someone would come into contact with the body-juices as they fell or were being collected and become 'contaminated', suffer spiritual injury. This, they insist, is what happened to ('*' in family one) two generations



FAMILY 3 GENEALOGY

FAMILY 1 GENEALOGY

KEY

- ALS Form
- Ataxic form
- 'Unclassified Neurological disorder
- No. of children, each sex
- family no.
- #x — case no.
- * — mentioned in Turner 1989

> ago. Now his 'fractured spirit' is showing up in his grandchildren, producing the same effect as if they had been the victims of sorcery. (1989:253)

Indigenous explanations such as these are important in any discussion of illness suffered by people in a different culture, as will be discussed in chapter three. However, the causal theories which I shall be discussing will come from a Western medical basis. This is not because I regard indigenous explanations as somehow less relevant than our own, because it isn't, especially in the context of reaching an effective solution. It is because most of the research carried out on the Bird Disease comes from within this medical context.

Within a European model of disease causation I shall be discussing the possibilities of genetic inheritance, manganese poisoning, malnutrition and cycad nut poisoning. To some extent this is because these are the theories that have been addressed in the literature and must therefore be evaluated, but also because the context seems naturally to give rise to them. I shall also be discussing theory from the field of slow virus research. Kiloh et al (1980) suggested a possible link between the Bird Disease and the Amyotrophic Lateral Sclerosis/Parkinson's Dementia disease complex of Guam and Japan. Following this lead I found that there does indeed seem to be a link, and research done on slow viruses may also help research into the Bird Disease.

Definitions of medical terms used in this chapter may be found in the glossary at the end of the thesis.

GENETIC INHERITANCE

If genetic inheritance is postulated as the cause of the Bird Disease, then two possibilities arise. Firstly that the disease might be a simple case of a genetically inherited disease; and secondly that genetic inheritance may not be a sole cause, but may play a part in causing a person to be more susceptible to other factors such as toxicity, malnutrition or viral infection.

With seventeen cases, the sample size is very small for the task of inferring a specific mode of inheritance. By that same token however, the task is more quickly undertaken.

The chances of the bird disease being a simple case of mendelian inheritance are slim. Such a case can be tested upon compilation of a genetic genealogy (pedigree) and is not apparent here. The types of genetic inheritance which can potentially be uncovered by the compilation of a genealogy include; sex-linked inheritance (x-linked, dominant or recessive; or Y-linked) and autosomal inheritance; (dominant or recessive).

The even balance between males and females in the incidence of the disease would seem to preclude the possibility that the disease is carried on either of the sex chromosomes. Such a possiblity might be suspected if there were a grossly imbalanced sex ratio, especially if the sufferers were all or preponderantly male. If a gene for the disease were carried on the Y chromosome, then one would expect the disease to be confined to males, as only

males receive the Y chromosome. If a gene for the disease were linked to the X chromosome and recessive, then one would also expect the disease to be confined to males[4] although females can carry it and pass it on to a son. (See figure 2.2). Rarely, a condition exists which is carried on the X chromosome and dominant. The genotype of the offspring will vary according to whether the male or the female parent is affected. Overall however, it is expected that, in a rare condition, the sex ratio of affected individuals will approximate to one male to two females (See figure 2.3). These conditions are not seen in the genealogies.

Figure 2.2 X linked (recessive)
X'/Y' = affected.

Mother a carrier

X'X + XY

X'X X'Y XX XY
= 50% unaffected,
25% female (carrier)
25% affected

Father affected/Mother not

XX + X'Y

XX' XY XX' XY
=100% females
100% males unaffected

Father affected, Mother a carrier

X'X + X'Y

X'X' X'Y XX' XY
=25% female, carrier
25% female, affected
25% male, affected,
25% male unaffected

[4] Males only receive one X chromosome; if the X chromosome they receive carries the disease then it will be manifested whether recessive or not.

Figure 2.3 X-linked dominant
X'= affected

<u>Affected Mother</u>

X'X + XY

X'X X'Y XX XY

=25% female, affected

25% female, unaffected

25% male, affected
25% male, unaffected

<u>Affected Father</u>

XX + X'Y

XX' XY XX' XY

=50% female, affected

50% male, unaffected

If a strict hypothesis of autosomal dominant mendelian inheritance is adopted (Fig 2.4), then for every affected person there must be an affected parent. Only five cases are entirely consistent with this picture, thus at less than fifty percent of cases the statistical basis is not strong. The fact that the children of other affected cases (1,4,5,6&7) do not appear to have the disease is not inconsistent with the possibility of dominant mendelian inheritance. Given that each new embryo conceived has the full range of genetic probabilities available, then it is possible that in a couple where one person is an affected heterozygote, all of the offspring may be unaffected, even if there is only a 25% chance of that occurence (see Fig 2.4 eg#2). Another possibility is that of incomplete penetrance, where the dominant affected gene does not 'penetrate' the normal side during development, or does so only to a small extent, in which case there is a pattern of variable expression of the phenotype.

If genetic susceptibility to another (as yet unknown) factor is the case, then these cases may still be

consistent with a picture of autosomal dominant inheritance, as it may be possible that the disease-inducing factor had not been activated in the parent's generation (around the turn of this century). If these extra cases did comply with the picture of autosomal dominance, then over fifty percent of cases can be seen as consistent with an autosomal dominant inheritance. However, with such a small number of cases overall, even one non-conforming case represents a substantial argument against the possibility. Cases 15 and 17 are two cases which are not compatible with a picture of autosomal dominant inheritance, as their parents do not seem to have the disease.

It is possible that there is a recessive pattern of inheritance occurring, and that the parents may be carriers (heterozygously affected and therefore phenotypically unaffected-see fig 2.5). As there are only two examples of this however. then the possibility that the overall pattern of inheritance could be compatible with recessive inheritance is highly unlikely. In recessive inheritance both parents must be carriers of a disease for it to be affected. As the disease appears to be confined to the two families, one would expect that if a hypothesis of strict autosomal recessive inheritance the cases would be confined to the children of couples of particular marriages, for example either of these two families and family four, from whom many of them are descended. This does not occur. It seems that the possibility that the overall pattern of

inheritance could be that of recessive inheritance is unlikely.

FIG 2.4 DOMINANT INHERITANCE
A =affected, a =unaffected

(Ratios of expected outcomes)

#1 <u>one affected parent</u>

Aa + aa
Aa Aa aa aa
50% affected heterozygously
50% unaffected

#2 <u>two affected parents</u>

Aa + Aa
AA Aa aA aa
25% affected homozygously
50%affected heterozygously
25% unaffected

#3 <u>Two affected parents</u>

Aa + AA
AA AA aA aA
50% affected heterozygously
50% affected homozygously

FIGURE 2.5 Recessive inheritance.
(showing ratios of expected outcomes)

(A=normal, a=affected)

1. <u>With one affected parent</u>.

