Health, disease & variation

A Biomedical and Genetic Analysis of the Orang Asli of Malaysia

A. Baer

COAC

HEALTH, DISEASE, AND SURVIVAL

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Dedication

To
IVAN POLUNIN
the *batin* of biomedical studies
on Orang Asli

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AIRPLANES DON'T FALL OUT OF THE SKY

When I was getting ready to leave Malaysia for the second time, a Temuan woman said to me, "Don't be afraid of getting on a plane at Subang. Airplanes don't fall out of the sky."

This statement told me two things. She had been to far-away Subang airport, which I hadn't known. And she felt I didn't want to leave. I rationalized that my work on Temuan biomedical issues, especially genetics, was not finished.

That was in 1972. Since then both the Temuan and their world have changed, probably more than I have. As a visiting American biologist, I was first introduced to the Temuan, Jah Hut, Semai, Temiar, and Besisi by friends in Malaysia in 1967. I have remained a student of the indigenous peoples of Malaysia ever since.

But, of course, airplanes do fall out of the sky. My Temuan neighbor in 1972 didn't know this. Nor did she know what a geneticist was. Neither of these facts mattered in the context of life in a Temuan village then. Today, however, Temuan people are much more aware of the behavior of airplanes, television boxes, road-building machinery, and AIDS victims – for better or worse.

The story of the health of Temuan and other Orang Asli groups of West Malaysia amidst the changes in their homeland over the past centuries and decades is the subject of this book.

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Many people over the years have helped me toward the conception and realization of this biomedical review project. I remember them all with appreciation.

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For those who kindly reviewed various parts of the manuscript, I am particularly indebted.

Discussions between reviewers and myself on "world views" helped me to broaden my vision of the dimensions of health for Orang Asli.

Since I did not take all the advice generously offered, the errors that remain are patently my own. This is particularly true for the cases where good data are absent; there my conclusions may reflect my assumptions.

Table 1
Orang Asli biomedical studies:
Estimated number of reports by topic and by group

	General	*Demography*	*Filariasis*	*Genetics*	*Goiter*	*Worms*	*Malaria*	*Mental Health*	*Nutrition*	*Schistosomiasis*
O. Asli	75	7	12	45	5	7	24	4	15	5
North	4	1	1	8	1	3	1	1	1	1
K. + K.	0	0	0	4	1	0	0	0	0	0
Jehai	1	4	2	2	1	1	2	0	1	0
Batek	2	0	0	0	0	0	1	0	2	1
Senoi	4	2	0	5	1	0	1	0	0	0
Temiar	4	1	3	4	2	1	7	2	3	1
Semai	7	8	5	20	1	3	11	4	8	0
Besisi	1	0	2	1	1	0	1	1	1	0
Semelai	5	2	3	6	0	1	3	1	1	2
Temuan	11	3	5	8	2	7	11	1	5	2
Jakun	2	0	2	6	0	1	1	1	1	0
Others	5	1	4	3	1	1	2	1	2	0

The "General" category includes poorly-studied topics such as cancer, dengue, and tuberculosis. The "O. Asli" category includes unidentified groups and conglomerated studies. "North" refers to northerners in general or to Lanoh. "K. + K." stands for Kensiu + Kintak. "Others" refers to small central or southern groups. "Worms" refers to intestinal helminths. Some reports are counted in more than one category.

PREFACE

In this book I present and evaluate biomedical and genetic findings published on the Orang Asli, the heterogeneous "original people" of the Malay Peninsula. My aim is to sketch out what is known and what needs to be known—for readers concerned about the welfare of the Orang Asli, for those engaged directly in basic science, and for those interested in Orang Asli biocultural histories. The book may also serve as a model for describing the health dimension of the worlds of other indigenous peoples.

I have generally taken a group-by-group approach. This allows a reader interested in a particular group or locale to obtain basic information on that microcosm. The caveat here is that I was forced to lump some groups together because of data deficits. I did this lumping on the basis of proximity, languages, and cultures. This gives four group-based chapters: northern Orang Asli, Temiar plus Semai, small central and southern groups, and Temuan plus Jakun.

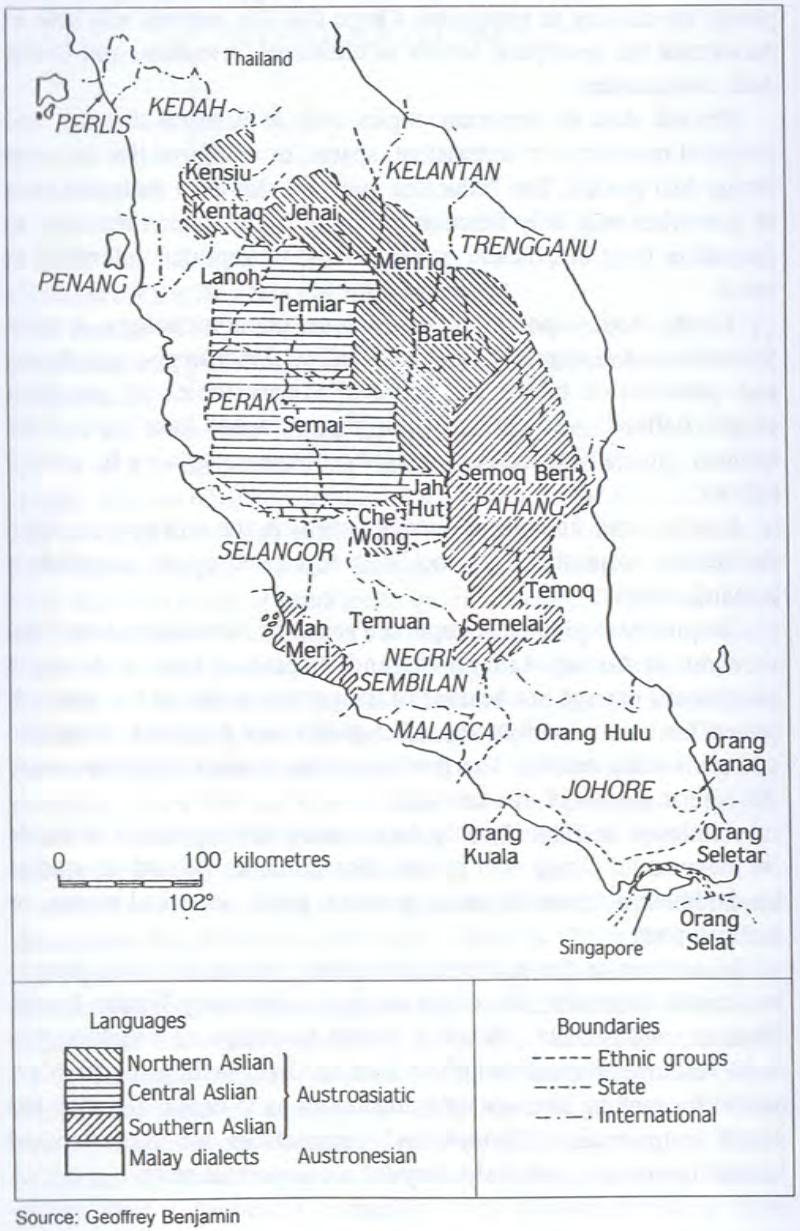
In addition, I have presented some comparative data on important issues for all Orang Asli in separate chapters, in cases where information is ample. Thus I discuss malaria (and relevant genes), nutrition, and general health history in chapters 7-9. Rarer comparative data are presented in the group-oriented chapters, as particular topics come to the fore. Overall, then, the book's format provides both a group focus and, to some extent, a disease (or problem) focus.

The information on Orang Asli groups generally follows a chronological scheme. Among other advantages, this approach highlights what was known and unknown in the past and what remains unknown today. It leads easily to a consideration of current problems and future prospects.

Before considering the substance of published reports on the Orang Asli, some deficiencies in the biomedical literature need to be mentioned.

First, the majority of reports listed in the biomedical bibliography (see appendix) are not on subjects identified as to ethnic group. Rather, they tend to be on a mixture of patients at Gombak Hospital (started in 1960 as a hospital exclusively for Orang Asli), on other mixed samples, or are literature reviews. This inattention to ethnicity is unfortunate since the various groups live in diverse ecosystems, have diverse coping mechanisms for dealing with illness, differ in their

ORANG ASLI GROUPS OF PENINSULAR MALAYSIA
(Approximate distribution)



Source: Geoffrey Benjamin

genetic histories, and live at unequal distances from Gombak (and so are not proportionately represented in hospital samples). Moreover, the art of doctoring is only one part of the science of health, as many people are coming to appreciate. I hope that this analysis will help to harmonize the perceptual worlds of clinicians, scientists, and Orang Asli communities.

Second, data on important topics such as venereal diseases and perinatal mortality are nonexistent, sparse, or uninformative for most Orang Asli groups. The "educated guesses" that exist on topics such as gonorrhea may only function to obscure. The human tendency to generalize from insufficient evidence must be kept uncomfortably in mind.

Third, most reports are focused on phenomenology, a vital foundation of research but not its totality. Accordingly, hypotheses and questions on biomedical and evolutionary topics are not often clearly defined. And perhaps some reports would have enjoyed the salutary effects of being peer-reviewed before acceptance by journal editors.

Lastly, some findings are unconfirmed, in the strict sense, since the results in question have not been replicated by an independent research effort.

Despite these problems, important scientific conclusions have been reached on Orang Asli health and disease. These wide-angle conclusions emerge not because of the perfect design of any research project but because various imperfect studies have produced compatible and reinforcing results. The results leading to these conclusions are the core materials of this analysis.

As shown in Table 1, only fragmentary information is available on many small Orang Asli groups. For example, I found no studies on the Batek in terms of cancer, genetics, goiter, intestinal worms, or scrub typhus.

In contrast to the scattered, tantalizing reports on most groups, substantial information does exist on others—especially Temiar, Semai, Temuan, and Semelai. While it would be remiss to conclude that these resources provide definitive facts on Orang Asli health, they are useful for making inter-group comparisons as to health histories and health status today. Although such comparisons are imprecise and should be treated cautiously, they tell an important story.

Chapter 1

INTRODUCTION

GLIMMERINGS AND GENERALITIES

Two reports sketch the health problems in Malaysian indigenous peoples several generations ago, foreshadowing the concerns of more recent decades.

In 1931 some 260 Temiar in the Ulu Plus area of Perak state, mostly men, were judged to be "fairly healthy" by a visiting medical worker. But the tabulated details tell a different story. Some 23% had ringworm, 20% had enlarged spleens – suggestive of endemic malaria, 11% had neuralgia, 8% had scabies, and 5% had wounds. There were scattered cases of bronchitis, sprains, ulcers, dyspepsia, dental caries, and boils. In addition, 9% of the Temiar living in a nearby area had Ascaris worms, and cases of yaws were noted elsewhere (Noone, 1936). Besides this 1931 report on Temiar illness, the "Medical Annual Report for the State of Kelantan" for 1933 noted that 119 Temiar along the Nenggiri River were vaccinated against smallpox, that a few had yaws or skin diseases, and none had goiter. More forebodingly, childhood fevers and enlarged spleens were common (Noone, 1936) (endnote 1).

The Temiar are one of the ethnolinguistic groups dispersed throughout West Malaysia collectively called the Orang Asli (see Box and Figure 1) (endnote 2). Orang Asli differ from other communities in West Malaysia in many ways, besides the fact that they are a small minority of the whole population. They have been residents on the Malay Peninsula for millenia, much longer than other groups. Most of their languages are unrelated to Malay, the official language of the country. By tradition, Orang Asli religions emphasize ways to ensure health and survival; and they are their own religions, not being Islamic or any other major type. Genetically, Orang Asli are distinctive from

other Malaysian groups in important ways. Also, their traditional economies are simple (mainly foraging, fishing, and horticulture), so that their impact on the environment is far less than that of tin mining, agrobusiness, or six-lane highways.

A biomedical overview for West Malaysia, provided by Lim (1993), considered demography, pregnancy, nutritional disorders, infections, and systemic diseases. He mentioned Orang Asli only in passing, perhaps because information on them has been piecemeal and dispersed. However, serious study of Orang Asli health began in the 1950's when Polunin carried out medical surveys on several Orang Asli groups, uncovering many health problems. At that time facilities were not available for conducting much in the way of laboratory or therapeutic studies. All that had to await a greater governmental awareness of health needs, a process that is still unfolding.

Polunin's surveys in the 1950's stimulated interest in the biomedical and bioevolutionary aspects of Orang Asli life. Much was learned and written by a wide spectrum of investigators. Their findings show that the Orang Asli health experience is somberly different from that of other Malaysians. This difference is related to the environment, lifeways, political powerlessness, and genetic makeup of Orang Asli – that is, to the biological and social context of their existence. The following list summarizes a few comparative points, some more important than others (note 3 gives details):

1. Orang Asli had an incidence of tuberculosis two times the national average for the period 1951 to 1971, but by 1995 the rate for Orang Asli children, at least in the state of Perak, was three times the state's average.
2. According to a 1977 report, 89% of Orang Asli tested had had malaria at some time in the past.
3. For the years 1991 and 1992, Orang Asli averaged 48% of all malaria infections recorded in West Malaysia, while making up less than 1% of its population. In more recent years they have averaged over 70% of West Malaysian cases.
4. The crude death rate for Orang Asli, a common measure of mortality, was 1% per year for the period 1984-1987, twice as high as the West Malaysian average (0.5%). The corresponding infant mortality rate was 5.2% for Orang Asli,

Who are the Orang Asli?

The Orang Asli are the original peoples of the Malay Peninsula. Numbering over 90,000 in 1996, they comprise at least 18 distinct cultural-linguistic groups that live in scattered villages and camps, from the coastal mangrove swamps to the rainforest of the central mountain range. They make their living by some combination of hunting, gathering, fishing, farming, arboriculture, selling forest products, producing petty commodities, and wage labor.

Orang Asli groups were politically autonomous until the 1950's. Then the British colonialists placed them under the jurisdiction of a federal Department of Aborigines, in order to win them away from the influence of communist guerrillas operating from forest bases. Laws established to control Orang Asli during this insurrection, which officially ended in 1960, are still in force today, and the Department of Aborigines continues to administer them.

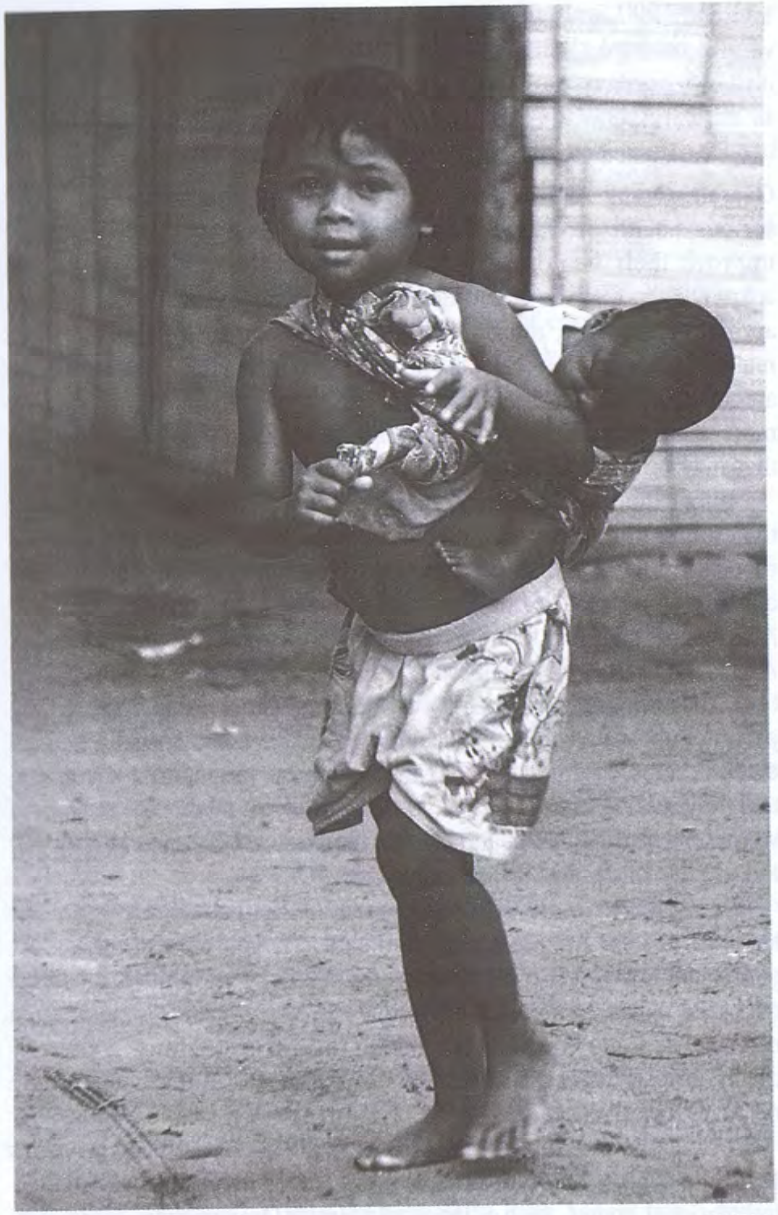
Since Malaysia became independent in 1957, the government has carried out a massive program for developing the land and natural resources of the peninsula. It has generally consisted of logging off the rainforest and replacing it with rubber and oil palm plantations, other industrial farms, dams and reservoirs, and so on. Much of the development has taken place on former or current Orang Asli lands.

Because the government does not recognize Orang Asli rights to their traditional lands, development has usually led to them being dispossessed, as well as to the destruction of the resources on which they depend. The government's solution to this problem has been to settle them in small agricultural schemes where they are expected to support themselves by growing food and cash crops, with minimal government aid and without enough land to support their population. Because they do not own the land they live on, they cannot get loans or assistance from government agencies to improve their economies.

Today most Orang Asli (80%) live below the official poverty line as "the poorest of the poor" (*termiskin*).

more than three times the West Malaysian average (1.6%). While the infant mortality rate for poorer countries, such as Nepal, is much higher than that for Malaysia, the rate for Orang Asli is strikingly high in a local context, given that Malaysia is by no means a poor country.

5. An even more dismal finding was the crude death rate for a northern group of Orang Asli, the Jehai, for the period 1978-1988. It was 4.5% per year, with 59% of recorded deaths being in the 0 to 4 year age group. This Jehai rate is the highest recorded, to date, for any Orang Asli group.
6. According to official records, among Malaysian women giving birth at home in 1994, 42 died; 60% of these (25) were Orang Asli.
7. On the one hand, malnutrition is common in many Orang Asli localities and groups. On the other hand, diseases of affluence are rare. According to a 1995 study, Orang Asli women are the most malnourished adult group in West Malaysia, with 35% of them having protein-energy malnutrition. For Orang Asli children, recent studies find 23% to 68% are underweight while 41% to 80% are stunted in their growth.
8. By serological tests, 82% of Orang Asli showed prior exposure to dengue-virus illness, a much higher percentage than found in urban Malaysians.
9. In 1994 leprosy was 23 times more prevalent in Orang Asli than in the general population.
10. Cultural differences are also important in terms of health. For example, in neo-Malaysian society the basis of social organization is occupational specialization, while in Orang Asli society the basis is kinship. Kinship ties are reflected both in work strategies and residence patterns. The mutual concerns and obligations of kin provide the Orang Asli with their first line of defense against illness, as well as against food shortages and other crises. According to one medical investigator writing in 1997, physicians are unaware of the social and cultural practices of Orang Asli, thus vitiating their chances of improving Orang Asli health.



Colin Nicholas

Young Semai baby-sitter, Tenau, Southeast Perak.
Compared to other Malaysians, the Orang Asli are more likely to suffer serious health problems. The tuberculosis rate for Orang Asli children in Perak, for example, was three times the state's average in 1995. Ignorance of Orang Asli social and cutlural practices contribute to this situation.

11. In 1990, only 67 of 774 Orang Asli villages (9%) contained a medical clinic, according to Lim (1997).
12. Orang Asli groups are genetically distinctive from other Malaysians, not the least in having substantial inherited resistance to malaria.

Overall, Orang Asli in their Malaysian setting appear to experience serious health problems. Much could be done to understand and improve their health status. At the same time, I recognize and acknowledge that the scientific representations of health and disease are not culture-free, nor do they necessarily coincide with "objective reality" (Good, 1994; Hepburn, 1988). While such representations are in many ways external to Orang Asli life, they are not thereby more authoritative. In fact, they can make simplifying assumptions that may be misleading.

Today, the field of tropical biomedicine covers much more ground than found in the reports on the Temiar in the 1930's or in Polunin's reports of the 1950's. One standard text on tropical biomedicine contains a list of 27 "exotic" communicable diseases found in Southeast Asia, more than are found in any other region except the middle of Africa (Warren and Mahmoud, 1991, p. 1114) (note 4). Notably, both these regions contain Old World tropical rainforest, where parasites abound. The biomedical text discusses these 27 listed tropical diseases along with mental health, genetics, nutritional disorders, and the social setting of health. Unfortunately, it slights the subject of women's reproductive health, which happens to be a critical issue for the vitality of Orang Asli communities.

On a broader scale, it is worth mentioning that disease is not the same as illness. As one Orang Asli said, "If you can eat and work, you're not sick." What a physician trained in cosmopolitan medicine (the kind taught in medical schools) considers a disease state is often disregarded by Orang Asli. Take fevers: an Orang Asli may metaphorically shrug his shoulders in the face of a fever. Fevers are ordinary occurrences.

It is also important to recall that biomedical research and practices depend on the collection of health statistics. Statistics collected by international bodies, such as the World Health Organization, are usually tabulated at the national level. For example, Malaysia reported 50,500 malaria cases for 1990. Even when broken down by state, the foci of Malaysia's malarial burden are not truly revealed. In fact, these foci

are characteristic of specific poor, rural, inland groups, not simply of all groups in such categories. That is, disease rates vary markedly by ethnic group – for both biological and sociocultural reasons. Medical researchers, aware of ethnic-group foci, utilize such communities as the objects of studies on malarial vaccines or on some disease-related hypothesis.

Polunin (1953, p. 68) commented in this regard that the Orang Asli:

> "...have advantages for the student of the health and diseases of populations. One of these is the ease of cooperation with the people...in their dealings with other races [they] find it difficult to refuse requests. This makes them especially liable to exploitation and ill-treatment, as most earlier writers have mentioned. A foreigner who is friendly and who brings wonderful drugs which cure yaws and malaria will soon find himself in a position where many things are possible. Provided he does not force his investigations on his hosts before they have understood and accepted them, and provided he respects without question the beliefs and customs of the people, he will find them extremely cooperative."

Certain Malaysian minorities, including both Orang Asli and groups in Borneo, constitute ethnic-group foci for various diseases. These minorities work many hours outdoors, live in environments that are home to Anopheles mosquitos, and have a range of ailments that make them vulnerable to infectious episodes. Such vulnerability applies especially to the children, who have yet to develop full immunity to malaria and other infections. Despite high malaria rates, these groups do not receive high-quality attention at the national level in terms of anti-malarial programs (note 5). More national-level worry appears to be given to the cardiovascular and other ills of urbanites (note 6).

MISCONCEPTIONS ABOUT ORANG ASLI BIOLOGY AND PREHISTORY

Misconceptions about biology are sometimes held longer by non-biologists than by people working in the science. Misconceptions in old textbooks seem to have an especially long "shelf life," but in science old speculations can wither quickly when tested against new findings. Misconceptions about a population's origin and prehistory are often maintained because they become entwined with social

prejudice and notions of race. Terms such as Australoid, Mongoloid, Negrito, Deutero-Malay, and Proto-Malay that have been applied to some Southeast Asian groups derive from folk prejudices. They are scientifically meaningless, given today's knowledge of the complexities of human prehistory derived from genetics, archeology, and related fields (Fix, 1979). Peter Williams-Hunt alluded to this problem long ago, when he referred to descriptive terms for Orang Asli groups as "horrible jargon" (1952, p. 14).

First of all, these terms are stereotypes, simplistically concealing the diversity found in any human group. A common misrepresentation of the cultural and genetic diversity within groups is to display photographs of individuals "representing" such stereotypes. A recent book by McNeely and Wachtel (1991) pictures a "Deutero-Malay" as a beautiful woman, well coiffured and gracefully posed, with eye makeup and fingernail polish prominently displayed. Pictures of a "Proto-Malay" and an "Australoid," however, show people in poor clothes without beauty-salon treatment. (If the latter two individuals had been well-groomed and dressed up while the former was not, a different message might have been conveyed.)

My second point is that cultural groups and their genes are not frozen in time or space. No one can ever know conclusively if the ancestors of, say, the Malaysian Jehai were in their present locale – or even the same region – 5,000 or 50,000 years ago (note 7). Even if they were in the same place, they undoubtedly took in "strangers" from time to time and also experienced some out-migration.

No group is pure or static in any genetical sense: pure "pureness" would mean that a group was a clone genetically, or at least was extremely inbred (note 8). But genetic studies show that Orang Asli groups – like other human groups – are robustly diverse. Within Orang Asli communities can be found people of B as well as O blood type, people with different enzyme variants, people with different levels of resistance to malaria, and so on and on. Indeed, like many small genetic isolates these communities have some unusual genetic profiles. This significant fact of genetic diversity, as well as its consequences, are discussed further in later chapters.

My third point is particularly important in view of an antiquated and flagrant misconception about the origin of the northern Orang Asli, often misleadingly called Negritos. Despite facile and repeated suggestions to the contrary, Andamanese, Aeta (and some other

impoverished groups in the Philippines), African "pygmies," Australian native people, and northern Orang Asli are not closely related (Cavalli-Sforza et al., 1994; Omoto, 1995).

As common sense might suggest, each of these groups is much more closely related to other groups in their own region than to any group in a different region (Cavalli-Sforza, 1991). While all these named groups happen to have been hunter-gatherers in recent times, at some time in prehistory they may have been seafarers, pastoralists, or even farmers – no one knows conclusively (note 9). But the Andamanese did not get to their islands and the Australians to their continent by walking.

Generally speaking, these named groups happen to have dark skin, but so do other tropical people, such as the Tamils of India. Some are relatively short, but so, in general, are the Inuits. In addition, some contemporary hunter-gatherer groups are left out of this uninformed roundup of "archaic" humanity – such as groups in Sumatra, Borneo, and Thailand. Perhaps surprisingly, the groups with the most distinctive genetic characteristics are all African, including some that are lighter in skin color than "Negritos" and live in the Kalahari desert, not in a tropical rainforest. They are the San (bushpeople) of southern Africa (Cavalli-Sforza et al., 1994).

Various writers have tried to rid themselves of preconceptions about Orang Asli origins and foreign relationships, but not always successfully. Carey (1976) described the "Pan-Negrito" theory (of northern Orang Asli being closely akin to African "pygmies," among others) as being "extremely fanciful" and "long discarded" (p. 12). He then remarked that northern Orang Asli "strongly remind one of East Africans," probably not referring to Ethiopians, and that "many of them look like...the Papuans" (p. 15). After positing that the northerners are related to Andaman Islanders and Filipino Aeta on the basis of "physical appearance" (p. 15), he took an aboutface later. On page 18 he reminded us that "physical appearance is notoriously misleading, relying as it does on the largely subjective impressions of the outside observer." Carey's difficulties provide a heuristic lesson of someone trying to muddle through a topic dominated by "false facts."

As this discussion suggests, anthropologists and others have long tried to demarcate ethnic groups and then to "explain" their existence by various theories. For Southeast Asia, these attempts at explanation

have tended to ignore genetical findings, although they may invoke tenets of population genetics in a speculative fashion. Culture studies, genetics, and the health components of evolutionary adaptation have yet to become mutualistic.

In a subsidiary matter, the notion that so-called Proto-Malay Orang Asli (such as Temuan and Jakun) came to the Malay Peninsula from Indonesia lacks scientific reasonableness. Available evidence points to these groups being genetically distinctive from the miscellaneous category called Indonesian. While these misleadingly named Proto-Malay groups speak Austronesian languages today, as do most Indonesians, it is worth emphasizing that languages and genes do not always coincide. In short, changing one's language is much easier than changing one's genes.

Orang Asli groups such as the Temuan may have once spoken Aslian languages but in recent centuries switched to the Austronesian language of their new neighbors, the immigrant Malays. Carey (1976) opined that the Jakun lost their language "recently," and Benjamin (1985) held that the speakers of southern Aslian languages, such as Semelai and Besisi, were shifting to Malay.

Finally, Orang Asli groups cannot correctly be called tribes. As Carey noted (1976, p. 37), "The concept of a 'tribe' often implies a central leadership, a common chief..." No Orang Asli ethnic group has a chief.

As mentioned in the preface, the next four chapters provide biomedical information on four clusters of Orang Asli groups, demarcated roughly by geography, language affiliation, and traditional economies. Later chapters of the book deal with some major biomedical problems that relate to all Orang Asli groups at present, among them malaria, nutrition, and the provision of health services.

Chapter 2

NORTHERN ORANG ASLI GROUPS

In this chapter I survey the health research, demography, genetics, and biological history of the northern Orang Asli. For these topics, the majority of work took place in the period 1970 to 1985, as shown in the accompanying timeline (Timeline No. 1). The dearth of more recent work is a key problem. After this survey, I consider the northerners' health status and the possibility of improved health in the future.

Among the northerners I include Kensiu, Kintak, Jehai (or Jahai), Batek, Mendrik, and Lanoh. They live today in the states of Kedah, Perak, Kelantan, and Pahang. Northern groups historically were forest hunter-fisher-gatherers or small-scale cultivators. As such, they used the environment extensively, not intensively, thereby sustaining it. Many northerners have now been administratively moved to setttlements designed for some cash cropping or wage labor.

According to official figures, there were only 2,364 northerners in 1980 and possibly 194 more in 1994 (Razha Rashid, 1995). Batek were the largest recorded group in 1980 but Jehai were the largest in 1994.

How to label the northerners has been called a semantic problem. But it is much more than that. The northerners are often called "Negritos," a Spanish name for these non-Spaniards that means dark-short people. But "short" and "dark" are relative. First, on ample diets the northerners tend to grow taller. Second, this exonym "Negrito" mainly reflects the view of taller, twentieth-century Europeans. In the seventeenth century, European travelers regarded Southeast Asians generally as having medium stature, with some groups being quite tall; but at that time most Europeans were themselves thin and short by today's standards (Reid, 1987). Third,

Annandale and Robinson (1903) measured northern men versus Batang Padang Semai men and found the two samples to be the same in stature (note 10). Polunin (1953) found Lanoh to be the same average height as lowland Semai: 56.7 in. versus 56.8 in. for women and 60.8 in. versus 60.5 in. for men.

Lastly, northerners are not uniform in skin color or curliness of hair. They range from dark to lighter skin shades, from "chocolate" to "olive"; and their hair varies from wavy to wooly (Annandale and Robinson, 1903) (note 11).

The northerners and other Orang Asli groups have erroneously been considered to be "relatively untouched by civilization" by some observers (Ong, 1973) (note 12). This is emphatically not the case for certain economic indicators and for some kinds of lethal epidemics. Influenza, for one, was no respecter of forest dwellers.

HEALTH AND HEALTH SURVEYS

Most early writers on the northerners skimped on biomedical reportage. Annandale and Robinson (1903) noted that some groups of northerners had skin diseases while at least one, the "Po-Klo," did not. In the 1920's Schebesta (1973) briefly reported fevers and scabies, two cases of yaws (*puruh*), and one case of elephantiasis in Jehai.

In the 1930's, yaws and skin diseases were common in northern Orang Asli groups, according to Noone (1936). Also from that era and area, Evans (1937, p. 12) impressionisticly concluded that malaria "certainly takes a heavy toll." He went on, "Smallpox is said to have wiped out the Menik Gul...of Perak, while the influenza epidemic of 1918 ravaged the Kintak Bong..." Later he mentioned (pp. 18-19):

> "Skin complaints are common, especially forms of Tinea, which sometimes cover the whole body with intricate patterns...In Negritos at Ijok..., I noted some individuals were suffering from a form of indurated ulcer...yaws. About the existence of syphilis...I am not sure, but I have seen a [suspected] case...Leprosy appears to exist. I saw one man in what appeared to be an advanced stage...It was said, too, that Dahabok, a Leggong Negrito,...contracted leprosy and died of it...I have come across one case of elephantiasis."

A potpourri of studies on northerners has been published in the last half century. The authors of these studies report finding a wide

variety of intestinal parasites, little schistosomiasis, variable levels of filariasis and malaria, no mental illness, and a high frequency of goiter.

Polunin (1952a) found *Tinea imbricata* skin problems in 18% of Lanoh in Upper Perak, ranging from toddlers to elders. This flakey fungal condition often itches, leading to scratching and then to scaring and depigmentation.

Dunn (1968b) reported that 22 species of intestinal parasites were discovered in a sample of northerners, more diversity than in any other Orang Asli group. Yet despite the fact that 96% of the northerners had one or more kinds of intestinal worms, they did not have the highest worm burden (worm numbers per individual); Orang Asli groups further south had higher burdens (Dunn, 1972).

Greer and Anuar (1984) demonstrated, by immunological tests, a low level of prior exposure to schistosomiasis (7%) in Batek near Kuala Tahan, Pahang. This finding fits well with the observation that the snails carrying the disease favor ditches and ponds, rather than the streamflows typical of the rainforest (Cohen, 1989).

Polunin (1951b) found a filariasis focus at the Lanoh village of Ulu Kendrong, Perak, where 66% of the 32 people sampled had microfilariae in their blood. But Mak (1978) reported an average microfilaremia prevalence rate (the proportion infected on a particular day) for the northerners of only 11% (3 infected out of 27 studied). Yet Hakim and coworkers (1995) found a pre-medication prevalence rate of 25% for Jehai in Banun, Perak, and of 23% for a mixed Lanoh-Temiar settlement in Dala, Perak (sample sizes were not stated).

Mak (1978) also reported a 4% malarial parasite prevalence for northerners. He later reported a 2.1% malarial parasite prevalence rate in Jehai at Pergau, Jeli, Kelantan, for 1986 and an 0.3% rate in Batek at Sungai Lebir, Kuala Krai, Kelantan, for 1987 (Mak, 1992) – perhaps in the face of anti-malarial programs, but this was not mentioned in the report. Earlier, however, Polunin (1953) and Kinzie and coworkers (1966) had found much higher malarial rates in Lanoh and Jehai: 25% and 37%, respectively.

Tan and Armstrong (1976) found no northerners were admitted to Gombak Hospital with a diagnosis of mental illness over a 5-year period. However, hospital records often give inaccurate data on medical problems, unless everyone who is ill is admitted to hospital. More recently, Osman and coworkers (1995a) reported that 30% of the

northerners at Lubuk Legong, Kedah (Kensiu and perhaps others), had goiters.

These bits of information, welcome though they are, only provide a shadowy outline of the northerners' biomedical situation.

Significantly, only twice during the 20th century has a health survey been published on any northern group. The first was on 164 Lanoh in Upper Perak, living in a traditional setting as hunter-gatherers, collectors of forest products for sale, and plant cultivators. Polunin (1953) estimated that childhood mortality was extremely high in this sample, since among 43 living mothers 48% of their ever-born children had already died. He found a "spleen rate" of 50% for all ages combined, suggesting a high and chronic level of malaria, while malarial parasitemia prevalence was found to be 25%. No evidence was found of vitamin deficiency, neoplasms, diabetes, or rheumatic fever. One man had epilepsy. But 23% had enlarged thyroids (goiters), 12% had hepatomegaly, 34% had microfilariae, and 2% had elephantiasis. Among the 13 Lanoh so tested, all had hookworm but few other intestinal worms. Polunin observed that loss of weight was uncommon (9%) but difficult to evaluate in terms of health status. While 37% of the Lanoh showed signs of yaws, 54% had some skin disease – mainly tinea infections such as ringworm. The average hemoglobin level for 71 Lanoh was 9.5 g/dl (range 4 to 15 g), indicating anemia was prevalent. Although dental caries were common, severe dental problems were quite rare. Just 2% of the Lanoh had middle ear infections (otitis media) but 33% had lung abnormalities, mainly coughs; 32% had positive tuberculin tests, indicating past experience with tuberculosis.

The only other report on tuberculosis in the northerners estimated the disease incidence for the years 1951-1971 to be 2.2%, about average for all Orang Asli groups studied (Bolton and Snelling, 1975).

Although all human groups have been subject to epidemic diseases, few historical data on epidemics are available on northern Orang Asli. Outside of Evans' remarks in 1937 on the 1918 flu epidemic, Polunin (1953) mentioned a high death rate for sedentized Jehai in the 1950's due to a dysentery epidemic. Undoubtedly smallpox, cholera, and other epidemic infections also were cyclic killers during recent centuries. Indeed, during a cholera outbreak in 1993 among Jehai at Jeli, Kelantan, the death rate was 17% (3 of the 18 cases detected) (Roslan Ismail, 1997).

The second northerner health survey was on a Jehai group displaced to Fort Banding on the Upper Perak River. Such displacements, or relocations, or resettlements, or dispossessions, still occur today. In their report, Kinzie and coworkers (1966) observed that game and fish were scarce around the "fort" and speculated that the diet of the Jehai there was deficient in protein, iron, and B vitamins. They estimated the pre-adult mortality rate to be 54%, an estimate based on faulty methodology. If the authors had believed their own estimate, one might surmise they would have reported on the health of Jehai children and adults separately. Instead, they combined the two age groups. They found 64% of Jehai (all ages) to have hookworm, 14% to have Trichuris (whipworm), and 5% to have Giardia. They also found 64% to have dental caries, 3% to have malaria parasites (although the population was receiving anti-malarial drugs), 17% to have microfilaremia (with 10% having elephantiasis), 7% to be goiterous, and 22% to have hypertension. They discovered little anemia or threadworm infection and no diabetes, Ascaris (roundworm), or heart murmurs. Yaws was not mentioned. Of special interest is the fact that they had found 37% of these Jehai to have malaria parasites in 1963, before anti-malarial drug administration commenced.

To sum up the northerners' health status on the basis of these reports is problematical. For example, even on the arbitrary assumption that all northern ethnic subgroups are basically equivalent, it is difficult to assess the meaning of the findings that 34% of one group had microfilariae in the 1950's, 17% of another did so in the 1960's, but only 11% of a mixed sample did so in the 1970's. Although such a temporal decline in filariasis may be the norm for many inland Orang Asli groups (Table 2 in note 13), in 1992 one Jehai area (Banun) had 25% microfilaremia and a Temiar-Lanoh area (Dala) had 23% microfilaremia, versus an overall rate for West Malaysia of 0.35% (Hakim et al., 1995).

On another topic, scattered published data only tell us that malaria parasitemia was 25% in Lanoh in 1953, 37% in Jehai in 1963, 2.1% in Jehai in 1986, and 0.3% in Batek in 1987, with all studies presumably (but not certainly) being on groups not receiving anti-malarial medical help. These sparse data are not particularly informative.

A small northern group, the Mendrik, has not been studied biomedically in any serious way over the last half century. Why this

should be the case is unknown to me. The entire set of published data I have seen consists of 5 Mendrik surveyed by Mak (1978); all were negative for filariasis but one had malaria. All the same, it is well known that small isolates such as the Mendrik often have particular biomedical and genetic characteristics worthy of attention. Given that the 1918 flu pandemic, various smallpox epidemics, and the post-1945 roundup of Orang Asli by the (then) British administrtion into congested locales produced appalling death rates (Polunin, 1953), some small groups probably went through severe "bottlenecks" in which they lost genetic diversity. This hypothesis has not been tested in the Orang Asli.