AA + aa
= Aa, Aa, Aa, Aa.
(all carriers)

2. <u>With two carriers</u>.

Aa + Aa
= AA, Aa, aA, aa.
(25% normal
50% carriers
25% affected)

3. <u>One carrier with one affected</u>.

Aa + aa
= Aa, Aa, aa, aa.
(50% carriers
50% affected)

4. <u>2 Affected parents</u>.

aa + aa
= aa, aa, aa, aa
(all affected)

Although the pattern of inheritance does not seem to comply totally with that of classic mendelian inheritance

in any form, the fact that the Bird Disease is so closely familially linked still suggests that there is a genetic pattern. The details of the genealogies were applied to a statistical package (GLIM) and this picture was found to be statistically viable. The test that was used was that of logistic regression. Ratios of affected to unaffected siblings were looked at in order to test the hypothesis that they are relatively homogeneous across the genealogies and consistent with a genetic basis. The data was found to be compatible with a model of genetic inheritance with a mean proportion of 0.28 affected. More formal statistical investigation was not viable because of the small sample size. The most plausible explanation of the familial profile of the Bird Disease seems at this stage that it is compatible with a genetic mode of inheritance characterised by incomplete penetrance.

MALNUTRITION

As with genetic inheritance, it would be hard to ascribe malnutrition on its own as the cause of the Bird Disease, though as it may play a part in rendering a person susceptible to other factors, it must be discussed here. Also, the dietary profile on Groote Eylandt is rather poor, and should perhaps be considered in this context.

The causes and effects of clinical malnutrition are, on the whole, well known. Protein and calorie deficiency lead to Marasmus and Kwashiorkor; lack of the various vitamins can cause pellagra, scurvy, beri-beri,

rickets; and lack of iron causes anaemia. These will be discussed in relation to their neurological effects.

Anaemia: Chronic anaemia is endemic on Groote Eylandt. This stems from a high level of hookworm infestation. Florence et al (Cawte & Kilburn 1987:26) noted that iron and manganese have the same uptake mechanism, and that when iron intake is increased, manganese uptake is also enhanced. Thus, while one could not say that anaemia itself gives rise to the Bird Disease, then if manganese has a role to play then iron deficiency may certainly increase the susceptibility of a person to contract it. Other nutritional deficiencies and their neurological effects include:

(a) Beri-beri. This is due to a deficiency of Vitamin B1 (thiamine) and occurs not only in third-world countries but also in developed countries, due to malabsorption syndromes or chronic alcoholism. It may generally be ruled out in connection with the Bird Disease, as the symptoms are very different, being those of swelling, paralysis of breathing muscles and limbs, sensory loss and heart failure. Treatment is in most cases visibly and rapidly effective.

(b) Pellagra is a deficiency of vitamin B2 (niacin) and may also be ruled out as a direct cause of the Bird Disease. It is characterised by gastro-intestinal disturbances and thickening of the skin (the Bird Disease is notable for the appearance of thinner, more flexible skin), and incoordination occurs only in the late stages. According to Brain (1977:850) "the

clinical picture is unique, and can hardly be confused with anything else".

(c) Pernicious anaemia is due to a deficiency of vitamin B12 (cyanocobalamin) and has been called 'subacute combined degeneration' because it may attack the nervous system. However, a classical (diagnostic) symptom of pernicious anaemia is loss of sensation, and Kiloh in his (1980) discussion of the signs and symptoms of the Bird Disease stated specifically that loss of sensation is *not* a feature of the Bird Disease.

(d) Kwashiorkor and Marasmus (protein and calorie deficiency) have no neurological manifestations as such, though both may retard intellectual and physical growth. While sufferers of the ALS from of the Bird Disease certainly suffer from retarded physical growth, the other condition is not met; neither does it apply in the case of the people with the ataxic condition, as their growth appears to have proceeded normally.

Thus, in the context of malnutrition, the nutrient deficiencies do not correlate with the neurological symptoms of the Bird Disease and that, where they do exist, treatment is visibly effective. Iron deficiency is the most plausible type of malnutrition that may be linked to the Bird Disease. Indeed, this may be taken further and linked in with other mineral deficiencies, for example calcium deficiency. The way in

which these dovetail into other possible causes will be approached in the discussions of the roles that manganese toxicity and slow virus-related diseases may play in the Bird Disease.

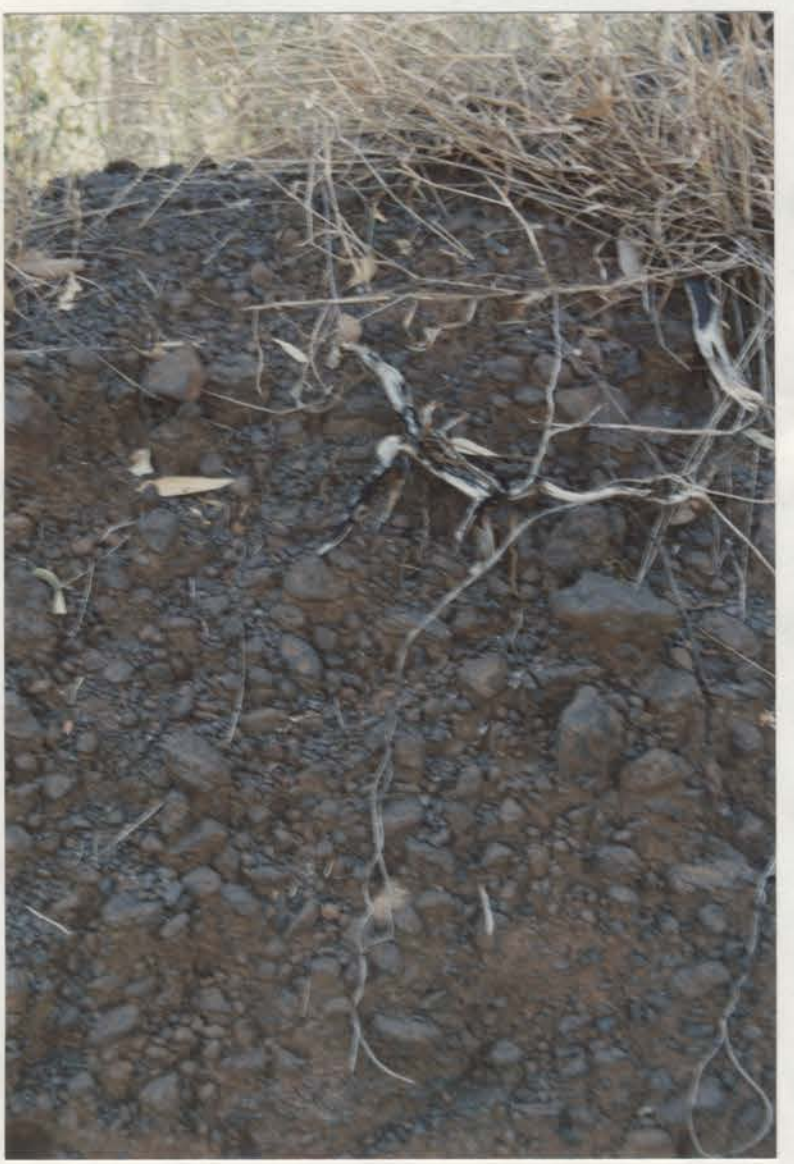
MANGANESE POISONING

Groote Eylandt is rich in manganese, particularly on the western side of the island where Angurugu is located. Indeed, Groote Eylandt is a major producer of the western world's manganese supply (Cole 1983:49). The mining situation on Groote Eylandt is unique - elsewhere manganese occurs far underground (e.g. Chile, India) and miners must work in deep pits. On Groote Eylandt the manganese occurs on the surface and outcrops are readily visible. Angurugu is thus built right on top of one of the world's richest supplies of manganese, and the soil that made the mission garden such a success is composed largely of manganese pisoliths (see photograph 2.1). Mine sites and manganese pervade the town, as demonstrated by the following series of photographs (2.2 - 2.6). I have mentioned in the previous chapter how Angurugu Aborigines wander through the edge of the stockpiles to the metal dump.

Manganese is known to have adverse effects on the central nervous system and as such, its potential role in the causation of the Bird Disease must be discussed here. Firstly I shall give an account of the classic presentation of manganese poisoning, and then I shall discuss possible

differences and similarities with the Groote Eylandt situation.

Over-exposure to manganese occurs most commonly in those people directly in contact with manganese; for example, manganese miners and workers in dry battery factories which use manganese oxide. Ingestion usually occurs via inhalation of the dust. Symptoms and signs occur in stages.

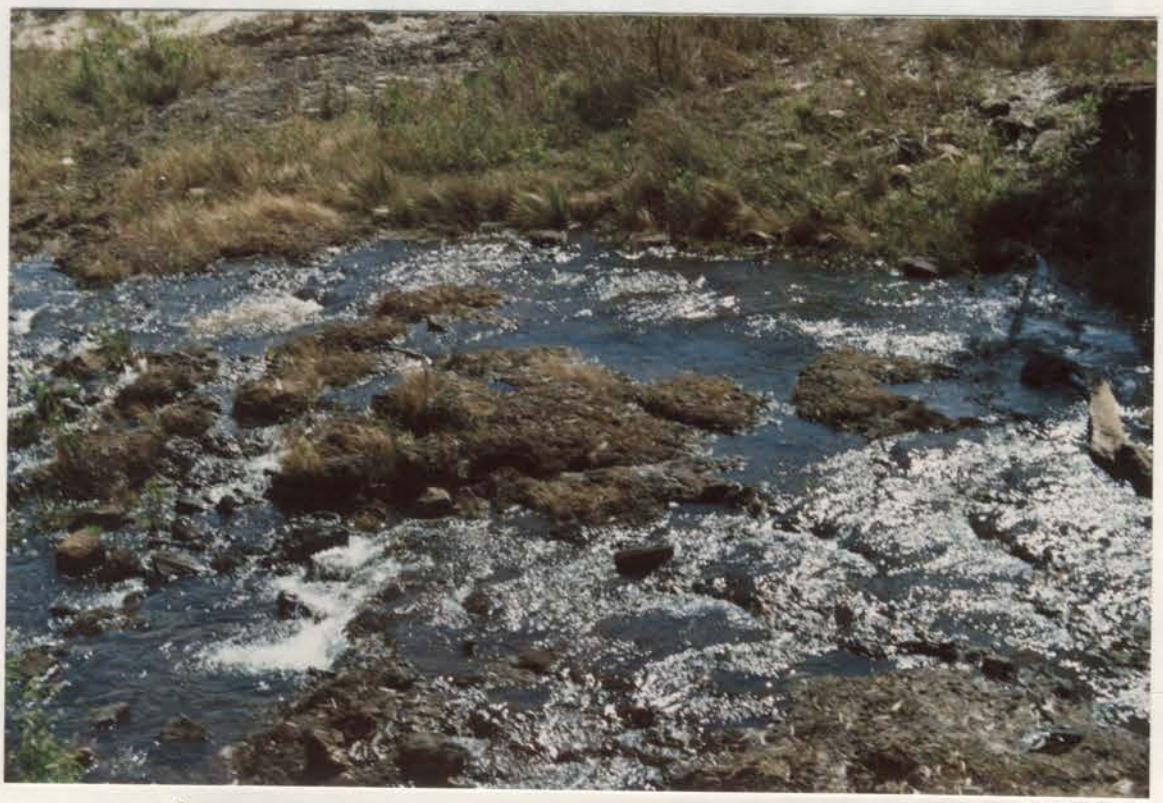


Photograph 2.1 Cross section of soil, mission garden, Angurugu.

photograph 2.2: Manganese outcrop at Angurugu (behind health centre)

photograph 2.3: Mine stockpiles, one km. from Angurugu.

photograph 2.4: A regenerated mine site at Angurugu. The mission garden is 50 metres away through the trees on the right.



photograph 2.5. A manganese outcrop in the Angurugu river.

Photograph 2.6 Soil sample, 3km North of Angurugu at mine site

black = manganese
red = iron
white = clay

The first group of symptoms to appear are the 'psychomotor' or behavioural signs. These are characterised by hyperexcitability, nervousness, compulsive actions and depression. In mining villages in Chile this is a well known phenomenon and is called 'locura manganica' - manganese madness[5] (Mena 1979:218) These symptoms last for one to three months, apparently irrespective of whether the patient is removed from the source of toxicity. If removal does not occur, then towards the end of the occurrence of the first group of symptoms, a second group of signs and symptoms emerges.

The second group of signs and symptoms is that of the neurological disabilities. These include abnormal gait, muscular weakness, increased muscle tone, 'stiffness' or 'heaviness' in the legs. These symptoms may be alleviated, and even reversed if the patient if removed from the source of toxicity, even after the disease progresses beyond this stage. However, after one to two years, the symptoms may become established (Mena 1979:220). Further symptoms include progressive speech impairment, sialorrhoea, expressionless faces, headache, clumsiness of movements, sexual impotence and tremor of the hands.