Many northern groups were not sedentary until recently. Cohen (1989) reviewed the history of human infectious diseases, albeit he gave only passing attention to the Asian rainforest environment. He did present a provocative conjecture on the eco-medical differences between undisturbed small-band foragers and village-settled swiddeners. The former, while not nomadic in Malaysia, traditionally moved around within a territory and thus did not accumulate large refuse heaps or suffer from fecal-contaminated village sites. Some band-size groups had relatively little contact with outsiders (thus affecting the frequency and severity of smallpox and such-like viral epidemics). Few had permanent houses that could attract vermin (insects and rats). These characteristics of foragers, Cohen argued, would tend to insulate them from many disease threats, such as intestinal worms and plague experienced by settled agriculturalists.

With respect to Cohen's hypothesis, I should mention that Polunin (1953) was unable to study some fairly rare diseases that are transmitted directly from vertebrates to humans or are transmitted indirectly, by an invertebrate vector such as a mosquito. For any Orang Asli people who practice hunting routinely, the handling of wild animals or their remains probably has produced a variety of infections. The directly-transmitted illnesses are generally rare but include rabies and hemorraghic fevers (viral infections); leptospirosis, brucellosis, anthrax, and salmonellosis (bacterial infections); toxoplasmosis (a protozoan infection); and even more obscure conditions (Cohen, 1989). However, sedentary groups that raise, slaughter, or eat livestock also incur particular health risks (such as tetanus, anthrax, and tapeworms). On the basis of Cohen's hypothesis, then, we might ask if northern Orang Asli traditionally were more, or less, healthy than

Orang Asli swidden farmers such as the Semai or Temuan (groups discussed in later chapters). While this question has yet to be answered, in line with Cohen's hypothesis Dunn (1968b) found the northerners to have lower intestinal worm burdens than southern Orang Asli (generally more sedentary). Perhaps surprisingly then, all 86 northerners studied turned out to have hookworm, Ascaris, and Trichuris, with hookworm showing the same burden of infestation as in other groups. However, the northerners' burden for Ascaris and Trichuris was relatively low (Dunn, 1972). Also, Polunin (1953) found no Ascaris in 13 Lanoh stool samples, but all 13 had hookworm ova and 2 of the 13 had Trichuris ova. And Kinzie and coworkers (1966) found no Ascaris in the not-long-settled Jehai.

These findings tend to show that the northerners do have a somewhat different disease profile than more southerly (and sedentary) Orang Asli, thus offering some support for Cohen's hypothesis. In the case of Ascaris worms, for example, transmission is fecal-oral, through contaminated food, water, or hands. As mobile northerners rotated through their territory, soil-deposited Ascaris eggs would come to a "dead end." In theory, the same should be true for the bacteria that cause diarrhea, for hookworm (because its eggs must develop in the soil), for schistosomiasis (because the fluke must develop inside a snail that favors stagnant water), and for malaria (because infected mosquitos do not fly as far as foragers trek). Still, as Dunn's report showed, hookworm, at least, was as much a problem in northerners as in other Orang Asli groups (note 14).

In addition, the few studies available on malaria in northerners point to them having had (before any antimalarial campaign started) a parasite prevalence rate similar to those found for other Orang Asli groups that live inland (see Chapter 7).

DEMOGRAPHY

Despite the many – largely unrecorded – medical stresses experienced by the northern Orang Asli historically, their populations have recently been expanding. Gomes (1982) studied Jehai living at Sungai Rual Post, Kelantan, who were displaced there in 1972. He found that their mean age was 19.5 years in 1978, with 51% of the population of 193 people being below 15 years of age. Only 13% were over 39.

All this suggests the community was then either reproducing rapidly or was dying off rapidly in adulthood, or both. Completed fertility

for living women over 40 averaged 4.5 live births in 1978, and the mean number of children born per woman increased from 2.1 to 3.1 between 1976 and 1979 – all indications of a population increase. On the negative side, reproductive-age women (15 to 49 years of age) had a high mortality rate, with the sex ratio for adults 25 to 49 years being 27 males to 17 females. Gomes theorized that this high mortality rate was due to maternal deaths during childbirth or to related problems.

Ancillary data led Gomes to estimate that 20.5% of Jehai deaths occurred among reproductive-age women, given a crude death rate (CDR) for all ages of 3.3% per year for the years 1956-1979. To put this in proper perspective, this 3.3% rate is much higher than the 1% CDR for all Orang Asli combined during the years 1984-1987, or the 0.5% CDR for all Malaysians for this period, given by Ng and coworkers (1987). While age-specific mortality was not determinable for the Jehai, only 67% of liveborn offspring (all ages) had survived at the time of this 1977 study. Overall, the Jehai's high birth rate in the 1970's seems to have more than compensated for their disastrously high death rate, netting out as a population increase (note 15).

By 1988, ten years after the 1978 census, the mean age of the Rual Jehai had increased to 21.1 years (from 19.5). The population was still growing rapidly, at 3% per year (Gomes, 1990). Over this decade the number of children born to living, ever-married women increased by about 14%. At the same time, the number of *surviving* children of women over 40 years of age decreased from 3.83 in 1978 to 3.54 in 1988 (note 16). And for all adult women, only 64% of their liveborn offspring were still alive in 1988 – not unlike the 67% survival found for 1978. Gomes had previously estimated the crude death rate (CDR) for 1956-1979 at 3.3%, but for the period 1978-1988 it rose to 4.5%, with 59% of recorded deaths being in the 0-4 year age group. In contrast, the Malaysian national CDR for 1970 was 0.7%. The Jehai's ravaging CDR of 4.5% is the highest recorded for any Orang Asli group.

Gomes (1990) recently hypothesized that high Jehai fertility in the 1980's was a response to their high mortality rate at that time, resulting from the poor living conditions in their crowded and resource-depleted governmental village – conditions predisposing to transmissible diseases. Presumably, no health survey was ever carried out on these Jehai before and again after their "settlement" – thereby

missing an opportunity to determine the health effects of such executive action.

To sum up the studies of Gomes and others, Rual Jehai can no longer "move on," leaving their refuse and infectious agents behind. In some way, this would seem to be related to their high CDR, especially their high infant-plus-toddler mortality rate. Nor do these Jehai any longer eat a high-protein, high-fiber diet (see note 45 on the variety and amount of Batek food), since hunting, fishing, and gathering are no longer productive in the surrounding, outsider-logged forest, many hectares of which are now cleared by non-Orang Asli farmers.

GENETICS AND HISTORY

Polunin and Sneath (1953) were the first, and the most industrious, researchers on genetic traits in the northerners. They studied an impressive number of genes, sometimes in samples of respectable sizes. But because they found much variation among villages or areas within ethnic groups, they were reluctant to say that their results identified the "true" genetic makeup of these groups. And rightly so.

Polunin and Sneath also studied other Orang Asli: Temiar, Semai, Temuan (called Orang Dalam and Orang Darat), Jakun (called Orang Ulu), Besisi (officially called Mah Meri), and Orang Laut/Seletar. Significantly, the vast majority of these genetic traits have not been studied in any Orang Asli group since Polunin and Sneath did their work in the 1950's. For the record, the traits studied by Polunin and Sneath are listed below. Most are blood groups.

Northern Groups

Lanoh: ABO, Hb S, MNS, Rh, P, Lu, K, Se, Le
Jehai: ABO, Hb S, MNS, He, Rh, P, Lu, K, Fy, Se, Le
Kensiu/Kintak: ABO, Hb S, He, Rh, P, Lu, K, Fy, Se, Le

Central Groups

Temiar: ABO only
Pahang Semai: ABO only
Perak Semai: ABO, MNS, Rh, P, Lu, K, Se, Le

Southern Groups

Temuan: ABO, Hb S, MNS, He, Rh, P, Lu, K, Fy, Se
Jakun: ABO, Se
Besisi: ABO only
Orang Laut/Seletar: ABO, MNS, He, Rh, Se

As is usual with adjacent groups, all those listed above had the same array of gene alternatives (alleles), with one exception. The Seletar, a small breeding isolate, were homozygous, or uniform, in their Rh blood type: all 30 people tested were CDe/CDe. Otherwise, these groups differed only in the frequencies of the alleles they shared in common. This is the general situation for so-called classical alleles among adjacent groups: differences in frequencies but not in kind.

A perennial genetic question about the northern Orang Asli concerns their source population. While little genetic information is available on them, Lugg (1957) found that the frequency of their PTC tasting ability (a test of bitterness) differed from the frequency in the central-peninsula Orang Asli. Gamma globulin was studied by Steinberg and Lie-Injo (1972), who found the northerners had frequencies of "Gm" variants that differed markedly from those of other Orang Asli groups. Livingstone (1985) tabulated malario-genetic data on the northerners from scattered studies that suggested they are rather different from more southerly Orang Asli (see Chapter 7 on malaria). Roychoudhury and Nei (1988) surveyed various "classical" genetic traits researched on the northerners, such as blood groups and enzyme data. Lie-Injo (1976) quantified some of these classical data in a way that placed the northerners closer genetically to the Malay and Thai ethnic groups than to other Orang Asli, but this is an implausible finding.

Using more reliable mathematical techniques, and larger arrays of alleles, Nei (1982) was able to show that the northerners were closer genetically to more southerly Orang Asli than to Malays. Ballinger and coworkers (1992), although misnaming a northern group (the Jehai), obtained molecular data (on a sample size of two) that seemed to distance the Jehai genetically both from the more southerly Orang Asli and from Malays. Finally, Chen and coworkers (1995) studied "Kensiu," probably a mixture of older genetic data on northerners, but did not study other Orang Asli groups. Their findings suggest northerners are as related to Malays as, for instance, Turks are related to Koreans.

While inconclusive, these various studies point to some – but not much – genetic distinctiveness of the northerners from other Orang Asli groups and from nearby non-Orang Asli groups. This is not a remarkable finding to geneticists. Semai are also somewhat distinct from Temuan, and both are distinct from Malays.

SUMMARY AND CONCLUSIONS

To reprise this review of northern Orang Asli, it is doubtful that enough has been reported about their biology and their health to come to any sweeping conclusions. That is, what is known is far less than what is unknown, or what could be known.

Studies to date have not been interdisciplinary enough, have had little time depth (except for Gomes' meticulous work), and have not been carried out on a prioritized basis – in order to deal with the most serious health problems first. This latter point depends on *knowing* which health problems are serious for all sectors of a community, especially including mothers and young children. What, for instance, is the cause of the high maternal mortality pinpointed by Gomes in his work? Such information appears to be unavailable.

Setting aside unknown health problems, the available data indicate that the health of the northerners today is at least compromised by a high frequency of goiter, some intestinal parasitism, and some malaria. In order to improve matters for the future, I would suggest that comprehensive health surveys be carried out as a first step. Such in-village surveys, if combined with solicitation of village-wide suggestions for health improvement, would set the stage for major health gains.

Timeline 1

NORTHERN ORANG ASLI HEALTH RESEARCH

(excludes review articles; numbered entries in the bibliography are listed in parentheses).

1928 miscellaneous (258)

▼

1930

▼

▼

▼

▼

▼

▼

1937 miscellaneous (195)

▼

▼

1940

▼▼

1950

1951 goiter (226); filariasis (227)

1952 tinea (228)

1953 health survey (231); genetics (239)

▼

▼

▼

1957 genetics (170)

▼

▼

1960

▼

▼

▼

1964 dermatoglyphics (126); genetics (157)

▼

1966 health survey (142)

1967 intestinal worms (206)

Colin Nicholas

Elderly Kensiu couple in traditional dwelling, Baling, Northern Perak.
Many of the Northern Orang Asli groups have now been administratively moved to settlements. Prior to this, they were accustomed to foraging in small bands and had less contact with outsiders. This helped to insulate them from many disease threats.

1968 intestinal worms (63)
▼
1970
1971 nutrition (25)
1972 malaria (24); nutrition (25); intestinal worms (65); genetics (266)
1973 demography (275)
▼
▼
1976 mental health (277)
1977 demography (97); miscellaneous (238)
1978 filariasis (172)
▼
1980 nutrition (70)
1981 malaria (288a)
1982 demography (100); genetics (190)
1983 demography (101)
1984 schistosomiasis (106)
▼
▼
▼
▼
▼
1990 demography (102)
▼
1992 mitochondrial DNA (20); several diseases (176)
▼
▼
1995 genetics (42); filariasis (110); goiter (220)
1996 filariasis (290)

Chapter 3

TEMIAR AND SEMAI

As the accompanying timelines on Temiar and Semai show (Timelines Nos. 2 and 3), Temiar have been relatively neglected in terms of health studies. Outside of malaria studies, most of the important work on Temiar was done in the 1970's. A key problem is that no general health survey has ever been published on them. For Semai, in contrast, information is sufficient to assemble a reasonable outline of their health history and health status today. A key problem is that many Semai health hazards recorded a generation ago seem to persist today.

The Senoi subgroup of Orang Asli contains Temiar and Semai, as well as four smaller groups. Temiar and Semai have closely related languages and adjacent territories, with Temiar occupying the more northerly part of the interior highlands (in upper Perak and Kelantan) and Semai the central part (in lower Perak and Pahang). Semai are more numerous, with a 1994 population of about 26,000. There are about 15,000 Temiar.

Temiar and Semai by tradition are swidden cultivators and collectors of forest products mixed in with hunting, foraging, and fishing. As to their appearance, it is noticeably diverse. Williams-Hunt wrote (1952, p. 17):

"Now there are so many types of Senoi that it is difficult to describe any particular type as being predominant...There are Senoi who look like Malays. There are Senoi who look like Chinese. There are Senoi with long heads and...Senoi with round heads."

Indeed, the Orang Asli of the middle-to-southern part of the Malay Peninsula all show such diversity. Already in 1903, Annandale and Robinson (pt. 1, p. 30) had observed that many Semai of southern

Perak "have yellower skins than the Malays of their district, while some approach ... a Chinaman in complexion."

Temiar and Semai are closely related in their genetic makeup, as shown, for example, in their profile of innate resistance to malaria (Chapter 7). While some of this relatedness is undoubtedly ancient, some is recent. Fix (1977) retold the oral history about one Temiar man, Tok Renuuit, who "became" a Semai. Tok Renuuit, born about 1810, migrated to the Satak area of Pahang, married, and had children. More than 30% of Satak area Semai in 1969 counted Tok Renuuit as one of their ancestors. Some of these descendents were already great-great-great-great-great grandchildren of Tok Renuuit.

Because of the closeness between Temiar and Semai in geography and language, I review the health status of both groups in this chapter.

It is known that Temiar and Semai underwent great stress after 1945 when the colonial government rounded up many of them and put them in unhealthy "camps." This tactic got them out of the way in the British fight against anti-colonial, communist insurgents during the so-called Emergency era. More has been written about the fate of Semai than of Temiar in these camps. Gomes (1983), for instance, has described the camps in the Batang Padang district of Perak. Disease was common and many people died.

Most biomedical reports treat the Temiar and Semai separately (and I will review them separately later in this chapter), but a few reports identify the subjects of study only as Senoi. A good guess is that Semai are well represented in such studies, because of their numerical advantage. For instance, Sumithran and Prathap (1976, 1977) were one of the few research teams to study cancer in Orang Asli. In one analysis they found that members of the Senoi subgroup showed particular evidence of non-alcoholic cirrhosis, with 45% of these 22 cirrhotics also having liver cancer. Chronic hepatitis B virus infection was implicated as a likely precursor in all 22 cases. In a second analysis they found that liver cancer was the leading type of cancer in Senoi and that it was more common in them than in other Orang Asli, according to Gombak Hospital records. Lung cancer, the next most common type in Senoi, occurred at the same rate as in "Aboriginal Malays." Cancer diagnoses on northern Orang Asli were too rare to permit reliable comparisons with them.

This sparse information can be supplemented with the only other published data on cancer in Orang Asli. Prathap and Montgomery (1974) reported that 9% of in-hospital Orang Asli deaths were due to cancer. But the vast majority of deaths were due to some kind of infection.

Gilman and coworkers (1976) compared Temiar and Semai as to their level of amebic dysentery. More to the point, this study compared an upstream, fairly isolated community (Temiar at Belatim, Kelantan) with a Semai community (Satak, Pahang) in a downstream area now neighbored by non-Semai. The team studied *Entameba histolytica* infection, which can cause dysentery, especially in children. The amebic cysts are disseminated by the fecal-oral route. By immunological tests Temiar showed far less exposure to this organism (4%) than did Semai (32%), who live in an ameba-endemic area. The authors conjectured that the high rate in Satak Semai was due to store-bought vegetables, perhaps grown with human manure as fertilizer, as well as to fecal pollution of the nearby river (where bathing, children's play, and clothes washing takes place). While the authors thought it ironic that "civilization" could both spread and control enteric disease, they did not mention providing any therapeutic measures in their report.

Most Orang Asli in Perak state are Temiar or Semai. For Perak Orang Asli as a whole, the official tuberculosis rate for ages 1 to 14 years was 3 times that of other groups in 1995 (New Straits Times, 12 May, 1997). For Perak Orang Asli of all ages, the rate has been unofficially estimated to be more than 11 times that of other groups.

Finally, I might mention that Hirayama and coworkers (1996) studied some genetical aspects of human leucocyte antigens (HLA) in a mixed sample of Temiar and Semai.

TEMIAR HEALTH AND HEALTH SURVEYS

The reports quoted in Chapter 1 show that Temiar had an impressive array of health problems in the 1930's, even upon superficial examination. Among the more serious were yaws, malaria, scabies, bronchitis, ascariasis, and childhood fevers. Later, issues in internal medicine came more to the fore. But no general survey of Temiar health has ever been published.

As previously shown in Table 2 (in note 13), Temiar were surveyed in the 1950's through the 1970's for filariasis. During this period their prevalence rate declined from 21% to 3%. Earlier, Polunin (1951) had seen one case of elephantiasis among 20 Temiar at Lawin, Upper

Perak, and 2 cases among 30 Temiar at Kuala Chenka in Ulu Plus, but no cases among 100 other Temiar in the Ulu Plus area. In 1961 Wharton and coworkers (1963) found 1 case of elephantiasis among 239 Temiar in Ulu Kelantan. In the 1970's the Temiar had the highest filarial worm burden, or density, among the Orang Asli groups so studied (Mak, 1978), largely due to the very high densities found in Temiar females. And for a Temiar/Lanoh village at Dala, Upper Perak, Hakim and coworkers (1995) reported a 23% microfilarial rate.

Polunin (1953) found 20% of 86 Ulu Plus Temiar to have malarial parasitemia. In 1970, despite monthly prophylaxis that started in 1964, up to 10% of Temiar in Kemar, Perak, had malarial parasitemia. And Legap Temiar had parasitemia rates of 37% to 49% in the 1986-1991 period (see Table 10 in Chapter 7).

In 1970 Burns-Cox and Maclean found 38% of 85 Kelantan Temiar over the age of 14 years to have palpable spleens, which the authors attributed to chronic malaria. Incidental cases of chronic tophaceous (joint-lump) gout, filariasis, and chronic bronchitis were also observed. With respect to gout, Dugdale and coauthors (1971) mentioned that it was common among Orang Asli, without providing any details. Wharton and colleagues (1963) earlier had found a malaria parasitemia rate of 15% overall for Temiar in Kelantan. In addition, Mak (1992) briefly reported a malarial rate of 22% for Nenggiri, Kelantan, Temiar for 1985, although he did not report sample size, ages surveyed, or other details for that datum.