The mask-like face, tremor and shuffling gait referred to above have, in the past, led researchers to link the degenerative changes caused by manganese toxicity to those

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5 This set of symptoms is not confined to manganese toxicity. Shiraki (1979) noted the presence of similar symptoms in patients suffering from organic mercury poisoning. He suggests that the symptoms might be an early reaction of the Central Nervous System to any noxious stimulus.

of Parkinson's Disease (dementia). Further cementing this impression is the fact that Levodopa, the drug of choice for Parkinson's Disease, is effective in treating many (but not all) of the signs and symptoms of manganese toxicity. (Mena 1979, C.C. Huang et al 1989). Barbeau et al (1976) however, believe that it is a mistake to link Parkinson's Disease and Manganism too closely, and that this dystonia is merely one manifestation of the dystonias that manganese toxicity may cause. Voss hints at a link between progressive bulbar paralysis and Amyotrophic Lateral Sclerosis with chronic manganese poisoning in his 1939 article. Kilburn (1987:40) notes that increased manganese levels may be found in the spinal cords of patients dying from Amyotrophic Lateral Sclerosis, which relates well to the Groote Eylandt situation and will be referred to in that context.

The actual mechanism whereby ingested manganese causes disease is largely unknown (C.C. Huang et al 1989). This could in part be due to the fact that varying forms of brain damage have been shown to result from manganese toxicity and consequently the physical abnormalities are more diffuse. Links between cause and effect are thus more difficult to establish than in diseases that are the result of a toxin on one particular area of the brain (e.g. mercury poisoning). Types of brain damage occurring as a result of manganese toxicity include varying amounts of degeneration of the pallidum, caudate nucleus, putamen and substantia nigra, as well as occasional damage to the pons, thalamus and pyramidal tracts. All these areas of the

brain are in close relation to the cerebellum (see diagram at fig 2.6) - the area which showed degeneration in the CT scans of affected Groote Eylandt patients. In itself, this is of course far from conclusive proof of a link between manganese and the Bird Disease.

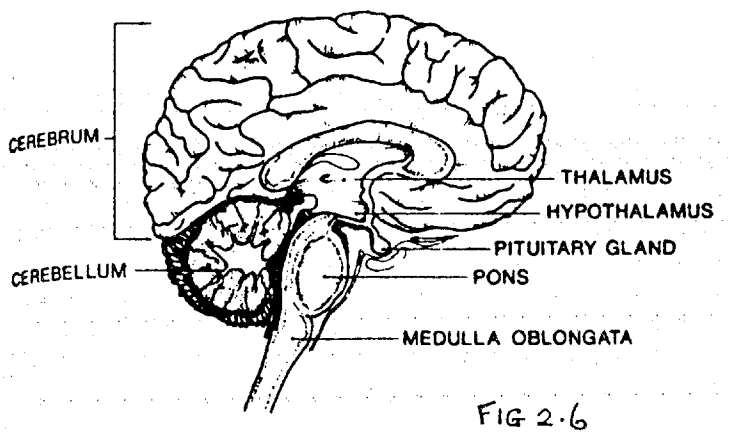

FIG 2.6

brain / brain stem 161

Parts of the brain. (From Chabner, D-E.: The Language of Medicine. 2nd ed. Philadelphia, W. B. Saunders Co., 1981.)

A discussion of the similarities and differences of the Bird Disease to Manganese toxicity now follows.

The classic presentation of manganese poisoning, i.e. that of the young to middle-aged male miner whose symptoms progress from early behavioural disturbances to Parkinson-like Dementia is not seen on Groote Eylandt. Nor does inhalation seem to be the principal mode of ingestion there. Using research by Mena et al (1969), Barbeau (1976) and Cawte & Kilburn (eds:1987), it can be shown however, that these differences are not irreconcilable and cannot be used to rule out manganese toxicity as a cause of the Bird Disease.

The pattern of exposure to Manganese on Groote Eylandt is unique, as are the unique geological conditions

referred to above. It is far more generalised than elsewhere, with a wider range of people being exposed simply by living in the district.

The fact that the manganese mine on Groote Eylandt is open-cut reduces the possibility that inhaled manganese dust could be a major source, as ventilation is not a problem here. Although at 5 mcg of manganese dust per cubic metre of air the Groote Eylandt levels are one hundred times the world average, this is still well below the occupational limit of 200mcg per cubic metre (of course, in a cumulative process even small amounts are important). This dust in the air becomes visible mainly when it falls on the flat surfaces - at any time of day a thin film of black may be wiped off any surface. The possibility of transdermal absorption is probably negligible though, given its low aqueous and lipid solubility (J.C. pers.comm), and is not addressed in the literature. A major point of interest in the literature is the work of Mena et al (1969) which concludes that even when inhaled, manganese must be transferred to the gastro intestinal tract for effective absorption to occur. Thus the possibility arises that if eaten, manganese will still be absorbed - and possibly more effectively if it by-passes the lungs. Thus I believe that it is from water and food that the Groote Eylandt Aborigines derive most of their manganese. The possibility of manganese from the water supply will be addressed first, then the factor of manganese in food.

As seen in photograph 2.5, the Angurugu River, which supplies the whole island with water, runs over an exposed bed of manganese. The possibility of water as a source of manganese arises where there is manganese in suspension in the water. As a result of the chlorination process (which renders the water *microbiologically* safe to drink) the manganese becomes oxidised, turning it into a form more readily available to the human body (M. Florence, pers.comm). In relation to the amounts of manganese present in food grown at Angurugu however, this source pales into insignificance (see Table 2.2)

TABLE 4 MANGANESE IN TRADITIONAL FOOD SOURCES COLLECTED FROM OLD GARDEN AREAS*

Table 2.2. levels of manganese in food.

Sample	ugMn/g fresh weight	
	Angurugu	Sydney
Fish, Angurugu River	36	0.3
Oysters, Mud Cod Bay	0.25	0.05
Yam, young	657	5**
Yam, old	484	5**
Citrus fruit	0.85	0.3
Banana	79	1.5
Billy tea	6.7	-

*U.S. intake: range 2-9 mgMn/day, mean, 3.7 mg/day.
**Root vegetables.

Levels of manganese in food range from five times the world average, in oysters, to over one hundred times the world average in yams (Florence et al in Cawte & Kilburn 1987:33). Yams grown in the mission garden were found to have up to 657 mcg of manganese per gram. In a moderate-sized yam (200g) this amounts to 131 mg - over thirty times the recommended daily intake of 4 mg of manganese. Fish from the Angurugu River have twelve times the world average, bananas from the mission orchard twenty-

six times that. Groote Eylandt Aborigines are thus exposed to unusually high levels of manganese through their food.

The amount of manganese absorbed and factors which affect that absorption, must briefly be discussed. Manganese levels in the human body are difficult to establish and only show recent exposure. Therefore chronic exposure cannot be measured. Manganese levels can be measured in blood, spinal fluid, urine, hair, skin and muscle. Higher levels of manganese are seen in exposed unaffected people than in exposed affected people, both in Groote Eylandt and elsewhere (see Table 2.3). Mena suggests (1979:223) that the metabolisms of unaffected people have a better turnover and excretion rate. It could also suggest that affected patients have a lower *tolerance* for manganese. On Groote Eylandt, blood samples of both unaffected and affected Aborigines, show higher levels than Europeans, reflecting increased exposure to manganese (Cawte & Kilburn 1987:32).