In 1972, Dunn reported that 78% of the Temiar he surveyed had hookworm, 23% had Trichuris, 2% had Ascaris, and 3% had Giardia parasites. About 16% were free of any such infestation. Yet Dunn rated the "sanitary status" of the Temiar as fairly good, compared to some other Orang Asli communities. At the same time, Dunn (1972) found that only 0.8% of Temiar had amebiasis, but Gilman and coworkers (1976) reported that 4% of Temiar at Belatim, Kelantan, showed prior exposure serologically. This rate, however, was much lower than found for Semai.

In 1975, Bolton and Snelling reported that Temiar had the highest incidence of tuberculosis among Orang Asli, although Semai were a close second. Southern Asli groups were less affected. In 1996, Lokman and Baharuddin noted that Legap Temiar in Perak had a relatively high prevalence of TB.

According to Mak (1992), Nenggiri, Kelantan Temiar showed the

greatest prior exposure to schistosomes of any Orang Asli group studied immunologically. In a sample of 327 Temiar, 38% were positive in 1986. However, chronic schistosomiasis in the Orang Asli appears to be mainly asymptomatic – or simply indistinguishable from the usual background of ill health.

Chan and coworkers (1974) studied schoolboys and men at Gemalah in Kelantan as to their cardiovascular fitness. These Temiar were considered to be well nourished and healthy. That is, they did not have malaria, elephantiasis, or yaws. However, 60% had skin problems, more than 30% had decayed teeth, and 80% had upper respiratory infections. In general, their cardiovascular fitness was found to be the same as urban males.

Finally, according to hospital records for 1969-1974, psychiatric illness was relatively uncommon among Temiar (Tan and Armstrong, 1976).

TEMIAR DEMOGRAPHY AND GENETICS

Apparently no demographic study has been carried out on Temiar. Noone (1936) provided some data for the Ulu Plus area for 1931. For a population of 2039 covering 30 locales, his data gave a sex ratio of 1.10 males to 1 female. Whether all 2039 persons were Temiar was not stated. Noone further reported that for 100 Temiar marriages, 294 children were born; 36% of these children predeceased their mothers.

For Temiar resettled in the Plus river valley of Perak in 1950, 64 deaths were reported over a four-month period versus 8 births (Williams-Hunt, 1952), a ratio of 8 to 1, higher than in other Orang Asli groups.

More recently, some data on a Temiar-northerner combination village were published by Tan (1973). He found a sex ratio of 1.24 males to 1 female in a population of 83 at Ulu Grik. Out of the 83, 56 were Temiar, with most of the others being Lanoh. In this government-established village, many young adults were not in residence (and not censused by Tan), being away at jobs or on outlying farms (swiddens). Given this caveat, 30% of the census group was less than 16 years of age and 28% was over 39. However, only 6% was in the age range of 0 to 2 years, suggesting to Tan either high infant mortality or low fertility (or both). Looking at then-current marriages, Tan found 18 women had had 47 children (averaging 2.6 children per woman), but

only 28 offspring (60%) were alive during his fieldwork period. The inferred 40% mortality rate is, in two words, disturbingly high. Moreover, Noone (1936) had earlier reported similar mortality rates for 294 Temiar offspring: 45% at down-river villages and 29% at up-river villages, for an average of 36%.

The village studied by Tan provides one example of how Orang Asli gene pools may be changing today, since that village brings together ethnic groups with different mating patterns. One might hypothesize that ethnic endogamy, or inbreeding, is becoming less common.

Relatively few genetic studies have been carried out on Temiar. Polunin and Sneath (1953) studied only their ABO blood group traits. Based on later work (Lie-Injo, 1976), Temiar appear to have the same array of genetic variants as Semai, who have been more thoroughly studied. They are also similar to Semai in terms of malaria-related and other allele frequencies. In contrast, of the 7 Temiar tested by Ballinger and coworkers (1992), none had the genetically well-known 9 base-pair deletion in their mitochondrial DNA, although this trait occurs in Semai.

For the Duffy blood group, Temiar have an "Fy-b" allele frequency of 0.03 – much like Semai (0.04), Temuan (0.07), and Jakun (0.05), but less like Semelai (0.32) (Lewis et al., 1988). Temiar are like Semai in "Gm" frequencies and unlike Jakun in "Inv (1)" frequencies (Steinberg and Lie-Injo, 1972).

SEMAI HEALTH AND HEALTH SURVEYS

Many aspects of Semai health have been studied, starting in the colonial era. In 1903 the majority of Semai men seen by Annandale and Robinson had ringworm. Nevin (1937) studied over 100 Semai at Kuala Denek, Perak. He found that 10% had filariasis, 26% had hookworm, 70% had Ascaris, and 14% had Trichuris. In 1935, the prevalence rate for malaria was 19%, with a rate of 6% in adults and 85% in those less than 10 years of age. After treating the population with plasmoquine for 2 years, the parasite rate fell from 19% overall to 5%, the spleen rate fell from 18% to 4%, and the average "hemoglobin percent" in the blood rose from 64 to 72 – a good sign.

Polunin (1953) reported that in 1950 approximately 2200 Ulu Jelai Semai were coercively crowded into an area about 1 mi. by 2 mi. at Bukit Betong, Pahang, giving a density of over 1000 people per sq. mi. Traditionally, Semai population density would have been below 10 or so per sq. mi. Fix (1974, 1975), for example, gave a density of 1 to 7 per sq. mi. for Semai in Satak, Pahang. Polunin blamed the crowding at Bukit Betong for the high level of dysentery and general ill health he found while conducting a medical survey there.

The Pahang Semai had been "resettled" for 3 to 6 months when Polunin arrived in Bukit Betong. They were receiving a handout of 1300 kcal of food per person per day. These energy-deficient rations were also deficient in fats, calcium, and iron. Administrators unrealistically assumed that foraging or cash purchases would supplement the food handout. Semai health thus declined, with the death rate being high and the birth rate low. Polunin found that the ratio of deaths to births at Bukit Betong was more than 5 to 1.

For the Bukit Betong Semai the sex ratio was 187 males to 162 females, or 1.15, suggesting poorer survival of females. Indeed, Polunin's graph of age versus sex for a combined sample of Semai and Lanoh (from another area) shows that those cohorts 40 years or older had a sex ratio of 85 to 48, or 1.77. Women were not surviving their child-bearing years well.

Semai mothers reported to Polunin that 48% of their ever-born offspring had predeceased them. Fortunately, sterility was not a large problem: among females over the age of 20 years, including unmarried ones, only 8% had had no children.

Among all Semai studied by Polunin, 49% had palpable spleens, 18% had enlarged livers, 18% had malarial parasites, 64% had filariasis (with one case of elephantiasis), 86% had hookworm, 7% had Ascaris, 2% had Trichuris, 18% showed signs of yaws, 52% had enlarged thyroids (goiter), 45% showed markedly low weight, 6% had ringworm, 6% had scabies, and 10% had ulcers or boils. While 34% had some sort of respiratory problem, usually a chronic cough, less than 1% had definite signs of pulmonary tuberculosis; but no tuberculin tests were performed. Blood pressure readings were generally low but anemia was common, especially in children and women. Cancer was not observed.

Semai deaths at Bukit Betong were reportedly caused by equal proportions of dysentery, respiratory diseases, fevers, and "other." Many deaths were of young children.

Although health was poor overall for Semai in Bukit Betong, when

Polunin analyzed the data by prior residence at low- versus high-elevation, he found that high-elevation Semai were healthier than those who had lived at lower levels. In categories such as childhood mortality, ringworm, scabies, palpable spleens or livers, ascariasis, coughs and rales, and yaws, the lowlanders were 8 to 45 percentage points ahead of the highlanders in their ailment frequencies. However, Polunin's altitudinal contrasts could also have been framed in terms of more versus less urban, more versus less formal education, or more versus less population density. Any of these differences may produce an effect on health, albeit in various ways – from differential access to medical care to the degree of pollution of drinking-water supplies.

Fix (1977), who studied Semai demography, reported on causes of death among Pahang Semai in a traditional area for the period 1950 to 1969. For infants (0 to 1 years), the leading cause of death was "unknown," followed by "sampuu" – involving anemia and wasting, and then respiratory problems. Infants accounted for 50.5% of all known deaths. The main cause of death for reproductive-age women was childbirth (45% of all their deaths). Respiratory problems, sampuu, and dysentery were the three leading causes of death for all ages combined (N=301), if one ignores unknown causes.

For 218 Semai, Dunn (1972) reported 66% had hookworm, 40% had Ascaris, and 51% had Trichuris, with 7% being free of intestinal parasites.

With regard to dysentery, Gilman and coworkers (1976) reported that 32% of Semai in Satak, Pahang, revealed prior experience with amebiasis immunologically, as I mentioned earlier. And Dunn (1972) found about 8% of the 218 Semai he surveyed harbored amebic parasites. He judged Semai living conditions to be variable, rating some fair and others poor.

Semai are the best studied Orang Asli group in the biomedical literature, having over 50 reports that contain information on them. Yet there has never been a comprehensive report on their health status since Polunin's 1953 publication. The reports by Khor come closest, although her main focus was on nutrition.

Khor (1985, 1988) surveyed Semai of Batang Padang district, Perak. She reported that over a third of the children had one or more dental caries. Among children 0 to 11 years of age, 36% of those living in up-river villages that practiced subsistence agriculture had intestinal worms, although the intensity of infestation was low. Among

same-age children at lower elevations, or those living in villages with market-oriented economies, 93% had such infestations, often with a heavy worm burden. Prevalence rates for malaria parasitemia, based on governmental sampling throughout the district, were 7% to 17% for the years 1980-1982, with 93% of the parasitemia occurring in the age range of 0 to 18 years (note 17). While these findings are highly suggestive of recent health problems in the Semai, the bulk of Khor's fieldwork was on malnutrition, a topic reviewed in Chapter 8. Here it is enough to mention that over 25% of Semai 13 to 18 years of age studied by Khor had iron-deficiency anemia; that 64% of pregnant women were anemic; that energy nutrition for her all-age sample averaged at least 50% below the recommended daily amount for Malaysians; and that 58% of those 0 to 18 years of age were underweight and 74% were stunted.

The findings of Khor were validated, in large part, by the more recent work of Osman and Zaleha at Lanai and Betau, Pahang (1995). For Semai 2 to 6 years of age, they found 5% had hookworm, 21% had Ascaris, and 24% had Trichuris. For comparison, the respective values from Khor's work were 14%, 57%, and 49% for children less than 12 years of age. Osman and Zaleha found 80% of those 2 to 12 years of age were malnourished versus 58% for Khor. In addition, Osman and Zaleha reported that 80% of the children they studied were goiterous. A year later Wan Nazimoon and coworkers reported that 79% of Sinderut Semai children 4 to 15 years of age were malnourished.

In temporal terms, the reports of Polunin, Khor, and Osman and Zaleha suggest that malaria, intestinal infestations, and malnutrition have persisted as health problems for Semai for over 40 years. Further support for this conclusion comes from the findings of other reports on Semai health.

As shown in the table on Semai health (Table 3 in note 18) and elsewhere, filariasis prevalence declined from 64% in the 1950's to 7% in the 1970's for the Semai (Table 2 in note 13). Malaria prevalence varied over the decades, being as high as 47% for one Semai group in the 1980's (Table 10 in Chapter 7). Hookworm infestations went from 86% in the 1950's to 5% in the 1990's. Ascariasis went from 70% in the 1930's to 21% in the 1990's. Trichuriasis increased from 14% in the 1930's to over 50% in the 1970's and 1980's but then decreased to 24% in the 1990's. Malnutrition (low weight, or other

measurements) increased from 58% in the 1980's to 80% in the 1990's. Goiter continues to be a noticeable and serious problem (Table 13 in Chapter 8), despite its widely known ease of prevention. Comparative temporal data are not available for many other health factors.

In sum, the Semai appear to have exchanged a lower level of filariasis and some intestinal worms for a higher level of malnutrition and the persistence of malaria and goiter. It is quite likely that other problems, including some poorly studied, continue as dangers to Semai health.

To take one example, the provision of medical services is far from effective. Bah Tony (1991) told of a doctor ordering the medical evacuation of an infant with severe diarrhea from Sinderut, a Semai village in Pahang, to Ipoh. Since Sinderut was "roadless," to get to Ipoh otherwise would take a two-day trek over mountainous terrain. When the helicopter arrived at Ipoh, no transport was waiting to take the child and his young mother to hospital. In half an hour he had died. He was buried later that day without any time-honored religious rituals. His mother was left stranded in Ipoh for an indefinite period, far from home and kindred.

SEMAI DEMOGRAPHY

During historical times, Semai population size has undoubtedly fluctuated, as a result of epidemics and punitive events. But warfare was not practiced by the Semai – or by any other Orang Asli group for that matter. Census counts on Semai taken before 1960, however, are virtually useless for historical analysis, as Fix (1977) discussed.

According to oral history, slave raiding by Malays over two centuries or longer decimated the Semai population (Endicott, 1983; Dentan, 1988). And one can only imagine the havoc, deaths, and ill health that resulted from the great Malayan flood of 1926, when some rivers rose 60 to 80 feet above normal. Later, in the 1950's, Semai populations were under some stress, at the least, given a 5 to 1 ratio of deaths to births in one crowded Pahang enclave (Polunin, 1953).

Some improvement occurred thereafter. This was shown by Fix's demographic analysis of Satak Semai in Pahang, covering the 1960's through the 1980's, a period of social change. In brief, Fix (1982a) found that in 1969 the infant mortality rate was 20% and the childhood mortality rate (for 0 to 15 years) was about 40%, based on life tables. The life expectancy at birth then averaged about 30 years, less than

Table 4
Estimates of life expectancy at birth (in years)

	Life expectancy			
	Date	Males	Females	Reference
Satak Semai	1969	31	28	Fix, 1982a
Satak Semai	1987	35	35	Fix, 1989a and b
Northerners	1990's?	48	46	Razha Rashid, 1995a
All Orang Asli	1985	54	52	Ng et al., 1987
West Malaysia	1985	72	68	Ng et al., 1987

half of the West Malaysian average at that time (66 years; Tan, 1982). But the population growth rate increased from 0.7% per year in the 1960's to an average of 2.05% for the period 1969-1987 (Fix, 1989a and b). Over the same period, life expectancy at birth increased from about 31 to 35 years for males, but even more substantially for females – from 28 to 35 years (Table 4). Alongside these increases, the total fertility rate for Semai women who survived through their reproductive years increased from 5.7 to 7.4 children. And in 1969 the crude birth rate (CBR) was 4.2%, versus 5% in 1987. With reference to this increase, Fix reported that few women (and presumably no men) were limiting family size by using contraceptives during this period (note 19). By 1987, according to Fix, the vast majority of Satak women were giving birth in hospital, a notable change from earlier decades.

While these data indicate an improvement in Semai population dynamics, Satak Semai mortality remained relatively high in 1987. Despite the fact that women were then living longer, they were still at a disadvantage relative to males: the sex ratio in 1987 for adults (>15 years of age) was 1.32 males to 1 female. That is, women were considerably less durable than men (note 20). Further, the nature of Fix's data did not permit him to estimate miscarriage or stillbirth rates. Such information would have been of interest, inasmuch as the maternal stresses of pregnancy and delivery (whether liveborn or not), let alone lactation and infant care, are known to be factors in the morbidity and mortality of women (note 21).

Some data also suggest that Pahang Semai women become sterile soon after age 35, but before menopause (Fix, 1971); here, malnutrition may be an underlying cause. However, Fix also estimated that absolute sterility for Semai women was around 5% in 1969, not a major deterrent to population increase.

Khor (1985) has added to the demographic picture for those Semai living in the state of Perak. She found that Semai of the Batang Padang district were less likely to survive to old age than the average Malaysian. For a spectrum of villages, from remote interior to peri-urban, only 2% of the Semai population (N=1807) was 60 years or older, while for Malaysia as a whole the figure was 6%. And among those Semai who were in this top age category, the sex ratio was 1.13 males to 1 female in the 1980's, as compared to 0.96 males to 1 female for the all-Malaysian average. Thus (again) older Semai women were relatively scarce; Semai women tended to die off at younger ages (Table 5). They also survived less well than the all-Malaysian average; this is particularly true for the most inland Semai sample.

Table 5
Semai sex ratio (males to females)
(After Khor, 1985)

	Age group (years)				
	0-14	15-29	30-44	45-59	60+
Semai: up-river, subsistence economy (N=409)	1.01	1.12	1.19	1.08	1.67
Semai: up- and down-river, various economies (N=1807)	1.05	1.04	1.06	1.04	1.13
Malaysia, 1980, Dept. of Statistics	1.04	0.96	1.03	0.98	0.96

SEMAI GENETICS

The first genetic report on Semai (Polunin and Sneath, 1953) surveyed 7 blood-group systems. Since then, more genetic surveys have been carried out on Semai than on any other Orang Asli group. Thereby, Semai have been found to harbor some rare genetic traits, but these may either illuminate or confuse questions of ethnic bio-relationships. In the Semai case, the allele AK-3 has been found in them, in Khmer Cambodians, and in the Stieng of Vietnam – all Mon-Khmer speaking groups. AK-3 has not been found in groups outside this language category, such as Thai, Cham, Temuan, Malay, Kadazan, or many Indonesian populations (note 22). This suggests AK-3 is an ancient genetic marker of Mon-Khmer speakers.

However, some relevant populations have not been tested for this AK marker, and some test samples may have been too small to plausibly reject the likelihood of finding AK-3 in other populations. If, say, a cluster of AK-3 alleles was found next year on the island of Okinawa, the Mon-Khmer origin hypothesis would fall down.

With these snares in mind, let us look at some other rare alleles found in Semai and often in other groups (Roychoudhury and Nei, 1988; Omoto et al., 1993; Saha et al., 1995; Baer, 1998):

- The allele PGD-F has been found only in Semai. It may be an allele exclusive to Semai, or to Senoi generally – a so-called private polymorphism.
- The allele PGM1-6 has been found in Semai, Thai, Nepalese, Bugi, Tagalog, Angeles Aeta, and Ifugao, but also in Hainan groups, in Europe, and in Africa.
- The allele PGM1-7 has been found in Semai, Temiar, Thai, Malay, Jakun, Javanese, Filipinos, Chinese, and some Pacific populations (see also Table 9).
- The allele TfD-Chi occurs in Semai, Thai, Khmer, northern Orang Asli, Malays, Borneans, some Indonesians, Tagalog, Japanese, Chinese, Koreans, Veddah, Oraon, Bhutanese, Arabians, and Finns.
- The allele PepB-2 has been found in Semai, on Flores, on Roti, on Timor, and in Irian Jaya, but also in Europe and Iran (see also Table 6 in Chapter 4).

- The blood group Lu-a has been found in Semai, Thai, Toba Batak, Land Dayak (Bidayuh), southern Filipinos, and the Miao on Hainan.

In terms of hypotheses about the ancient relationships of Semai to other populations, the known distributions of these rare alleles fit no popular model. Clearly, alleles PGM1-6, PGM1-7, TfD-Chi, and Lu-a are widespread in Southeast Asia, if not also elsewhere. It would be of some interest if the allele PepB-2 indeed has a disjunct distribution on the Malay Peninsula and in the far eastern region of Indonesia. Unfortunately, the PepB genetic system has been poorly studied in mainland Southeast Asia, on Borneo, or in the Philippines; so the distribution of the B-2 allele may turn out to be more continuous than is now evident.

A much-studied genetic trait is the so-called 9 base-pair deletion for mitochondrial DNA. It appears in various East Asian groups and also in indigenous Americans. The deletion is quite common in some groups, such as South Chinese (22%), Malays (26%), Ilocano (36%), south Kalimantan villagers (24%), the indigenous people of Taiwan (42%), and also the Semai (37%), according to Melton and coworkers (1995). Out of 30 unrelated Semai tested by this team, 11 had the deletion. Earlier, however, it was found in only one of 32 Orang Asli so tested, a Semai (Ballinger et al., 1992). This trait appears to be a widely-dispersed one of very ancient origin.