Table 2.3.

TABLE 1 BLOOD ANALYSES ON GROOTE EYLANDT INHABITANTS

Subject	Mn µg/L	Fe µg/L	Hb g/dL	Ferritin µg/L
1. GEMCO workers				
1	22.3	503	16.6	233
2	6.0	454	15.5	280
3	9.9	420	14.2	124
4	9.1	407	14.9	146
5	12.6	498	16.1	165
6(F)	10.7	364	13.3	87
7(F)	6.9	406	14.5	158
8	6.3	495	16.8	93
9(F)	7.7	391	14.1	53
10	6.3	448	15.9	125
2. Caucasians in Angurugu				
1(F)	5.8	419	13.8	-
2	4.9	413	15.1	110
3(F)	8.8	361	12.1	30
4(F)	9.6	359	12.7	67
3. Affected Aborigines				
1(F)	38.7	331	11.3	28
2(F)	36.3	297	10.4	5
3(F)	15.7	356	11.4	0.3
4	42.3	191	8.9	11
5	42.1	-	-	-
6	41.2	-	-	-
7(F)*	9.3	381	13.1	367
4. Unaffected Aborigines				
1(F)	25.5	351	-	-
2	9.9	472	16.4	354
3(F)	16.8	334	12.3	34
4	17.6	453	15.0	96
5	24.9	-	-	-

*Lived in Umbakumba for 15 years.

A third possible source of exposure to manganese is that of foetal absorption - that is, the unborn baby absorbing manganese from its mother's manganese load through the umbilical cord. Normally manganese is only encountered in small amounts (it is a trace element) and the developing embryo absorbs all it can get. Neither at the foetal or the neonatal stage does the body have an excretion mechanism for trace elements (Cawte 1985:214). In situations like Groote Eylandt, where such high levels of manganese is present in food, it is possible for the unborn baby to absorb far more manganese than it ever needs. Studies of blood samples from umbilical cords are currently being undertaken at Gove Hospital where most women on Groote Eylandt are sent to have their babies.

Certain factors may affect the absorption of manganese in exposed populations. Mena et al note the role that malnutrition may play in the metabolism of manganese (especially that of iron deficiency as mentioned previously) and note the increased possibility in "a mining population which has sub-optimal nutritional standards and is also prone to alcoholic excesses" (1969:1005). This picture is seen in close reflection on Groote Eylandt.

There is then strong evidence in favour of manganese playing a causal role in the Bird Disease. Its symptoms and form of brain damage do not exactly match those of classic manganese toxicity, however I have demonstrated that the causes and effects of manganese toxicity are not as clear cut and they may appear. Further

interesting considerations arise when research into the field of slow viruses is taken into account.

SLOW VIRUSES AND RELATED RESEARCH

In any neurological degenerative disease in which the aetiology is unknown, the possibility exists that it may be due to a 'slow virus' infection. Slow viruses may exist in the body for some time before the symptoms become apparent. This latency period makes it difficult to establish a direct cause, as the disease may occur at a minimum of one year and up to several years after contact with the virus occurred. Kuru, a neurological disorder that affected people in the Fore villages of the New Guinea highlands is one such example. It is interesting to compare with the Bird Disease because for some years Kuru was also a mystery disease and eventually it was the collaboration of medical and anthropological research that was responsible for solving the mystery of its cause. At first Kuru was thought to be a genetic disorder. Detailed anthropological research showed however that the pattern of disease distribution was the direct result of food sharing customs practised in cannibalism. Matthews et al (1979:76-77) provide a detailed summary of the articulation of epidemiological and ethnological data in the case of Kuru. This may be seen in Appendix two.

To avoid confusion, it should be stated here that the Bird Disease has not been attributed to a slow virus in the literature. The concept of the slow virus attains its

relevance to the Bird Disease in the peripheral discoveries that slow virus researchers have elicited and the precedents that these set for the Groote Eylandt case.

The anthropological link is not the only interesting correlation which Kuru has to the Bird Disease. Carleton Gajdusek and his colleagues have followed up the physiological mechanisms in Kuru and linked it with a 'disease complex' containing such diseases such as Creuzfeldt-Jakob disease (a disease leading to premature senility) and Scrapie (sheep encephalopathy). All these result from spongy degeneration of the brain. The fact that such a disease complex exists, where there may be different manifestations of the same form of brain damage, stemming from the same cause is, I believe, highly interesting and relevant in the Groote Eylandt context. Moreover, although Creuzfeldt-Jakob disease is principally caused by slow virus transmission, Baron et al (1986) have suggested that it may occur familially, in an autosomal dominant pattern.

The Kuru/Creuzfeldt-Jakob/Scrapie disease complex is not the only one with relevance to the Bird Disease. Parkinson's dementia and Amyotrophic Lateral Sclerosis occur together in Pacific regions such as Guam, the Kii peninsula of Japan and in the Aiyu and Jakai people of Western New Guinea. Given the Parkinson-like degeneration of classic manganese poisoning and the ALS manifestation of the Bird Disease, it is particularly interesting that such a linkage is possible.

There are also some similarities in the environments of Groote Eylandt and the Pacific areas mentioned above. Garruto and Gajdusek have put forward a hypothesis (1985) that the markedly low calcium levels in the regions lead to hypoparathyroidism, a condition that causes increased Central Nervous System and intestinal absorption of heavy metals such as manganese and aluminium. Groote Eylandt is also very low in calcium, including the Angurugu River. The Angurugu River rises in the central hilly district of sandstone and quartz and is particularly low in calcium (M.Florence personal communication). Medical records on the island indicated that some patients have presented to the health centre with tetany - the result of low calcium in the body. Levels of calcium have not as yet been measured in the course of research into the Bird Disease, but would be of benefit. Given the low levels of calcium both in the environment and in the diet[6] of Groote Eylandt Aborigines, it seems likely that Garruto and Gajdusek's theory would hold up on Groote Eylandt as well. Thus theory from the field of slow virus research sets some interesting precedents for the Bird Disease.

The existence of 'disease complexes' and the linkage set up between A.L.S. and Parkinsonism Dementia is of particular use, as is the genetic inheritance of what is supposed to be a transmissible disease (Creutzfeld-Jakob disease). Perhaps it should be unsurprising therefore that the ALS-PD complex of Guam has also been linked to a

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[6] A list of groceries observed being bought on shopping day is presented in Appendix Three.

genetic pattern of inheritance. Stern (1973:863) quotes Kurland, who worked on the Guam case, as postulating a pattern of genetic inheritance characterised by incomplete penetrance to explain the distribution of ALS-PD on Guam. Stern goes on to suggest that ALS and PD on Guam is possibly the result of a 'genetically controlled susceptibility to virus infection'.

To sum up; theory from slow virus research creates a precedent for applying the idea of the 'disease complex' to the Bird Disease, indicating the possiblity of different symptoms stemming from the one cause of brain damage. Also, the possibility of genetic transmission of such a disease-complex is foreshadowed both in the case of Guam, and the Kuru/Creuzfeldt-Jakob/Scrapie complex. What the discoveries teach the researcher most effectively is not to be too quick in ruling out a possibility because of surface dissimilarities. One last possibility that may in fact be ruled out, but must be discussed, now follows. It is the possibility of cycad (Burrawang) nut poisoning.

CYCAD POISONING

Recent research in the ALS/PD complex of Guam, Japan and New Guinea (Horizon video, BBC 1989) and the Bird Disease (Cawte 1984) suggests that cycad nut (Burrawang) poisoning may be implicated in the cause. I do not consider this to be a strong possibility for two reasons. Firstly, the type of washing process used to detoxify the nuts is different in Groote Eylandt. In Guam, the seeds are soaked in bowls

for a long time and the water changed at intervals. On Groote Eylandt running water was used to wash the poisonous sap out of the nuts and away downstream. This was repeated at each step of the process of turning the nuts into flour for damper (Tindale 1925:77, Levitt 1981:49). Secondly, wheat flour, brought by the missionaries and then available from the store, which opened in 1970 has largely superseded burrawang flour as a basis for the staple food of damper. It is comparatively cheap and involves considerably less effort. (Also, the large storage tins - 16kg - are extremely useful once the flour they contain has been used.) It is mainly for these reasons that the case for cycad poisoning on Groote Eylandt is very thin.

In this chapter I have presented the medical details of the Bird Disease and the theories that have been postulated regarding its cause. I have also presented some research of my own (mainly concerning the genetic information) as a result of fieldwork on the island. In the third and final chapter, I shall present my theory of the cause and articulation of the Bird Disease by placing these medical details in the wider social context that was presented in Chapter One.

CHAPTER 3

ANALYTICAL CONCLUSION

In the previous two chapters I have presented data relevant to the Bird Disease, and discussed the theories of its aetiology. In this chapter I shall present my conclusion of the aetiology and mechanisms of the disease. I will argue that the two approaches I have explored in reaching this conclusion - the social and the medical - are inextricably intertwined, and should be understood as such in order to arrive at an effective resolution. Some pragmatic suggestions will also be made with the aim of reducing the number of predisposing factors.

My theory is that the Bird Disease is caused by a genetic susceptibility to manganese toxicity. This was manifested after a concentrated period (of some years) of eating manganese-rich food from the mission garden. The signs and symptoms are a little different from those of classic manganese toxicity possibly because the type of exposure to manganese on Groote Eylandt is different from other places where manganese toxicity is known to have occurred. Data on the physiological effects of manganese reveal one consistent result of toxicity, which is dystonia, and although the Bird Disease has two different manifestations, dystonia is a feature of both.

The high levels of aggression seen on Groote Eylandt are not directly correlated with the Bird Disease, although the psychomotor aspects of manganism might lead one to believe that this is the case. Chronologically it is ruled out as high levels of aggression were manifested on Groote Eylandt well before 1940, for example in the killing of the Japanese in the 1930s. Nor is such aggression restricted to those families susceptible to the Bird Disease. Although, as a generalised phenomenon it might reflect higher than average manganese exposure, other causes are probably also involved. Varying amounts of tension are seen whenever the families settled in close proximity. It seems that close proximity combined with the practice gerontocratic polygyny was the major provocation of violence. As Turner says:

> Much of the unrest reported during the early years of settlement life appears to have been occasioned by the prolonged contact ... with old feuds rekindled and new opportunities for wife stealing. (1974: 15).

Other, recent sources of violence are the twin addictions of petrol sniffing and alcoholism. It is unlikely therefore that there was historically an unduly high level of aggression on Groote Eylandt, nor could the levels that exist be successfully linked to central nervous system initiation by manganese. Thus manganese toxicity is unlikely to be a major cause of the widespread aggression and hyperexcitability, but this does not corrupt the link between manganese and the Bird Disease.

Manganese has always been present on the western side of Groote Eylandt, especially in the region of Angurugu. Yams grown in this area must always have had higher than normal concentrations of manganese. However, two factors prevented the picture of absorption and toxicity that has been the case since the 1940's. The first factor was the constant movement throughout clan territories that was associated with food availability. Thus while some of the year may have been spent in areas that contained food high in manganese, other periods of time were spent in areas that did not, so historically the manganese intake would have been lower than today. The second factor was that the families who show the disease did not live in the most manganese-rich area of Groote Eylandt.[7] When the mission was moved to Angurugu, these factors ceased to operate. Firstly, the population at Angurugu became largely sedentary and food from the mission garden was consumed all year round. Secondly, the families who have the Bird Disease moved at that time to the mission at Angurugu and settled there. It is possible to apply a time line to the genealogies and confirm this argument.

A critical point in the link is the historic absence of the disease despite the high environmental

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7 Some confusion arose here in my final analysis because family 3 is known to have come from Bickerton Island, which also contains high manganese levels. However, as their move to Groote Eylandt is estimated to have occurred over 100 years ago (Turner 1974:10) this may not be as important as it at first appeared.

manganese. The genealogies indicate the absence of the disease in people of earlier generations. As stated before, neither health department records, mission records or anthropologists who visited the island (Tindale, Worsley, Turner and Rose) mention, nor can locals recall any such disease. I wrote to Rose giving the details of the Bird Disease and asked him if he could recall any such disease on the island while he was there, and he replied that he was not aware of any such condition. It seems safe then, to say that the first case appeared in 1951 and cases have occurred sporadically until the present (see Table 2.1). Not all of the people with the ataxic form developed it at the same age, or following the same period of exposure. In classic cases of manganese exposure the timing and response to toxicity also varies. The major point to be made here is that the first cases only appeared after vegetable gardening had been established at the Angurugu mission, in the early 1940s According to Worsley (1954) gardening was a major activity at Angurugu when he was on the island in 1952, and it appears that gardening was carried out approximately until the beginning of the 1960's. There was thus about twenty years of exposure to high dietary levels of manganese.

Another pattern which is obvious from the genealogy is that the younger generations suffer exclusively from the ALS manifestation of the disease. This suggests the possibility of foetal absorption and retention of manganese, as the ALS type is congenital.

The genealogies do not only provide a reference point for the time frame of the appearance of the Bird Disease - they also provide the picture of the genetic nature of the disease. Indeed, it was through an elaboration of the genealogies by means of field research that I was able to take the genealogy of Family One back a generation and establish the two genetic links with Family Three. This seems to strengthen significantly the case for a genetic basis, which is useful as the statistical base is so small.