By studying the differences in allele frequencies among populations – rather than the presence or absence of rare alleles as already described, several reports have compared Semai with other ethnic groups. The earliest was that of Lie-Injo (1976) who found Senoi (mainly Semai) to be equally close genetically to "aboriginal Malays," Malays, Chinese, and Thai. Melton and coworkers (1995) compared Semai (a sample size of 30) with 10 non-Orang Asli but Asian populations, based on information about the 9 base-pair deletion in mitochondrial DNA. Their analysis located the Semai genetically closer to Filipinos (Ilocano and others) than to the other 9 groups tested. Saha and coworkers (1995) also compared Semai with non-Orang Asli, regional populations, 14 of them. These authors concluded that in terms of non-mitochondrial alleles Semai were much closer genetically to Khmers than to northeast Asians or to Malays, while Malays were closest to Chinese. Since Semai and Khmer both speak Mon-Khmer languages, their close genetic relationship is not surprising.

The genetical structure of Semai subpopulations has been well analyzed. Fix (1975, 1977, 1982a) showed that the composition of hamlets and villages traditionally were in flux, with migration hither and yon. Epidemics or quarrels could precipitate these moves. But migration between Semai settlements was not random.

Kin groups splintered off from one village and joined another, or started a new, offshoot village. This process came to be described as fission-fusion. Perhaps more accurately it could be called "fission followed either by fusion or offshooting." As a result of this process, villages were neither genetically static nor genetically isolated for long periods. And because kindreds share many genes, the number of people in any splinter group was larger than the number of the genomes that were migrating – which Fix called a kinship effect. If such a migrating kindred was large, it might "swamp" the gene pool of the village to which it fused, abruptly changing the gene pool of the recipient village in dramatic fashion. Primarily because of such migratory patterns, Semai villages were found to be quite dissimilar in their frequencies of genetic traits; that is, they were microdifferentiated.

These findings suggest that one would be hard put to identify any particular Semai village as genetically "typical." In addition, observing the genetic makeup of a Semai village today provides little insight into its genetic makeup 10 or 100 years ago (note 23).

In one historical reconstruction provided by Fix (1975), in the year 1935 or thereabouts village A was small. Soon thereafter, a kindred migrated into village A from village B, located 10 miles away. By 1975, 43% of the people in village A were descended from the "B" immigrants. In the meantime, another splinter group from village C fused with village A. This was followed by the complete fissioning of a large village nearby (village D), due to a virulent epidemic. It resulted in some kindreds moving into village A, while others from village D went off and established a new village, village E.

Related to all these micromovements, Fix (1975) uncovered a range of allele frequencies in 7 Semai villages by studying their genetic profiles. For the ABO gene, one village had a frequency of about 13% for allele B while another had 51%. For hemoglobin E, one village had about 17%, another 35%. For the OV allele of ovalocytosis, the range was 3% to 29%. These differences were certainly due, in

part, to chance events. Such events include genetic drift, inter-village migration decisions of kin groups, and, to a certain extent, where one happened to find a marriage partner (note 24). Other processes may also have been involved, such as selection favoring the hemoglobin E and OV alleles strongly in villages with relatively high levels of malaria. Fix (1982a) favored splinter-group migration as the main cause of this genetic micro-differentiation. Malarial selection was not, however, studied.

Hemoglobin E is a genetic variant that provides some resistance to malaria, as discussed in Chapter 7. It is found throughout mainland Southeast Asia and spills over to nearby parts of island Southeast Asia. It reaches its highest frequencies in the Semai and other Mon-Khmer speakers of the Austroasiatic language family. Various writers have theorized that this wide distribution reflects ancient migrations in the region, coupled with selection favoring the E allele in malarious environments. Fix (1981) pointed out that when kin groups having a high proportion of the E allele migrate together, they can spread its advantageous effects rapidly to new locales. He postulated that this kinship effect was important in establishing the E allele throughout its range.

Semai matings are not random, as geneticists use the term, but involve marriage choices based largely on ethnicity and proximity. Semai tend to marry other Semai, especially those living within a 12 mile radius (Fix, 1982a). But first-cousin marriages are rare (Fix, 1977), being less that 1% of all marriages. In general, Fix found that the level of inbreeding was low in Semai (F=0 to 0.004). As with other groups, the Semai ideal is to avoid inbreeding altogether.

Outbreeding of Semai to other ethnic groups, particularly marriage between Semai women and Chinese men, was rather common during the upheavels of war and its aftermath, in the period from 1942 to about the mid-1950's. Indeed, Bolton and Lie-Injo (1969) documented the introduction of the genetic trait called beta-thalassemia into a Semai enclave in Gopeng, Perak, via a Chinese grandfather. As this grandfather had 9 children, the trait became well established in the population.

Gajra and others (1994) studied inherited variants of apolipoprotein E in Semai, in relation to cholesterol levels in the blood. Among other findings, the Semai were shown definitely not to have "a cholesterol problem."

SUMMARY

It is difficult to give "chapter and verse" about Temiar biomedical issues because the patchwork of studies done so far does not form a coherent whole. On the basis of current information, there is some reason to think that the Temiar situation is generally similar to that of the Semai, even though the Temiar in the past were geographically more isolated than the "average" Semai. Unlike some other groups, however, Temiar may continue to experience high rates of filariasis.

In-village general health surveys as well as demographic research are urgently needed in order to understand Temiar health conditions today – and also to allow plans to be developed for future improvements.

For Semai, it seems clear that their biomedical history has been affected by many factors. Ecological, parasitic, cultural, and genetic factors are tightly interwoven, like strands in a mengkuang mat. While all of these factors have been important in shaping Semai health status, social-political forces over which the Semai have no control may take precedence in this regard. To cite one example, Fix (1977) mentioned the encroachment of Malays and Chinese since the 14th century, at first to mine gold. In the 19th century, large-scale tin mining forcibly displaced many Semai in the Perak lowlands. Undoubtedly, food production suffered, stress rose, and infectious novelties abounded. Land expropriation still goes on in Semai country. Semai health in the future largely depends on whether there are improvements in nutrition, sanitation, and concern for women's health – on the basis of the biomedical information now available.

Genetic analysis of the Semai has been rich in detail, but such work would profit by more attention to the relationship of Semai to other (less well studied) Orang Asli groups. Future work in this area could make a key contribution to genetical insights.

Analysis of Semai demography shows that life expectancy at birth was still low in the 1980's: about 35 years. Contrast that figure with the average life expectancy at birth for all West Malaysians in 1985: 68 to 72 years (Ng et al., 1987). The perplexed may well ask how this disparity could exist. Future work on childhood health seems warranted in this regard.

Timeline 2

TEMIAR HEALTH RESEARCH

(excludes review articles; numbered entries in the bibliography are listed in parentheses).

1937 miscellaneous (195)

▼

▼

1940

▼▼

1950

1951 goiter (226)

▼

1953 genetics (239)

▼

▼

▼

▼

▼

▼

1960

▼

▼

1963 dermatoglyphics (125); malaria and filariasis (298)

1964 genetics (157)

▼

▼

1967 filariasis (206)

1968 malaria (49)

▼

1970 blood pressure (35)

▼

1972 malaria (24); nutrition (25); intestinal worms (65); genetics (266; 296)

1973 demography (275)

1974 cardiology (40)

1975 tuberculosis (29); genetics (162)

1976 amebiasis (96); genetics (156); mental health (277)

Colin Nicholas

Medical Transit Center for Orang Asli, Tapah, Perak.
Originally intended to house long-term Orang Asli inpatients and their accompanying relatives, these centers were recently converted into Alternative Birthing Centers in an apparent move to stem the high maternal deaths among Orang Asli women during childbirth. Some have argued that this is a poor alternative to deliveries at home, where the presence and involvement of the family and relatives provide an emotionally more-conducive environment. What is needed instead is perhaps village midwife training and better provisioning of medical supplies.

1977 nutrition (136)
1978 filariasis (172); schistosomiasis (261)
▼
1980
▼
▼
▼
▼
▼
▼
1987 malaria (128)
1988 malaria (149); genetics (153)
1989 malaria (51; 148)
1990
1991 malaria (103)
1992 mitochondrial DNA (20); several diseases (176)
▼
▼
1995 filariasis (110); obstetrics (131a)
1996 tuberculosis (169); filariasis (290)

Timeline 3
SEMAI HEALTH RESEARCH
(excludes review articles; numbered entries in the bibliography are listed in parentheses).

1937 health survey (191)
▼
1939 demography (196)
1940
▼▼
1950
1951 goiter (226); filariasis (227)
1952 tinea (228); hypertension (229)
1953 health survey (231); genetics (239)
▼
▼
1956 dengue (264a)
1957 genetics (170)
▼
▼
1960
▼
▼
▼
1964 genetics (157)
1965 nutrition (52)
▼
1967 mental health (53); filariasis (206)
1968 mental health (54); pentastomids (244)
1969 genetics (28)
1970
1971 demography (73)
1972 malaria (10); nutrition (25); intestinal worms (65); genetics (163; 266; 296)

1973 genetics (293)
1974 demography (74)
1975 demography (75); genetics (88; 162)
1976 amebiasis (96); mental health (277)
1977 demography (76)
1978 genetics (77; 278; 297); filariasis (172)
▼
1980 cardiology (48)
▼
1982 genetics (80; 89); demography (81)
▼
1984 genetics (82)
1985 nutrition (138)
▼
▼
1988 nutrition (129; 139); genetics (153); malaria (175)
1989 demography (84; 85); schistosomiasis (107); nutrition (134)
1990 malaria and filariasis (45)
1991 demography (86); malaria (134)
1992 mitochondrial DNA (20); genetics (91); diseases (176); nutrition (182); illness (269)
1993 diabetes (213); nutrition (215); goiter (217)
1994 genetics (92); malaria (177); goiter (219)
1995 genetics (87; 183; 254); goiter (221); nutrition and disease (222)
1996 malaria (8; 135); nutrition (224; 291)
1997 lipids (37a)

Chapter 4

SMALL CENTRAL AND SOUTHERN GROUPS

The small central and southern Orang Asli groups have a wide range of ecosystems, as implied by their locations on the map (Figure 1). By tradition, particular groups are inland fishers, hunter-gatherers, swiddeners, or coastal fishers. A problem is that far more has been reported about their modes of subsistence than about their diseases, demography, or DNA, as the combined timeline for these groups shows (Timeline No. 4).

SEMELAI

Among the small groups in the center and south of the Malay Peninsula, biomedical information is most readily available on Semelai. Like many other Orang Asli, Semelai have traditionally had a mixed economy of fishing and swiddening, with sidelines in foraging, hunting, trading forest products, and keeping livestock (chickens, goats, and pigs in their case). They now number about 4000.

Semelai are notable among inland groups for several reasons. First, their traditional lifestyle centers on the ecosystem of a marshy lake, Tasek Bera, in Pahang, and particularly on its fish resources. Gianno (1990) discovered that Semelai recognized over 112 different kinds of fish in the Tasek Bera ecosystem, as well as knowing their locations and behaviors. Semelai make and use at least 32 devices (traps, lines, spears) for fishing.

Second, although speaking an Aslian language, Semelai identify themselves culturally with their more populous "Aboriginal-Malay" neighbors, who speak Malayic languages. Indeed, Semelai are at the southern border of the Aslian-speaking world. And, according to Carey (1976), males must be circumcised before they can marry –

following a pre-Islamic tradition and one unknown to, or rare in, other Orang Asli groups, except posssibly the Jah Hut.

No thorough health survey has been published on the Semelai. Exiguous information does exist on the following diseases: trypanosomiasis, filariasis, intestinal worm infestations, malaria, schistosomiasis, and scrub typhus. No information is available on cancer, goiter, leptospirosis, tuberculosis, dengue, and other conditions. Related topics such as psychiatry, demography, genetics, and nutrition have received some modest attention.

Early notes on Semelai health were provided by Morley (1949). He observed much skin disease (tinea), much childhood malaria, and much undernutrition among about 200 Semelai at the new "security" center, Post Iskandar – in all, "a considerable amount of ill health." He attributed this morbidity largely to the fact that Semelai hid in the forest both during the Japanese war (1941-1945) and the disturbed period called the Emergency that followed (1948-1960). Lacking their usual variety of swidden field crops, their diet suffered – becoming mostly cassava and fish.

The first general report on Semelai was by Hoe (1964). While based on a short period of fieldwork, Hoe's report managed to cover a wide swath of Semelai life. Biomedical aspects were not so much overlooked as outside his purview. Hoe was only able to surmise that in the Post Iskandar area overcrowding, malnutrition, and poor housing were party to the many illnesses he observed. Later Dunn (1972) rated the Semelai health environment as "fairly poor," being below average among Orang Asli groups he observed. The Semelai intestinal parasite profile included 6% with Giardia, 70% with hookworm, 71% with Ascaris, and 72% with Trichuris. Only 4% harbored no parasites in Dunn's survey.

In a small sample, Else and coworkers (1976) found 2% of Semelai to have trypanosome parasites. Wharton and co-workers (1963) found 7% malarial parasitemia in Semelai; later Else's group reported 4% and Mak (1978) reported 1%. According to Gianno (1990), malaria was largely eradicated from the Semelai area by 1980, although it seems to have returned in the 1990's. Tan and Armstrong (1976) reported a level of hospital admissions for Semelai psychiatric cases that was similar to that for many Orang Asli groups. While no nutrition study has been published on Semelai, Bolton (1972b) listed their traditional food taboos.

Colin Nicholas

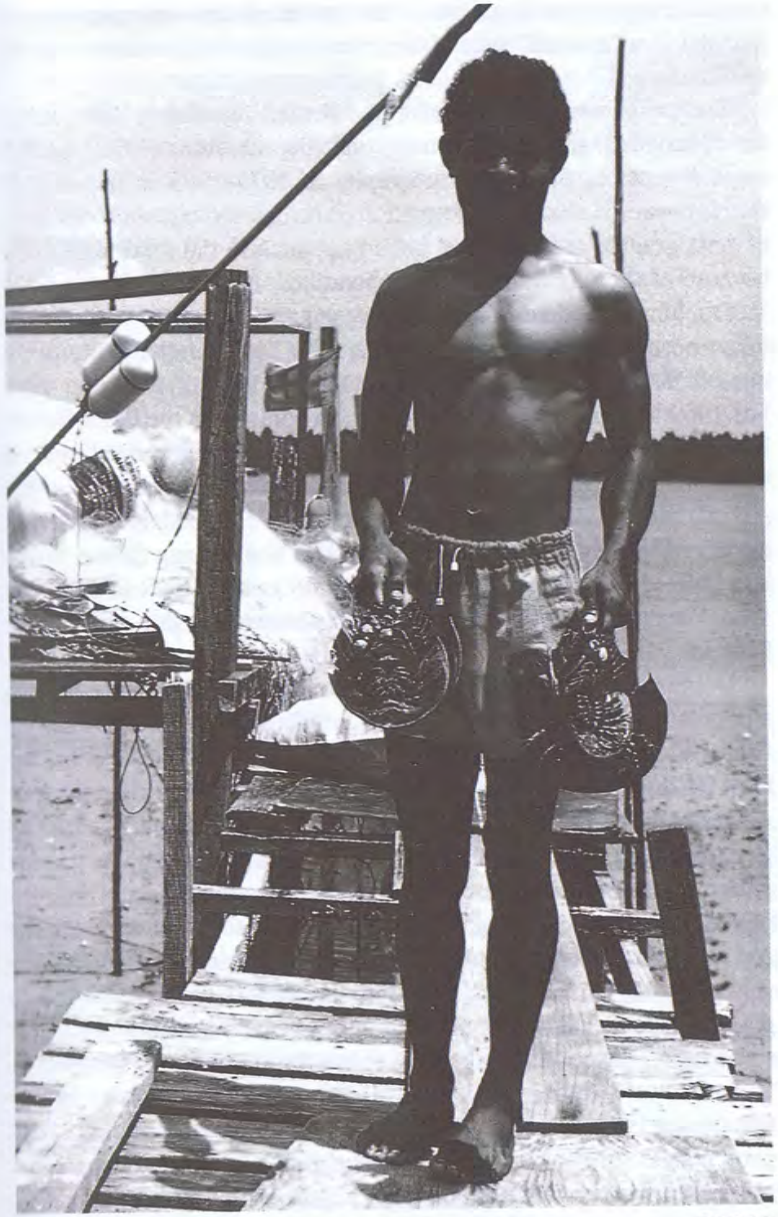
Semelai boy with fish catch, Tasik Bera, Pahang.
The traditional lifestyle of the Semelai centers on the ecosystem of a marshy lake. They recognize over 112 different kinds of fish and, using knowledge of their locations and behaviors, employ at least 32 devices for fishing. The Semelai also cultivate swiddens and engage in foraging, hunting, trading forest products, and keeping livestock.

For filariasis, the lymphatic disease caused by nematodes and transmitted by mosquitos, Onyah (1967) found a 20% parasite rate in Semelai, the highest among the Orang Asli groups he studied. But Wharton and co-workers (1963) found a 15% rate in an earlier survey, a lower rate than found in some Temuan and Temiar communities. In the 1970's Else and co-workers (1976) found an 11% rate and Mak (1978) found a 4% rate. In Mak's study adults had the highest rate, suggestive of their greater exposure from working in wooded areas. There, among other complications, leaf monkeys are found that are natural reservoirs of the parasite. Still, filariasis has probably not been as serious a health problem as have other conditions in recent decades. The extreme form of filariasis, elephantiasis, seems to have always been rare among Semelai.

Greer and Anuar (1984) and Mak (1992) reported that over 25% of the Post Iskandar Semelai had had exposure to schistosomiasis prior to 1975, according to immunological tests. By 1982 the test-positives had dropped to 7% (whether with or without medical treatment was not stated).

Schistosomiasis is a chronic, debilitating disease in which the trematode parasite causes lesions in the liver and other organs, but it is usually not fatal. The infection is often first detected in young children. Fresh-water snails are the intermediate host for the parasite, shedding its infectious stages into water supplies. The parasite enters the human body through the skin, often when bathing. Other animals are reservoir hosts, also acquiring it from water. The Semelai's Tasek Bera and other riparian lowlands and hills provide good conditions for transmission of the disease and constitute endemic areas (Shekhar and Pathmanathan, 1987).

Scrub typhus is the most common cause of fevers in the Semelai world. This acute bacterial disease is transmitted by chiggers, commonly when people work outdoors in brushy areas. Rodents are the reservoir hosts of the parasite. Brown and coworkers (1978) studied scrub typhus in Semelai of the Tasek Bera area and also in Temuan (note 25). By serological tests, they determined that new infections occurred at the rate of 3.9% per month among Semelai. (Treatment was provided.) Their studies also showed that 83% of people over 44 years of age had had prior infections. Among those with new infections, only a few were feverish. This suggests that most of the Semelai infected with scrub typhus were not seriously ill. Perhaps



Colin Nicholas

Besisi youth with horse-shoe crabs, Port Kelang, Selangor.
The Mah Meri, as the Besisi are officially known, are largely coastal dwellers and have greater access to marine foods which are rich in iodine. As such, goiter – a consequence of iodine deficiency – is significantly less prevalent among the Besisi as compared to the other Orang Asli.

paradoxically, the authors found that febrile illness was regarded by Semelai as a "normal" part of life, even though it sometimes leads to death.

Except for the Jehai, Temuan, and Semai, few Orang Asli groups have been studied in depth demographically. Dobbins (1979) studied some aspects of Semelai demography in 1974-1975 in the area of Post Iskandar. He omitted information on birth rates, a basic component of demographic analysis. And he did not analyze the sexes separately because of the small size of the population.

Dobbins estimated Semelai life expectancy to be 54 years for an infant born during the period 1965 to 1974. He found a crude death rate (CDR) for Semelai for the years 1965 to 1969 of 1.1% per year. For 1969 to 1974 the CDR was 0.8%, in contrast to the CDR for the Rual Jehai of 4.5% in the 1980's, as discussed earlier. The Semelai infant mortality rate (IMR) was estimated to be 6.9% per year for the period 1965 to 1974, a relatively low figure considering that Fix (1971) had found an IMR of 20% to 30% for Pahang Semai in the late 1960's. For comparison, Cleves (1990) gave an IMR of 4.1% for West Malaysia for 1970, a rate noticeably lower than that even for the Semelai.

Dobbins did not provide any analysis of fertility. Nor did he furnish a population pyramid of the Semelai population. But Gianno (1990) furnished such a pyramid for the year 1978. As was the case for the Rual Jehai in the same year (Gomes, 1982), about 50% of the Semelai were below the age of 15 years according to the pyramid, indicating an expanding population. And for both Semelai and Jehai, only about 13% of those censused were in their fourth decade or older. However, unlike the Jehai's population pyramid, that of the Semelai does not reveal any preponderance of female mortality during the reproductive years. By 1978, however, the Semelai had been accustomed to governmental medical services for prenatal care and childbirth for some time – much longer than the sedentization period for the Jehai.

In contrast to the above, Hoe (1964) reported 327 males to 275 females for Fort Iskander Semelai, a ratio of 1.19, suggesting relatively poor female survival in the 1960's or earlier.

Gianno (1990) estimated Semelai population densities, increasing from 1.6/sq. mi. in 1965 to 3.2/sq. mi. in 1974. She considered these densities to be low, given the favorable ecological niche of the Semelai. In addition to the constraits of ill health, she mentioned

slave-raiding by Minangkabau and Mendiling Malays up to 1915 as a possible factor that had controlled population density.

Only five genetic surveys have been done on the Semelai. They provide a smattering of results that give mixed signals in terms of relating the Semelai biologically to their cultural neighbors, as shown in Table 6. In comparative terms, Semelai are most similar to Temuan in their ADA-2 and Pep B-6 allele frequencies, most similar to Perak Semai in their Hb E frequency, and most similar to the Jakun of Johore in G6PD-, Gm 1;21, and Gm 1,2:21 frequencies. They are quite different from other groups in their Fy-a frequency. More data are needed to make much sense of this, both in terms of larger samples to be surveyed (to bolster data reliability) and of more genetic systems to be studied.

As Table 6 implies, no studies exist on many Semelai genetic traits. A study of ovalocytosis, for example, would be of great interest in terms of genetic resistance to malaria in the Semelai (see Chapter 7). In a larger context, a systematic, synchronic study of 10 or more genes on Semelai and other neglected groups (Jakun, Jah Hut, etc.) would be a singular contribution to Orang Asli studies.

BESISI AND OTHERS: A DEARTH OF STUDIES

There is little enough that can be said about Besisi (Mah Meri) health, and even less about the health of the Aslian-speaking Semaq Beri, Che Wong, Temok, or Jah Hut, or the Malayic-speaking Orang Kanaq, Orang Seletar, or Orang Kuala. The last three of these groups, all coastal, are sometimes lumped together as "Orang Laut," meaning people of the sea. Mak (1978) was able to survey small samples of some of these seven groups for filariasis and malaria. For Semaq Beri, 10% had filarial worms and 5% had malarial parasites. Che Wong (N=13) and "Orang Laut" (N=16) had no filariasis or malaria. Two of 7 Jah Hut had filariasis but none had malaria. Earlier, a dentist (Mummery, 1948) visited 51 Che Wong, "the whole tribe," and wrote that in this group caries increased with age, fevers were common, and 2 people had yaws.

Carey (1976) implied that Orang Kanaq health was greatly improved in the 1970's when the government furnished them with new houses, wells, and bathing huts, although no corroborative health studies were reported.

The sparse demographic data on Semaq Beri – all that are available – differ little from those on Semelai (Ramle Abdullah, 1991) (note 26). Morris (1997) reported that the Semaq Beri's TB, leprosy, and malaria were treated "only irregularly" and that skin diseases were rampant in a their displacement area.

Polunin (1952a; 1953) studied the coastal Orang Seletar of Johore. They were well nourished and evidenced no riboflavin deficiency or goiter. Skin diseases and lung abnormalities were quite rare. Unlike other groups surveyed, the Seletar had no malaria, filariasis, or yaws. On average, Seletar women reported having had 4.9 ever-born children, but 34% of them had already died, leaving an estimated 3.3 living children per woman. In addition, Polunin and Sneath (1953) reported on 5 blood-group genetic systems in the Seletar.

In 1995, after a long hiatus, the Seletar once again came to the attention of the medical world. Those in Johore Baharu (the group formerly at Setulang Laut) experienced a cholera outbreak in which one person died (out of 40 cases detected) (Roslan Ismail, 1997).

Turning now to Besisi, they are a small, coastal group centered on Selangor's Carey Island in the Malacca Straits. They number about 2000. Traditionally many of them "faced" the sea, being fishers and coastal utilizers, although now they have been greatly dispossessed of these resources. Strauss and coworkers (1969) found no cases of melioidosis among the Besisi. Onyah (1967) reported that 2 of 27 Besisi (7%) showed evidence of filariasis at Judah and Tanjong Sepat villages, and Mak (1978) found 1 of 17 Besisi at Gombak Hospital (6%) with filariasis a decade later. The same 17 people had no malaria.

Tan and Armstrong (1976) recorded hospital admissions for 22 Besisi psychiatric patients over the years 1969-1974. They "explained" this over-representation (1.8% on a population basis) by the proximity of Besisi villages to the Orang Asli hospital at Gombak (35 miles). But the real explanation may lie elsewhere since many villages of the large Temuan ethnic group are even closer to the hospital, and the Temuan had only a 0.2% psychiatric rate during this period.

Recently, Karim (1995) mentioned a viral epidemic in a Besisi village in which 5 infants died, following fever and vomiting. Whether this episode was isolated or part of a larger health problem for the Besisi has not been reported.

There are only three other published biomedical reports on the Besisi. In the first, Polunin and Sneath (1953) studied ABO blood

Table 6
Genetic findings on the Semelai and neighboring groups

	ADA-2	G6PD-	Hb E	Gm 1:21	Gm 1,2:21	Pep B-2	Pep B-6	Fy b
Semelai	0.10	0.09	0.17	0.08	0.04	0	0.004	0.32
Semai, Pk.	0.03	0.22	0.25	0.01	0	0.01	0	0.04
Temuan, S.	0.12	0.12	0.01	0.02	0	0	0.005	0.07
Jakun	0.03	0.08	0.01	0.03	0.05	0	0	0.05
Jah Hut, P.	NA	0.23	NA	NA	NA	NA	NA	NA

Variant allele frequencies are given for adenosine deaminase (ADA), glucose-6-phosphate dehydrogenase (G6PD), hemoglobin (Hb), immunoglobulin G (Gm), peptidase B (pep B), and Duffy (Fy). (NA=not available.) Data for ADA are from Welch et al., 1978; for G6PD⁻ and Hb E from Lie-Injo, 1976, except Jah Hut data from Lie-Injo and Chin, 1964; for Gm from Steinberg and Lie-Injo, 1972; for Pep B from Welch, 1973; and for Duffy from Lewis et al., 1988. Data on Semai, Temuan, and Jakun in reports not dealing with the Semelai are excluded here. Other abbreviations: P. for Pahang, Pk. for Perak, S. for Selangor.

groups. In the second, Lewis and coworkers (1988) surveyed 14 Besisi for Duffy blood group. The frequency of allele Fy-a was similar to that of the other groups surveyed, except for the Semelai. In the third, Osman and co-workers (1995a) found that goiter was only one-fifth as common in the Besisi as it was in the northerners of Baling, Kedah (6% versus 30%). Goiter was attributed to iodine deficiency, even though the iodine level in drinking water in the villages sampled was higher inland than in the Besisi coastal villages. However, Besisi have greater access to marine foods, which are rich in iodine, than do inland groups, the authors suggested – probably correctly. Women were found to have somewhat less bodily iodine than men (urine sample tests).

Iodine deficiency is known to contribute to neurological maldevelopment of offspring in utero or before weaning. It also causes higher levels of goiter in parous women than in men, on average. A complicating factor in Malaysia is the intake of foods, especially cassava, which contains goitrogens (i.e., compounds that sequester iodine). And cassava tuber is a starchy, staple food more of the inlands than of coastal areas. I will return to the topic of dietary iodine in Chapter 8, since it impinges on several Orang Asli health issues.

SUMMARY

The health of the small Orang Asli groups mentioned in this chapter is largely unreported in the biomedical literature. Even for Semelai, it is unclear if their health has improved or worsened from the "fairly poor" conditions reported in the 1970's, since there has been no general report on their health since that time – fully a generation ago.

In addition, none of these groups has received adequate attention demographically or genetically.

No health survey has ever been published on the Temok, Jah Hut, Che Wong, Orang Kanaq, or Orang Kuala, as far as I have been able to ascertain. And since the 1970's only one medical report has been published on any of these small central/southern groups. It was on goiter in the Besisi (Osman et al., 1995a).

All this suggests that a concerted biomedical effort is needed to assess these smaller groups, in order to plan mindfully to ensure their future health.

Timeline 4

HEALTH RESEARCH ON SMALL CENTRAL AND SOUTHERN ORANG ASLI GROUPS

(excludes review articles; numbered entries in the bibliography are listed in parentheses)

1948	teeth (188)
1949	miscellaneous (186; 200)
1950	
1951	goiter (226)
1952	tinea (228)
1953	health survey (231); genetics (239)
▼	
▼	
▼	
▼	
▼	
▼	
1960	
▼	
1962	genetics (287)
1963	malaria and filariasis (298)
1964	miscellaneous (121); genetics (157)
▼	
▼	
1967	filariasis (206)
1968	psychotherapy (236)
1969	whooping cough (114); melioidosis (268)
1970	
▼	
1972	nutrition (25; 90); intestinal worms (65); genetics (266)
1973	genetics (293)
1974	trypanosomes (57)
▼	
1976	trypanosomes (69); mental health (277)
▼	
1978	typhus (32); filariasis (172); genetics (297)

1979 demography (60)
1980
▼
1982 malaria (281)
▼
1984 schistosomiasis (106)
▼
▼
▼
1988 nutrition (144a); genetics (153)
▼
1990 demography (93)
1991 demography (5)
1992 several diseases (176)
▼
▼
1995 goiter (220)
▼
1997 nutrition (186a); malaria (193)

Chapter 5

TEMUAN AND JAKUN

Temuan and Jakun are fairly large southern Orang Asli groups (see map, Figure 1). Biomedical research on these two groups has been asymmetric, as shown in the accompanying timelines (Timelines Nos. 5 and 6). A key problem here is that Jakun have received far less attention than Temuan, despite their near equality in population.

Temuan, Jakun, and several other groups are often lumped together, largely because they do not speak Aslian languages. Rather they speak languages related to Malay. This has led to semantic imagery with such catch-phrases as "Proto-Malay" and "Aboriginal Malay." The "aboriginal" term has not met with much favor, on the argument that the word is pejorative and is, moreover, used inconsistently for some native groups (Australians) but not others (such as Bornean Penan, Filipino Ifugao, or Nagas) (note 27). The term Proto-Malay has similar flaws. Since Malays in Malaysia are by legal definition Muslims, does a Jakun who converts to Islam thereby change from a Proto- to a Deutero-Malay? Biologically speaking, such a religious shift has no meaning. Thus there seems to be no accurate umbrella term for Orang Asli who do not speak an Aslian language. Southern Orang Asli will have to suffice here as a generic term.

According to Carey (1976) the forest-living Orang Asli received better medical services, because of the "flying doctor" and the "posts," than did more accessible Orang Asli – such as Temuan and Jakun. Today, this may or may not be the case, but no reports on this topic appear to be available.

Temuan live mainly in the states of Selangor and Negri Sembilan, with some outliers in Pahang and Malacca. By tradition they are lowland swiddeners and collectors of forest resources with some

hunting, foraging, and fishing on the side. According to official figures, in 1994 they numbered about 16,000.

Since the Temuan live in the prosperous western lowlands near growing cities, they have long feared urban pressures. Universities, airports, and soccer grounds have been built on their lands. The Temuan of Bukit Lanjan once lived in the heart of Kuala Lumpur, then in the area later used for a British club, then at Bukit Nenas, then at Sungai Penchala. Nearly a hundred years ago they were displaced to Bukit Lanjan when the Selangor government leased their land to a quarry company. The Lanjan site is now "growing" a housing estate for millionaires. And the Bukit Lanjan story has a close parallel at Labu, Ulu Lui, Ulu Serendah, and other Temuan communities. From their doorways they now may gaze at a golf course or a drug rehabilitation center. As such, these onlookers of urbanization have experienced some stresses in recent history. No longer able to grow, hunt, or forage for enough food for their subsistence, their physical health may be rapidly changing.

Such changes may not be new. An earlier ecological alteration in both Temuan and Semai country was open-pit tin mining, started some 130 years ago and involving Chinese work crews. More recently, timber clearing, rubber and palm oil industries, urban sprawl, and other upheavels have influenced Temuan (and other Orang Asli) lives.

An early non-ecologial event was slave-raiding by Malays. Later I review this misfortune in terms of malaria-related genes (Chapter 7). The memory of that time is still very much alive in Temuan villages in Selangor.

Dunn (1977) gave an overview of Temuan subsistence and history. According to him, many Temuan lands were taken away decades earlier for plantation agriculture, tin mining, and settlements. Then during the war years (1942-1945), most of the Temuan retreated into the rainforest, living from hand to mouth without large-scale food cultivation. Later, some Temuan were displaced during the so-called Emergency years, when anti-government guerrillas roiled the countryside.

Temuan villages in recent decades have been like islands in a sea of other people. But Temuan have a penchant for "island hopping." Up to 20% of a community may be absent at any one time, often visiting relatives elsewhere. Such mobility, of course, is a boon to viruses and other go-along parasites.

A. Baer

Temuans in Ulu Tamu, Selangor, 1972.
Temuan demographic data for the 1970s reveal a high maternal mortality in pregnancy and childbirth, resulting in a lower mean age for women, suggesting some survival disadvantage for women.

Dunn (1977) discussed the psychosocial stress on Temuan as islanders. He wrote (p. 91):

> "As populations exceed the carrying capacity of the modern, much reduced subsistence zones, what will happen to the inevitable out-migrants? Those who remain behind will no doubt perpetuate a Temuan ethnic identity so long as the 'islands' survive, but will those who leave remain 'islanders' wherever they may go? Where can they go? Where should they go? I see all of these interlocking questions as relevant to the broadest view of Temuan health."

TEMUAN HEALTH

Probably the first Temuan health disaster to be recorded was the smallpox epidemic of 1947 (Williams-Hunt, 1952). It is also known that a whooping-cough-like epidemic occurred in northern Selangor Temuan in 1971, with several dozen childhood deaths.

Polunin (1953) observed a few medical problems among Selangor Temuan, although his surveys of Temuan were less thorough than of some other Orang Asli groups. He reported finding an infant with vitamin-D deficiency (rickets) and 2 cases of ascariasis (among 14 people tested) at Ulu Langat. At Ulu Kali, 3 of 3 Temuan tested had ascariasis and 2 of 7 tested had dysentery. Dengkil had 2 cases of leprosy. A boy at Bukit Mandol was diagnosed as having severe anemia. However, Polunin judged that the Temuan relocated at Kuala Kubu Bahru were better off than Orang Asli he had seen at other round-up areas; at least their birth rate was keeping up with their death rate.

In 1988, among 137 Selangor Temuan surveyed at Ulu Kuang and Bukit Lanjan, 5 (4%) had new tuberculous infections, but fewer had leprosy, according to a newspaper report (Nicholas et al., 1989).

In 1966, Kinzie and coworkers reported on the health of Temuan at Bukit Cheeding, Selangor. They considered nutrition to be adequate (without presenting any data), except for the possible deficiency of B vitamins. None of the Temuan were anemic, goiterous, or diabetic. While 17% had microfilariae, only 2% had elephantiasis.

Despite being on malaria suppressant drugs, 14% had malarial parasitemia, mainly the falciparum type. A vast majority needed dental "repairs." Intestinal infections were common: 88% had Trichuris, 53% had Ascaris, 58% had hookworm (with 70% in the 6 to 16 year age group), 9% had threadworm, and 11% had Giardia. And 20% had otitis media. Among the children surveyed, 39% had upper respiratory infections.

Weerekoon (1973) reported on the eye conditions occurring in 617 Orang Asli, including 166 Temuan. Few medical problems were found, but the Temuan had a disproportionate number of blind people. Overall, most cases of Orang Asli blindness (11 of 16 cases) were in one eye only, arising from injuries.

Dunn (1977) rated the health environment of Temuan as "fairly poor." While he regarded malaria as being a problem coming under control for Temuan, this opinion was rather optimistic, as I discuss later in Chapter 7. He suggested the Temuan's high prevalence and intensity of intestinal worm infestations (hookworm, 79%; Ascaris, 59%; Trichuris, 91%) was relatively recent, as they increasingly became hemmed in by "development." Yet less crowded Orang Asli groups, or communities, also show high worm prevalences, as I have mentioned elsewhere in the text.

Dunn (1977) also remarked on another hazard of Temuan life, "a high rate of maternal mortality in childbirth." He opined that such mortality had also been a problem for other Orang Asli women in the past. He further reported that infections accounted for 61% of diagnoses of Temuan patients at Gombak Hospital, according to hospital records.

On the subject of mental health, Tan and Armstrong (1976) reported that Temuan had an average amount, for an Orang Asli group. While these authors recorded little epilepsy, in the 1970's I myself knew of three cases among some northern Selangor Temuan, one child and two adults.

Brown and coworkers (1978) studied Bukit Lanjan Temuan (and also Semelai at Tasek Bera) for scrub typhus infection by serological means. Its incidence was calculated to be 3.2% per month in 1974-1975 for Temuan, slightly lower than that found for Semelai.

As mentioned in Chapter 1, Orang Asli are highly exposed to dengue infections. Rudnick and coworkers (1986) studied antibodies to dengue virus in blood sera collected in 1968 at Gombak Hospital from 175 unspecidied but healthy Orang Asli. Of these, 82% showed evidence of past dengue infection. Exposure to the virus increased with age. These authors particularly studied the Temuan village of Tanjong Rabok, Selangor, located in a swamp forest that harbored various monkeys, but they did not report on these Temuan results

separately (note 28).

Thomas and Sinniah (1982) found that over 8% of 84 Temuan at Bukit Lanjan, Selangor, had *Entameba histolytica* infections, but none had acute amebic dysentery.

Mak (1992) reported that among 350 Selangor Temuan at Bukit Kemandul in 1989, 69% had Ascaris, 97% had Trichuris, and 48% had hookworm. In addition, 10% of these Temuan had had exposure to schistosomes, based on an immunological survey in 1986.

Osman and coworkers (1992, 1993c) found 27% of the Kuala Pangsoon Temuan they studied and 11% of the Bukit Lanjan Temuan to be goiterous. At Lanjan the condition was shown to increase with age, reaching 24% in those over 17 years. In addition, Osman's team (1992) reported that for Pangsoon Temuan 7 years or older, females had a "mean hemoglobin level" of 9.9 g/dl, much lower than for males (12.7 g/dl), a level indicating much anemia in females.

Mohamed Kamel and colleagues (1994) surveyed Pangsoon Temuan in 1993, finding that 82% of those surveyed had either a helminthic or protozoan infection intestinally. About 7% had amebiasis, 20% had cryptosporidiosis, 7% had Giardia, and 71% had intestinal worms.