On this basis it can be predicted that the number of cases will remain small, and even if new cases appear, that they will be confined to these generations, because the disease is a result of the combination of dietary change and constant exposure to manganese in people with a genetic susceptibility to its absorption. If this is the case and that no more (or very few) cases appear then perhaps we shall never be able to establish precisely the form of genetic inheritance. The pragmatic recommendation that I would make relates to an observation made in the first chapter. It appears that the fruit and yams grown near the mission garden are still eaten by the Angurugu inhabitants. If people in family one or family three, especially those who are connected by marriage to family four, were to refrain from eating food grown in such manganese-rich areas as the mission garden, then the possibility of manganese toxicity may be forestalled, and the consequences avoided.

The role of malnutrition, especially of anaemia and calcium deficiency has not been forgotten here. Undoubtedly it would be impractical to remove all Groote Eylandt residents for iron or calcium treatment, perhaps short term treatment of those most at risk, undertaken off the island, might address some of the problem.

The diagnosis of the Bird Disease is clearly provisional, yet it underlies my contention that one must look further than the biomedical base in order to approach fuller understanding of the process of disease. Indeed it was through Anthropological data rather than medical reports that I was able to examine the recent social history of Groote Eylandt and the articulation of kinship and genealogical data.

The separation of the social from the medical in the construction of this thesis may cause the reader to recall the mind-body split of Cartesian Dualism. (This dualism is a theme frequently discussed in the context of medical anthropology). That implies however, a dialectic base, which I feel is inadequate in many situations. At what point can one break into a whole and manufacture arbitrary opposites? It would be impossible to assign either the social or the medical to an antithetic position since they are complementary. They should perhaps be seen as positions on a continuum. The most effective position in relation to this continuum is one from which information from each sector can be absorbed and utilised. Thus in the Bird Disease medical information and knowledge is necessary to build

a picture of symptoms and causation, but only knowledge of the ethnography and recent social history of Groote Eylandt can fit the final piece in the jigsaw and explain how it was possible to develop the disease. Reid (1982) also approached the issue of medicine and society in a discussion on Aboriginal Health Centres and Medical care:

> ...The crucial question is: Do health services deliver health? Most doctors and nurses who have worked in Aboriginal communities, not to mention the Aborigines themselves, know they do not. Physical and emotional well-being also depend on the adequate provision of water, power, sewerage, housing and education, and on less tangible but equally important factors such as social cohesion, cultural integrity and financial support (XIV).

There is one major deficiency in the literature on the Bird Disease, and that is the lack of an indigenous explanatory model. Cawte (1984) and Turner (1989) are the only two who address it. Theory within medical anthropology emphasises the existence of the explanatory model of the patient (Young 1982; Capra 1982, Engel 1984). The rationale for the emphasis is that illness and disease do not happen in a vacuum. It happens to people, and affects their lives and general outlook. Naturally a model will be formulated by the affected people that explains their interaction with the disease as well as ideas of its cause. If an effective resolution is to be formulated then the investigator needs to listen to the affected people (and to the surrounding population). It is no use recommending ways

in which to alleviate a problem if the affected people do not agree with the diagnosis. Torrey (1972) relates the effectiveness of a practitioner to four criteria, the first being that both healer and patient should have a similar view of the cause. (The other criteria include the personal qualities of the healer, the expectations of the patient and the patient's acceptance of the techniques used in the healing). With regard to the articles of Cawte (1984) and Turner (1989), it appears that this criterion is not met in the Groote Eylandt context. Indeed, according to Berndt (1982) and Reid (1982) this appears to be a general problem with health care delivery in most Aboriginal communities. Devenesan (1983) advocates a 'bicultural' approach to medicine and health. In concurrence I would suggest that unless indigenous conceptions of health and disease prevention are taken into account and integrated into a model of health care delivery, health centres will not be able to do more than chip away at the edges of Aboriginal health problems.

Indeed, the whole issue of health-seeking behaviour on Groote Eylandt is an area which urgently needs to be studied, not only in the context of the Bird Disease, but in view of the wide range of health problems experienced there (diabetes, middle ear infection, hookworm etc). Myra Tonkinson has addressed this in the Western Desert in her study of selection of treatment, from traditional healers or Government Health Centres (in Reid 1982)

GLOSSARY

Aetiology: The cause of a disease.

Allele: One of two or more alternative forms of a gene at the same site in a chromosome.

Ataxia: Failure of muscular coordination.
Autosomes: Chromosomes that are not linked to sex determination.

Brain Stem: the stem-like portion of the brain connecting the cerebral hemispheres with the spinal cord, and comprising the pons, pallidum, medulla oblongata and midbrain. It is here that the trunk nerves cross over and produce the phenomenon where one side of the brain controls the other side of the body.

Bulbar Paralysis: Due to changes in motor centres of the medulla oblongata, marked by progressive paralysis and atrophy of the lips, tongue, pharynx and larynx.

Cerebellum: 'Little brain'; is attached to the brain stem, and coordinates the muscles in voluntary movement.

Cerebral Cortex: The convoluted layer of grey matter covering the cerebral hemispheres, which governs thought, reasoning, memory, sensation and voluntary movement.

Chromosome: A supercoiled linear thread of DNA which transmits genetic information.

Congenital: Any condition that is present at birth.

Dystonia: Impairment of muscle tone.

Gene: A segment of DNA which codes for a particular characteristic.

Genotype: The entire genetic constitution of an individual- also, the alleles present at one or more specific loci on the chromosome.

Heterozygous: State in which the two alleles at the same site on the chromosome are different.

Homozygous: State in which the two alleles at the same site on the chromosome are the same.

Incidence: The number of new cases of a specific disease within a specific period.