More recently, Norhayati and coworkers (1995) surveyed Temuan children 0 to 13 years of age in the area of Dengkil, Selangor. They found 31% of 193 children had hookworm infestations. After providing treatment (albendazole), the research team studied the reinfection rate. At 4 months post-treatment, the rate was 30%, still quite high.

While the dental health status of Orang Asli groups has largely been ignored, several recent reports exist on the Temuan (Abdul-Kadir and Adnan, 1989; Abdul-Kadir and Yassin, 1989 and 1993). Adults were not studied clinically, and no mention was made of periodic dental treatment being provided to Orang Asli. However, 25% of the Temuan children surveyed had periodontal disease and some 37% had caries or other defects. Adults were, however, questioned about their dental problems and practices. Of those with toothache who consulted the dentists at Gombak Hospital, over 70% were not given restorative treatment (fillings), but the surveyers could only speculate as to why no treatment was given. They went on to suggest that static clinics were unsuitable for Orang Asli dental care and suggested mobile clinics as being most appropriate (Abdul-Kadir and Yassin, 1993).

Earlier, Kinzie and coworkers (1966) had found 71% of Temuan and 64% of Jehai had caries. In another report on dental health already

mentioned (Chan et al., 1974), 30% of the Temiar boys and 50% of the Temiar men studied had caries. And Khor (1985) found at least a third of Semai children in her Perak sample had caries. Based on my own observations, Orang Asli adults commonly have caries, as well as "lost" teeth.

Veeman (1986/87) stated that dental services were provided to Orang Asli admitted to Gombak Hospital and to people in "jungle" villages via mobile health teams. By subtraction then, the remainder, constituting the majority of Orang Asli, are not given dental services.

TEMUAN DEMOGRAPHY

According to official censuses, the Temuan population growth rate has averaged about 3% per year since the 1960's.

Temuan demographics has received some attention. Table 7 gives abbreviated population profiles of the Temuan of Paya Lebar, Selangor (Gomes, 1982), and of Tekir (or Tikir) Labu, Negri Sembilan, as well as three villages in Ulu Selangor (Ulu Serendah, Ulu Kuang, and Bukit Legong) in the 1960's and early 1970's (Baer, unpublished). The profiles are much like that of the Batang Padang Semai of Perak in the 1980's or of the Jehai during the period 1978-1988. These samples of Jehai, Perak Semai, and Temuan appear to have had a much younger population than the Pahang Semai studied by Fix (1977). Among these groups, the Temuan had perhaps the highest percentage of children. This may be the result of several demographic forces. Such forces might include high fertility, relatively good childhood health, high mortality of adults, and a cluster of young families moving into some villages. Here we are in the realm of speculation, however, as the study of Temuan demography has had little longitudinal perspective.

Dunn (1977) mentioned high maternal mortality in pregnancy and childbirth for Temuan women, based on his unpublished population data that showed substantial losses for all female cohorts over 30 years of age. He also mentioned that Temuan rarely survived past the age of 60 years, without specifying the size of his sample.

My own unpublished observations for 1972 were based on a sample size of 452. For three Ulu Selangor villages plus Tekir Labu in Negri Sembilan, the mean age of females was 17.5 years and of males was 20.1 years, suggesting some survival disadvantage for females. The overall sex ratio was 1.09 males to 1 female. The sex ratio for the 0

Table 7
Temuan versus Semai, Jehai, and Jakun population structure

	% less than 15 years old	% over 50 years old
Temuan, Tekir Labu: 1965	52.7	4.8
Temuan, Tekir Labu: 1969	59.0	5.4
Temuan, Tekir Labu: 1972	57.1	3.8
Temuan, Ulu Selangor: 1972	49.1	5.1
Temuan, Paya Lebar: 1978	48.8	3.5
Semai, Pahang: 1965	35.1	7.8
Semai, Satak area: 1969	36.3	8.2
Semai, S. E. Perak: 1980's	50.5	5.8
Jehai: 1978	50.8	7.3
Jehai: 1988	41.2	5.5
Jakun, Langkap: 1990's	49.5	11.0
Jakun, Batu Tiga: 1990's	44.1	14.2

Data on Temuan from Table 2 in Gomes, 1982, and from Baer, unpublished; on Semai from Table 4.4 in Fix, 1977, and from Table 6 in Khor, 1985; on Jehai from Table 2 in Gomes, 1990; and on Jakun from Siti Noor, 1996.

to 20 year group was 1.01; for the 21 to 40 group, 1.05; and for those over 40 years, it was 1.79. Only 5 Temuan were over the age of 60, four of them men. These data tend to support Dunn's comment about poor female survivorship into older ages among Temuan.

Gomes (1982) found a greatly skewed sex ratio for 86 Paya Lebar Temuan in 1978: 1.40 males to 1 female. For those 0 to 19 years of age, the ratio was 1.27; for 20 to 39 years, it was 1.38; and for those over 39, it was 1.83. These data confirm the poor survivorship of older Temuan women noted earlier. Only 3 people in 1978 were in their 50's and none were older, perhaps reflecting Temuan deaths during the period 1900 to 1920 caused by migrant Malays who coveted the Temuans' land.

Based on related information (Baer, 1988), perinatal and childhood mortality is probably substantial among the Temuan, although reliable data are scarce. For a sample of 18 ever-married Temuan women under the age of 40, 31% of their ever-born children had predeceased them. For 30 ever-married women of all ages, the average number of live births was 4.3 (Baer, unpublished). Three of the 30 women may have been sterile. Because 31% of the children of these 30 women were dead, their average number of surviving children in 1972 was 3.0. The younger women in this group could be expected to suffer more offspring deaths in their lifetimes. This would drag the average number of children surviving to adulthood down below 3.0.

For 16 postmenopausal Temuan women in Paya Lebar, whether living or dead in 1979, Gomes (1982) found that they averaged 5.1 live births. On average, only 3.25 offspring survived at the time of Gomes' study, giving a 36% death rate. For a comparable Rual Jehai sample, there were fewer live births (3.7) and a 33% death rate.

Based on these data, infant deaths were 5.3 times more common among the Temuan than the Jehai. Moreover, childhood deaths (for 0 to 14 years) were 2.1 times more common among the Temuan. Thus while the Temuan had more children than did the Jehai, their early-life death rate was also higher (note 29). In this connection, it is relevant that the Temuan say their first-born children often lack *bunga* (the word for flower) and thus die in childhood. Like trees, children must first flower before they *buah* (fruit).

Over the period 1957-1979, the crude death rate for Temuan in Paya Lebar was 2.4% per year, lower than the rate for the Rual Jehai that Gomes had also studied, but higher than the Semelai or the national rate (Table 8). (It is worth recalling that the Jehai CDR for 1978-1988 was 4.5%.) While no data are available on the Semai for the 1970's, their crude death rate in Satak, Pahang, for 1955-1969 was 3.0% (Fix, 1971).

In an important further finding, Gomes (1982) reported that most Temuan deaths (94%) and most Jehai deaths (96%) were attributable to disease. But for Temuan 15 to 44 years of age, death took a higher toll among women than men: 16 to 2 for the Temuan and 27 to 21 for the Jehai. Some of these 43 female deaths occurred during childbirth.

Table 8
Annual crude death rate (CDR) estimates

	CDR	Reference
Jehai, Sungai Rual, 1956-1979	3.3%	Gomes, 1982
Temuan, Paya Lebar, 1957-1979	2.4%	Gomes, 1982
Semelai, 1965-1974	1.0%	Dobbins, 1979
Orang Asli, 1984-1987	1.0%	Ng et al., 1987
West Malaysia, 1970	0.7%	Gomes, 1982
West Malaysia, 1989	0.5%	Ng et al., 1987

TEMUAN GENETICS

Polunin and Sneath (1953) initiated genetic studies on Temuan, investigating 9 blood-group systems.

A striking feature of the Temuan gene pool is its high frequency of the trait called ovalocytosis (OV). The OV allele provides resistance to malaria, as discussed in Chapter 7. Among Temuan, the evidence suggests that OV enhances female fertility, in part because women having the OV trait live longer than those that lack it (Baer, 1988).

Another genetical feature of Temuan is their genic microdifferentiation among villages. For example, the village Tekir Labu lacks the alleles for hemoglobin E and for PGD-C, while Ayer Banning has no colorblind males and Senebai lacks G6PD- males, according to surveys conducted in the 1970's (Baer et al., 1976).

By conglomerating the Selangor Temuan in terms of some genetic findings (as shown in Table 11 in Chapter 7), it is apparent that they have less hemoglobin E than Aslian speakers such as the Semai and Semelai. But they have more than the Jakun. While the Selangor Temuan are similar to neighboring groups in some other regards, both they and the Senebai and Tekir Labu Temuan in Negri Sembilan have remarkably high frequencies of ovalocytosis.

The Temuan say they do not marry relatives such as close cousins. And, indeed, no first-cousin marriages existed in the villages I came to know well in the 1970's. But Temuan tend to marry other Temuan. Among the 152 Selangor people living in Ulu Serendah, Ulu Kuang, and Sungai Klubi in 1972, all marriages were among Temuan except

for one involving a Semelai man. That marriage was childless.

Outbreeding of Temuan to other ethnic groups perhaps occurred more commonly in the recent past (note 30), as an addendum to nation-level warfare. Three of the 65 adults in the above-mentioned villages, none closely related, had Chinese fathers in the 1970's. Another adult had a Malay father. The overall ancestry of this Temuan subpopulation (adults and children) by ethnic group was 94% Temuan, 4% Chinese and 2% Malay. That is, the known gene flow, as it is called, into the Temuan gene pool was roughly 6% at that time. The amount of gene flow from the Temuan out to other groups was not determinable.

The only genetic distance analysis of a general nature relevant here is one by Lie-Injo (1976) which, however, comingled Temuan and Jakun information. For this and other reasons, its usefulness is doubtful.

More recently, a few data on the HLA genetic system were published on the Temuan (Hirayama et al., 1996).

JAKUN HEALTH

Jakun live mainly in the states of Johore and Pahang. They now number over 16,000. Far less has been published on them than on Temuan. And no general health survey has ever been carried out on Jakun, to my knowledge.

In surveys of intestinal worms, Dunn (1972) found Jakun to be somewhat less similar to Temuan than to Semai. He rated the Jakun health environment as "fair." For hookworm, the prevalence rates were 64%, 79%, and 66% for Jakun, Temuan, and Semai, respectively. For Ascaris, 65%, 59%, and 40%. And for Trichuris, 62%, 91%, and 50%. Only 8% of Jakun lacked any kind of intestinal parasite. Earlier, Sandosham (1954) had found among "Aboriginal Malays" of the Lower Pahang River – most likely Jakun – 49% with hookworm, 80% with Ascaris, and 24% with Trichuris. In some 20 years, then, hookworm and Trichuris prevalences in Jakun may have risen and that of Ascaris fallen.

My earlier discussion of filariasis omitted Jakun (Table 2), given that a fair proportion of their settlements are coastal. Mak (1978) found a microfilaremia rate of 9.4% for 502 Jakun in Endau, Johore. Earlier Onyah (1967) had found a 15% microfilaremia rate (N=20) for Jakun (for Endau, Segamat, and Lenek, Johore). And Polunin

(1951) found a rate of 17.9% for Lenek, with 2 cases of elephantiasis.

In terms of the reported burden of psychiatric illnesses in the 1970's, Jakun and Temuan were both about average among Orang Asli groups, with hospitalized psychiatric patients accounting for about 0.2% of their population (Tan and Armstrong, 1976).

In some areas, poor nutrition has been recognized as a problem for Jakun. Food baskets were given by the government for Jakun childhood nutrition at Langkap, Pahang, in the 1990's (Siti Noor, 1996). These baskets, however, did not contain the full complement of items listed in an official report (Tee and Cavalli-Sforza, 1993). In particular, the Jakun baskets contained no green gram (mung beans/ kachang hijau) and no oil, only two-thirds the official amount of sugar and of biscuits, and vitamin C alone, rather than multivitamins.

Lim (1997) studied 34 reproductive-age Jakun women in two Pahang villages, Sungai Soi and Batu 15, as to their health and reproductive status. Of these 34 women, aged 16-44 years, 26 were ever-married. By means of open-ended interviews, Lim discovered that one of the women was under treatment for leprosy and a second for gastritis. One of the 26 had had a stillbirth and three had had a miscarriage. None of the women observed any food taboos during pregnancy but all did so postpartum. Among the 34 women interviewed, 91% had calorie deficits (in terms of the Malaysian recommended daily allowance, or RDA). In addition, all were iron-deficient, 65% were protein-deficient, and 74% were calcium-deficient. At least 50% were deficient for four vitamins: A, C, thiamine, and riboflavin.

No demographic information has been published on Jakun. Siti Noor (1996) recorded a sex ratio of 1.27 males per female for 109 Jakun living at Langkap village and a ratio of 1.17 for 315 Jakun living at Batu Tiga, both in Rompin District, Pahang. These ratios suggest poorer health for females than males. Some aspects of Jakun population structure are presented in Table 7.

JAKUN GENETICS

As was shown in Table 6 on Semelai genes, Jakun have been studied in relation to other Orang Asli populations to some extent. Jakun allele frequencies appear to be similar to those of their Perak and Selangor neighbors, based on this table.

Lie-Injo (1976) summarized some genetic informaiton on Jakun

Table 9
Allele frequencies for PGD (6-phosphogluconate dehydrogenase) and PGM1 (phosphoglucomutase-locus 1) in Jakun and other Orang Asli groups, based on data from Lie-Injo, 1976

	PGD-C	PGM1-2	PGM1-7
Jakun	0.052	0.196	0.024
Temuan	0.049	0.101	0
Semai, Pahang	0.038	0.476	0.016
Semai, Perak	0.038	0.208	0.013
Temiar	0.064	0.356	0.017
Northerners	0.050	0.221	0

(and other Orang Asli groups), including the small amount of blood-group data provided by Polunin and Sneath (1953). In addition to data on Hb E and G6PD deficiency, shown in Table 6, she reported on the allele frequencies of two other malaria-related alleles in Jakun: OV (ovalocytosis) with a frequency of 0.048 and Hb CoSp (Constant Spring) with a frequency of 0.013. The Jakun are strikingly unlike their Temuan neighbors with respect to their OV frequency (see Table 11 in Chapter 7). Unfortunately, these malaria-related genetic data on Jakun are based on unreported sample sizes and, at least in some cases, are based on blood samples from hospital patients rather than on village surveys. The results may therefore be biased in unknown ways.

Lie-Injo (1976) also provided information on PGD (6-phosphogluconate dehydrogenase) and PGM1 (phosphoglucomutase-locus 1) for the Jakun and other groups, as shown in Table 9.

SUMMARY

Temuan and Jakun are mainly inlanders, but quite a few Jakun communities are coastal. The villages of these southerners tend to concentrate in the lowlands of the Malay peninsula. As such, the social and physical environments of Temuan and Jakun have been quite different from those of more forest-dwelling or highland groups during the last century or so. Surrounded more by non-Orang Asli communities, Temuan and Jakun have long been exposed to epidemics

that maundered through this region. They have absorbed various nutritional habits of other groups; some of them, for example, may refrain from eating useful sources of animal protein such as monkey or pork because of the stigma attached to this practice by Muslim neighbors. These are just a few details of the biomedical setting in which Jakun and Temuan "adapt" to outside society.

Hookworm is one example of a serious medical problem for Temuan; it is a known cause of anemia. To recap, in the 1960's 58% of the Temuan studied had hookworm (Kinzie et al., 1966); in the 1970's 79% had it (Dunn, 1977); in the 1980's 48% had it (Mak, 1992); and in the 1990's 31% of Temuan children had it (Norhayati et al., 1995). These data suggest some improvement, but, as Norhayati and coworkers emphasized, 6-monthly chemotherapy control programs are ineffective in preventing high rates of reinfection. These authors advocated a long-term strategy to include hygiene education and the provision of toilets and safe water supplies for Orang Asli villages.

In some parts of the Temuan-Jakun range, malaria is a critical problem. As malaria is the subject of Chapter 7, however, I will defer comment on it.

A key problem is that the health status of the Jakun has been only thinly reported, but what is known is not reassuring. In contrast, a fair amount is known about Temuan health. In both cases, however, it is unclear if these groups have received as much attention in health services as have more remote groups. In addition, topics such as nutrition appear to have been poorly studied on them. And for the Jakun, malaria data are virtually nonexistent.

For the future, I would suggest that lacunae in biomedical information – especially on Jakun – be remedied promptly. Afterwards, it will be possible to establish a priority list of actions to improve the health status of these two groups.

SUMMARY OF GENETIC STUDIES ON ORANG ASLI GROUPS

This chapter is the last one on particular Orang Asli groups. It may thus be appropriate to summarize the genetic findings that have been presented in Chapters 2 through 5. Excluding the malaria-related genes that are discussed in Chapter 7, relevant genic information is quite sparse. What there is suggests that each group is somewhat genetically differentiated from the others.

Chapter 2 on the northerners mentioned the many genetic traits studied in the 1950's on Orang Asli groups. Yet since the 1970's virtually no genetic information has been forthcoming on any northern Orang Asli group. The same statement holds for Temiar, except for one report in the 1980's on Duffy blood group. In contrast, genetic reports on Semai continue to appear; so it is quite possible to compare Semai with other ethnic groups, both near and far (as was done in Chapter 3). Also, important molecular studies have recently appeared on Semai, principally on mitochondrial DNA, that link them to Southeast Asians generally. All the same, because other Orang Asli groups are less well studied, the genetic relationships among them, or between them and the Semai, remain elusive.

Still, genetic data on Semelai suggest they are somewhat distinctive from other Orang Asli groups for several traits (as shown in Table 6). Data on Jakun (Tables 6 and 9) also indicate intra-Orang Asli distinctions.

Finally, data on Temuan in the same two tables, as well as elsewhere in this chapter, indicate a like measure of genetic separateness. But Temuan are most distinctive genetically in terms of their frequency of ovalocytosis. This trait, and others involved in surviving malarial infections (discussed in Chapter 7), provide the best genetic evidence at hand that many – perhaps all – Orang Asli groups have been localized in the malaria belt of Southeast Asia at least for much of the Holocene period of geological time. Indeed, genetic and other evidence suggests their continuity in this environment for longer than 10,000 years ago, that is, back into the Pleistocene era (Baer, 1995).

Timeline 5

TEMUAN HEALTH RESEARCH

(excludes review articles; numbered entries in the bibliography are listed in parentheses)

1950
1951 goiter (226); filariasis (227)
▼
1953 genetics (239)
▼
▼
▼
▼
▼
▼
1960
▼
▼
1963 malaria and filariasis (298)
1964 genetics (157); filariasis (248)
1965 malaria (185a)
1966 malaria (123); health survey (142)
1967 filariasis (206)
1968 toxicaria (22)
1969 whooping cough (114)
1970 pentastomes (259)
▼
1972 nutrition (25); intestinal worms (65); genetics (163; 266)
1973 malaria (151); genetics (160; 293)
▼
1975 malaria (152); genetics (162)
1976 genetics (19) mental health (277)
1977 intestinal worms (67)
1978 typhus (32); filariasis (172); genetics (297)
1979 demography (99)
1980
▼
1982 filariasis (178); amebiasis (283)
▼

1984 schistosomiasis (106)
▼
1986 dengue (253)
1987 malaria (179)
1988 genetics (16; 153); intestinal worms (271)
1989 teeth (1; 2); intestinal worms (270)
1990 teeth (3)
1991 nutrition (211)
1992 several diseases (176); nutrition (212)
1993 teeth (4); diabetes (213); nutrition (214; 215); goiter (217)
1994 intestinal worms (184)
1995 intestinal worms (198); goiter (221)
1996 nutrition (224)

Timeline 6

JAKUN HEALTH RESEARCH

(excludes review articles; numbered entries in the bibliography are listed in parentheses).