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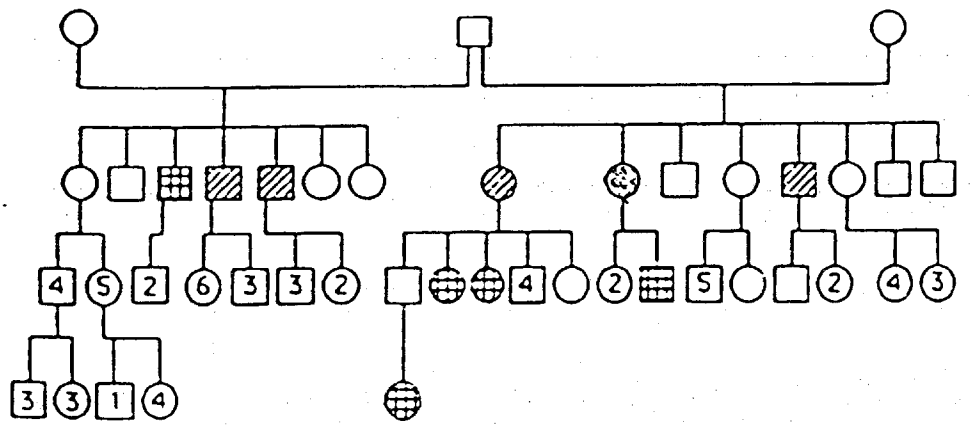
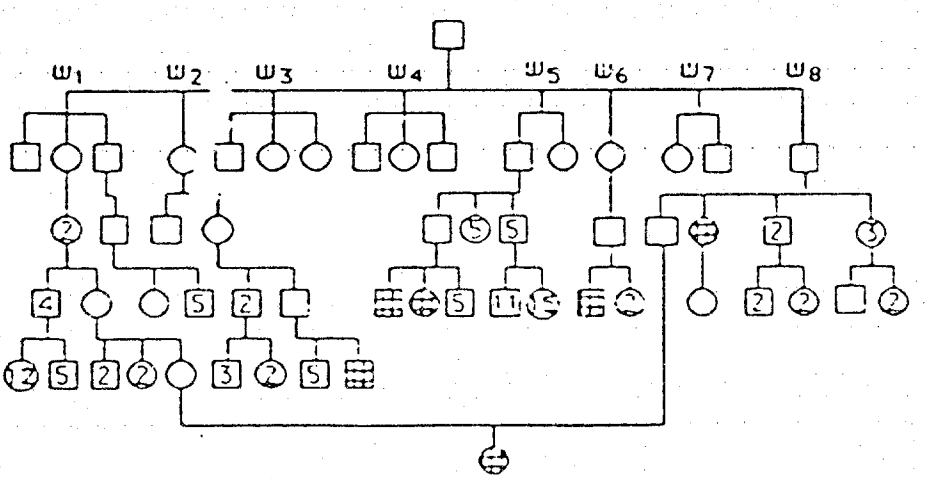
Appendix One

Genealogies from Kilburn (Cawte and Kilburn 1987)

A



B



A. Condensed pedigree of family 1.
B. Condensed pedigree of family 2.
Enclosed numerals represent numbers of offspring.
In B, "W" with subscript indicates wife number. None of the progeny of the offspring of wives 3, 4 or 7 are affected and have been omitted.

Unaffected male Unaffected female

Case with amyotrophic syndrome

Case with ataxic syndrome

Unclassified neurological disorder

APPENDIX 2

Summary of Ethnographic and Epidemiological Evidence Linking Cannibalism and Kuru Transmission

Epidemiological or genealogical observation	Ethnographic explanation	Comments or deductions
is a familial disease	Cannibalism was a matter of kinship; certain dead relatives were eaten for gastronomic reasons, or to show respect. Enemies occasionally eaten too	Familial aggregation of disease does not necessarily indicate genetic determination
is common in women, but rare in adult males rizontal transmission)	Cannibalism by adult males was rare, especially in the South Fore; they avoided female flesh and male kuru victims. Women commonly ate kuru victims, and more often consumed brain and visceral material than did men	Adult males were likely to consume little of kurugenic agent, and it is possible that only high titre brain and visceral tissues would consistently transmit the disease after cooking (see text)
presents in the children of women who die from the ase (vertical transmission)	Young children often shared at cannibal feasts with their mothers	Vertical transmission to children could be just a special case of horizontal transmission by cannibalism, but a more biological type of vertical transmission is possible, perhaps to give kuru of longer incubation period (see text)
is more common in the South Fore than in the North e	There was more resistance to the eating of kuru victims in the North Fore	Early reactions to the appearance of the disease gave way to the widespread belief that kuru was due to sorcery
ly, the kuru epidemic spread slowly from village to age, sometimes with a latent period of 5 years or re	At first there was some reluctance to eat kuru dead even in the South Fore	Incubation period likely to be up to 5 years or more; agent unlikely to spread readily other than by cannibalism, or if it does, unlikely to cause either immunity or kuru
st kuru victims in any place were often women who come as wives from a previously affected village	Such immigrant wives would be likely to eat kuru victims in the home village either before marriage or on a return visit	
could spread into a previously affected village if an grant wife developed kuru elsewhere	Maternal kin (in the home village usually) had rights in the body after death	One can account for the tempo or "explosive nature" of the epidemic by postulating that 3-6 secondary cases followed 4-10 years after each preliminary case

plosive kuru epidemic had developed in the South e by the 1940s and 1950s	Consumption of kuru dead had become more general and many females were likely to eat each victim	
es affected later in the kuru epidemic tend to have a er incidence of the disease	Cannibalism was suppressed at an early stage of the local epidemic	Low incidence due to limited opportunities for kuru cannibalism
is a decreased incidence of kuru across linguistic and ural boundaries	There is less intermarriage and shared cannibalism across such boundaries	Could also be due to the importance of genetic factors in predisposing to kuru
was no kuru amongst the Awa women unless they rried into Fore villages	The Awa were probably not cannibals, but Awa wives in Fore villages could have taken part in kuru cannibalism	Suggests that genetic factors are of little importance in determining the relative incidence of kuru in the area
w-incidence areas, the risk of kuru in wives of thers of kuru index cases is higher than the risk for tives of control proposti, and is almost as high as t for mothers, sisters and daughters of the same kuru ts (see fig. 2)	The victim's mothers, sisters, daughters, and brothers' wives were likely to have had similar opportunities for kuru cannibalism	As for item 11
about 1960 there has been a general decline in the idence of kuru, along with a definite increase in the rage age of people dying from kuru. There have been kuru deaths in anyone born since about 1954	Cannibalism was largely suppressed in the 1950s due to the activities of administration and mission personnel	Transmission of the kurugenic agent (at least to give kuru of a shorter incubation period) may have ceased with the suppression of cannibalism. If a hypothesis, most cases of kuru seen now must have been incubating the disease for 10-12 years or more; the increase in the average age of victim reflects the ageing of cohorts exposed 10 or more years ago
imi have yet to show such a significant decline in u incidence, and hitherto there has been no increase he age of kuru victims	Cannibalism probably persisted for longer among the Gimi	Surreptitious cannibalism was harder to detect in the Gimi and other isolated areas
oungest kuru victims currently observed come from lated hamlets	Cannibalism was probably practiced for longer in these places	
al men over 40 years of age have been seen with kuru ently and rare cases of kuru in old men have been orted by native informants	Adult men have occasionally eaten kuru victims; in South Fore those of fighting age were most likely to abstain	The disease in middle-aged men could represent kuru of long incubation period following vertical transmission in the 1920s, or it could be due to occasional kuru cannibalism by the men. The disease in old men is almost certainly due to the latter

APPENDIX THREE

'Basket of Goods' Angurugu.

EVERYONE	MOST PEOPLE	SOME PEOPLE
Sugar	Tinned vegetables	Fresh meat
Flour	Tinned meat	Fresh vegetables
Cordial	Biscuits	Fresh milk
Smoked oysters	Rice	Orange juice
Smoked mussels	Cigarettes	Nappies
Tea	Frozen cakes	
Dried milk	Tinned meals	
Bread	Soap	
Coca cola	Toilet paper	
	Laundry powder	