1950
1951 filariasis (227)
▼
1953 genetics (239)
▼
▼
▼
▼
▼
▼
▼
1960
▼
▼
1963 malaria and filariasis (298)
1964 genetics (157); dengue (264a)
▼
▼
1967 filariasis (206)
▼
▼

▼
1970
▼
1972 intestinal worms (65); genetics (266)
1973 genetics (160; 293)
▼
1975 genetics (162)
1976 mental health (277)
▼
1978 filariasis (172); genetics (297)
▼
1980
▼
▼
▼
▼
▼
▼
▼
1988 genetics (153)
▼
1990
▼
1992 mitochondrial DNA (20)
▼
▼
1995 pentastomes (6)
1996 demography (263)
1997 nutrition (166)

Chapter 6

OTHER INSIGHTS

This is a convenient midpoint for considering some informal perspectives on Orang Asli health. Glimpses of daily life can sometimes be revealing about biomedical issues. In other chapters I have mentioned some important external factors. These include governmental expropriation of Orang Asli forest, farm, and orchard lands; the pollution of potable water supplies by outsiders; the poor availability of iodized salt in country stores; and cultural discrimination. This is not an exhaustive list. Such factors contribute to malnutrition, epidemics, intestinal infestations, psychological stress, and death.

Among positive factors for Orang Asli survival are the way traditional houses are sited and built; the healthy variety of traditionally foraged foods; and the community spirit that shares out food and produces cooperation in caregiving when illness occurs. This also is not an exhaustive list (note 31).

Based in part on personal observation, in this chapter I give several sketches on Orang Asli life. They show both its vitality and its vulnerability to the forces beyond the control of Orang Asli communities.

THE TEMUAN
The Story of Nah's Grandfather

Nah's grandfather with his third wife, 1972 A. Baer

I met Nah in 1971 in northern Selangor. She was, and is, the first wife of the headman (*batin*) of her village.

During the year Nah and I were neighbors, she helped me to learn Temuan culture and its domestic landscape, among her many duties. Like many Temuan, she was a hard worker. She was almost always busy in her family's vegetable field (*ladang*), washing children or clothes, weaving beautiful pandanus mats, making basketry backpacks, foraging for cassava tubers, fishing, cooking, or helping to build houses. Including the house built for me.

In 1971 Nah's village was in an ecologically precarious state, with an alkaline tin-mining pond and tin tailings on one side and townspeople moving closer on another. A rutted road ran through the village, a momento of upstream logging. Game was scarce. But the river ran clean – except after deluges. Hillside springs produced good drinking water and bathing nooks. There was enough land for ladangs, and ancestor-planted durian and petai trees existed nearby. A special treat was the upstream waterfall where the Temuan children played in carefree abandon.

Nah had four living and amiable children. After many months of friendship, I learned that a fifth had died at a young age, years earlier. She still mourned his loss, in her quiet fashion.

That year I was conducting malarial, medical, and genetic surveys of several Temuan villages. This work was done in conjunction with Allan Lewis, an M.D. then at Gombak Hospital, technicians from the malaria unit of the Institute for Medical Research (IMR) in Kuala Lumpur, and Lie-Injo Luan Eng, an M.D. then head of the hematology and genetics unit at IMR. Medical services were part of the survey effort.

One day I mentioned to Nah that our survey team would be going to a village some 10 miles distant. She was pleased and wanted to come along. Her grandfather lived there, she said (note 32).

Based on the Temuan geneologies I had compiled, helped by Nah and others, it was clear that Nah's grandfather had at least 50 great-grandchildren, a formidable number. I was eager to find out if he had a history of malaria or other health problems and – because I was a geneticist – if he had some malaria-resistant genes.

On the appointed day, we all assembled at the village, and to my delight Nah's grandfather was at home and hale. Even a bit frisky, for all he looked to be perhaps 70 years old. Nah introduced us.

I soon learned he had strong views on a number of subjects. When asked his age, he said 100 years. When I demurred at the unlikelihood of this statement, he showed me his "identity card." It officially proclaimed him to be 94, but I wasn't going to quibble. Birth certificates not being in vogue much in the 1970's, let alone the 1870's, verification would not be easy.

Then we got down to the reason for the visit by all the outsiders that day, and I asked him to provide a finger-prick blood sample. What for? he wanted to know. For a health survey and a survey of malarial parasites, I replied.

He said: I never get malaria, never get sick. I doctor myself, I always have.

One moral of this story is that I received "informed dissent." I never got a blood sample from Nah's grandfather, although he sent me on my way with a twinkle in his eye. I never found out what genetic traits (if any) made him so successful as a patriarchal figure. Surely it was not worldly goods, since he was dressed almost in rags and his house was as bare as old tin tailings. And I had been told, or told off, that there is more than one way to a healthy (and long) life. Grandfather's way!

Commentary:
My initial interest in Nah's grandfather was "scientific," in the sense that if he had had some anti-malarial alleles, it would have helped to explain to me his longevity (and fertility?). The interest of local Temuan in Nah's grandfather was more respectful, in the sense that they simply recognized and accepted his dignity and individuality as a human being.

A Temuan Story of Giant Problems

The *Gasi* are giants. They have tusks like an elephant or wild boar. Their bodies are as large as half the world. When they wade through the ocean, the water only comes up to their ankles. There are two *Gasi*, one man and one woman. They are *nyongal*, having no parents and no children. They kill and eat people.

Their black hair is beautifully curled, *maiyang*, like the curls of goat hair. Like the hair of Jakun, very curly. They are fat and large. When they walk, one stride takes them from here to Rawang, another from Rawang to Batu Caves. With a third stride, they reach Kuala Lumpur.

Today they live on a faraway mountain, banished there by God because of their evil ways. When they ate a whole village, it was not enough. When they ate all day, it was not enough. Just as we can get a sliver of meat caught between our teeth, they could get a whole lower leg caught between their teeth. Among us, who has teeth like that?

Commentary:
The term gergasi comes from the Sanskrit, referring to a tusked giant of legend. The ancestors of the Temuan and of other Orang Asli may have been introduced to such Indian ideas a thousand or more years ago when Hinduized states first arose in Southeast Asia. Possibly the gasi still serve Temuan as a metaphor for deadly forces over which they have no control.

A. Baer

Traditional Temuan hut in forest swidden, Ulu Serendah, 1972.

THE SEMAI
The Vitality and Death of Bek Enda

Colin Nicholas

Bek Enda and his youngest son, 1996.

Bek Enda, the father of Enda, had a pure and unwavering interest in preserving and promoting tradition-al Semai culture. He died in June, 1996, at the age of 35, after three days of fever and headache – not an uncommon prelude to death (note 33).

His wife and six children survive him, with difficulty. Bek Enda lived in a hamlet in the upper reaches of a small river in Perak. The people there have not succumbed to governmental promises of betterment if they move into a "modern" village.

In this up-river hamlet, in the past, the music of Bek Enda could often be heard, as he played his nose flute. And often heard was the chop-chop sound as he felled trees with the traditional whippy axe in preparing a new swidden field.

Bek Enda, like many a Semai father, did a lot of "baby sitting," by carrying his infant son in a cloth sling anchored on one shoulder. Since Bek Enda had six children, and since Semai youngsters are treated fondly by their parents, a major factor for his children's health was the attention of both parents prior to his death.

Commentary:
Bek Enda's illness could have been caused by scrub typhus or by several other diseases. Scrub typhus is difficult to diagnose because its possible spectrum of fevers, headaches, chills, and cough are also suggestive of other problems. Hence case rates are often understated. Antibody against the typhus parasite was found in 73% of "deep jungle" (remote) and 48% of "jungle fringe" adult Orang Asli, some of whom were Semai (Cadigan et al., 1972). According to Brown and coauthors (1978), the clearing of areas for hill gardens (selai *is the Semai term) disturbs the rats that carry the chiggers that harbor the typhus parasite. The fact that Bek Enda was clearing a* selai *for two weeks before he died may thus be germane.*

THE JAH HUT
Vital Signs

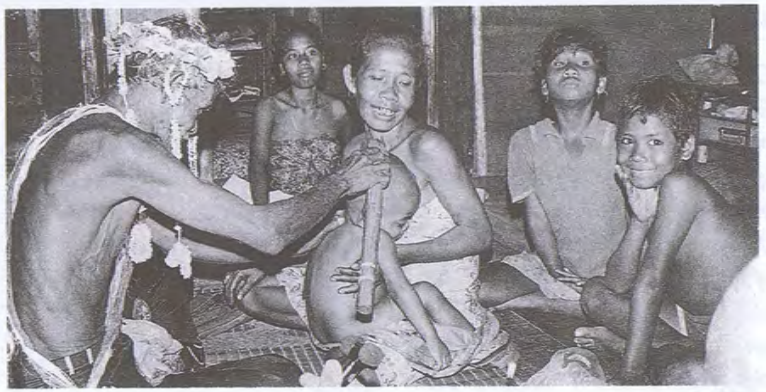
'Mosquito spirit' (Jah Hut) A. Baer

The importance of social support for those who are ill or impaired in some way is often overlooked. Such support may be crucial, as Polunin wrote (1977, p. 18):

"One afternoon I had to make a quick and exhausting walk to reach a settlement of the Jah Hut...Later that night I felt exhausted. My assistant, himself a traditional healer, offered to make a therapeutic incantation, which of course I accepted.

I was surprised by the great effect his ministration had in inducing a feeling of mental well-being...I had a strong intuition that this was because he showed me that somebody cared, in a situation where I felt isolated and helpless. I learned from this something I had scarcely been aware of: that an important part of the therapeutic situation is the social support it can provide."

Commentary:
The biomedical literature on Orang Asli contains little on the psychological aspects of therapy.



A young beneficary of a Jah Hut *menisoy* ritual, 1985. Duncan Holaday

Chapter 7

MALARIA: AN EXEMPLAR FOR BIOMEDICAL CONSIDERATIONS

MALARIA IN MALAYSIA: THE HISTORY AND EVEN MYSTERY

In the 1970's, 89% of Orang Asli showed immunological evidence of having had malaria at some time in the past, as I mentioned earlier. And in the 1990's, Orang Asli were the major group in West Malaysia with malaria infections. They accounted for 48% of all cases recorded in the early part of the decade, rising to over 70% later (Roslan Ismail, 1997) (note 34).

Malaria is a widespread human disease caused by a one-celled parasite (protozoan) that infects red blood cells and blood vessels. It is transmitted from person to person by anophiline mosquitos. The parasites multiply in the blood cells, later bursting out of them and causing fever episodes. While cerebral malaria can be fatal within days, ordinary malaria is typically debilitating for longer periods, accompanied by anemia and weakness. Anti-malarial drugs can both prevent malarial episodes and, if taken after an infection occurs, dampen their severity (note 35). Malaria is endemic in Malaysia.

Early reports on malaria in Orang Asli gave high, if often fuzzy, estimates of its damaging effects on virtually all groups (note 36). Williams-Hunt wrote (1952, p. 35):

"Both Noone and I have the impression that malaria is largely responsible for the infant mortality rate. The children who do survive have enlarged spleens and thereafter periodic fevers...Generally speaking, malaria appears to be a major cause of death with Aborigines."

When malarial surveys began in earnest, parasitemic prevalence rates began to be reported, that is, the percentage of people sampled that had parasites in their blood on the day of the survey.

In the 1930's prevalence rates for all-age samples were in the range of 20% to 38% (Polunin, 1953). After World War II, more precise data became available, with an overall rate for Orang Asli, excluding Johore, of 22% during the period 1950-1952 (Polunin, 1953).

No malaria survey data have been published on some Orang Asli groups over the last half century. Batek and Jah Hut are notable examples. But when we look at the data available on the other groups (Table 10), we find quite high parasite rates in most of them, in situations where people were not on anti-malarial prophylaxis, or at least were not reported to be (note 37). Among northerners the published rates are 25% to 37%; among Temiar, 5% to 49%; among Semai, 7% to 47%; and among Temuan 16% to 67%. The information in Table 10 indicates that malaria is common in small-scale societies such as the Lanoh, as well as in the larger villages of settled agriculturalists. It is common both at higher elevations (some Temiar) and lower elevations (Temuan). And for all groups so studied, rates have been higher in children than in adults (e.g., Andre et al., 1972; Baer et al., 1976; Khor, 1985). Lambros and coworkers (1989) mentioned a rate for Temiar children 0 to 9 years old at Legap, Perak, that "approaches 60%."

It seems fair to say that the majority of Orang Asli in the past have had "experience" with malaria, at least in childhood. Most of this experience has been with the most severe form, falciparum malaria (e.g., Thomas and Dissanaike, 1977; Baer et al., 1976). A strong minority of cases has been of the more chronic, vivax form of malaria. The third form found in Orang Asli, called malariae, makes only a mild and small contribution to their total malaria burden.

For all that, prevalence rates of malarial parasites in blood samples do not tell the whole story, or even most of it. First of all, many Orang Asli – especially adults – have malarial parasites in their bloodstream but do not regard themselves as being ill. Second, many Orang Asli children have low-grade mystery fevers that are not easily identifiable as malaria, barring a blood test. Third, and quite surprisingly, in studying Semai at Betau, Pahang, Khoo and coworkers (1996) found that some malarial infections are weak – so weak that the parasites are rarely found by the usual method of scanning a sample of blood cells on a glass slide microscopically. By using molecular techniques, they found that 12% of "noninfected" Semai actually

Table 10
Published malaria surveys on all-age Orang Asli groups *in situ*

Group/Locale	Parasite rate (%)	Study dates	References
Lanoh; Jehai/Upper Perak	25; 37	1950-2; 1963	P, 1953; K, 1966
Temiar/Chabai, Kelantan	34	1961	W, 1963
Temiar/Betis, Kelantan	13	1961	W, 1963
Temiar/Nenggiri, Kelantan	5	1961	W, 1963
Temiar/Perias, Kelantan	9	1961	W, 1963
Temiar/Up. Perak & Kelantan	29	1963	C, 1968
Temiar/Perak & Kelantan	35	1963	Bo, 1972a
Temiar/Upper Perak	20	1950-2	P, 1953
Temiar/Legap, Perak	49; 37	1986; 1989-91	L, 1988; G, 1991
Semai/Kuala Denek, Perak	20	1935-7	P, 1953
Semai/Btg. Padang D., Perak	12; 7; 17	1980-2	Kh, 1985
Semai/Ulu Jelai, Pahang	18	1950-2	P, 1953
Semai/Ulu Jelai, Pahang	25	1980's	Mk, 1988
Semai/Shean, Pahang	22	1970	Bo, 1972a
Semai/Betau, Pahang	47	1985	Mk, 1992
Semelai/Tasek Bera, Pahang	7	1961	W, 1963
Besisi/Selangor	2	1960	W, 1963
Temuan/Pangsoon, Selangor	48	1960	W, 1963
Temuan/Ulu Selangor	20	1972	B, 1976
Temuan/B. Manchong, Sel.	67	1960	W, 1963
Temuan/Pulas, Selangor	20	1961	W, 1963
Temuan/B. Mandol, Selangor	28	1960	W, 1963
Temuan/B. Tampoi, Selangor	31	1960	W, 1963
Temuan/B. Cheeding, Selangor	30	1963	Bo, 1972a
Temuan/Ulu Lui, Selangor	49; 13	1960; 1984	W, 1963; M, 1987
Temuan/Tekir, Labu, N.S.	43	1972	B, 1976
Temuan/Ayer Banning, N.S.	16	1972	B, 1976
Orang Selatar/Johore	0?	1950-2	P, 1953
Orang Asli, Johore	2	1990	A, 1992

Reports with subjects stated to be on anti-malarial drugs or having villages sprayed with DDT, or samples of N<50, are excluded. *Locale abbreviations*: B., bukit; Btg., Batang; D., district; N. S., Negri Sembilan; Up., upper. *Reference abbreviations*: A, Arasu; B, Baer et al.; Bo, Bolton; C, Collins et al.; G, Gordon et al.; Kinzie et al.; Kh, Khor; L, Lee et al.; M, Mak et al.; Mk, Mak; P, Polunin; W, Wharton et al.

harbored falciparum parasites. Thus many more parasitemics probably exist than have been routinely detected.

A last point is that if only, or mainly, adults are sampled in malarial surveys, the infection rate will appear to be very low, suggesting erroneously that malaria is not a serious health problem. The standard explanation for the known low-infection rate in adults has been that children build up immunity to malaria over time, given repeated exposure to malarial parasites in their environment, so that they become asymptomatic as adults. This is an oversimplification. Unfortunately, as Diggs (1992, p. 104) pointed out, "This remarkable immunity to disease (malaria) but not to infection (parasitemia) can persuade local health officials that malaria...is not so bad." But toddlers and school-age children are quite vulnerable to severe malarial disease. Without treatment, a fourth of this age group contracting the disease may die.

As Sandosham warned in 1970 (p. 223):

> "...the immunity the people have gained is at the expense of a high child morbidity and mortality. Those children must be saved from dying and from suffering from the harmful effects of malaria. They deserve to have the benefits of modern science so that they can develop to the fullest their potentialities to enjoy life..."

Furthermore, other health problems can make malaria more severe. "Modest" malnutrition in children or adults may do this by suppressing the function of the immune system. Weatherall and coworkers (1983) noted that in malarious areas those ill with the disease often have pathologies such as iron or folate deficiency, bacterial or parasitic infections, or other complicating factors that increase the severity of the illness (note 38).

True, infants may have a quite low risk for malaria. They receive anti-malarial antibodies from their mothers (via cord blood), which somewhat suppresses clinical disease until they are 6 months of age or so (Reuben, 1993). Also, breast milk is deficient in a vitamin (PABA) needed for the parasite's growth. And, true, antibodies to malarial parasites are at higher levels in Orang Asli adults than in young children (Thomas and Dissanaike, 1977; Mak et al., 1987). But two important factors, often overlooked, also enter into the malarial mosaic: genes and culture.

THE GENETICS OF MALARIA RESISTANCE IN THE ORANG ASLI

Genetic studies on Orang Asli have been piecemeal and often inconclusive as to their import. Yet overall we know more about genes that are easy to study and seem to be important for basic health than those that are elusive and obscure. For Southeast Asia, numerous researchers have looked for variant genes (alleles) that might have something to do with malaria, because particular alleles elsewhere were already connected with the disease. For example, Polunin and Sneath (1953) looked for, but did not find, hemoglobin S (Hb S) in Orang Asli because it was connected with malaria in Africa.

Human alleles found in Malaysia include some with no known relationship to malaria and others that provide some resistance to it (note 39). This latter group includes the dominant autosomal allele OV (ovalocytosis), the X-chromosome-linked G6PD- (glucose-6-phosphate dehydrogenase deficiency), and the E allele for beta-hemoglobin (Hb E). It also includes the much rarer Constant Spring allele for alpha-hemoglobin (Hb CoSp). These anti-malarial gene variants are in higher frequency among Orang Asli, on average, than among non-Asli in West Malaysia, indicating – among other things – that Orang Asli have been in this area for thousands of years. Over time, many generations of time, these gene variants have tracked malaria in Malaysia. The variant frequencies have increased under the relentless pressure of malaria on the original population. These frequencies are shown in Table 11.

While the mechanisms of anti-malarial gene action in blood cells are not fully understood, they involve both decreased red-cell invasion and decreased intracellular growth by the parasite (note 40).

From the biological point of view, it is important to understand the complexity of the relationship between genes and malaria. Malaria in Malaysia and elsewhere is not combatted genetically by any one trait, but by a hodgepodge of traits that have arisen by chance mutations in human DNA during the distant past, usually in different places. Studies on single traits versus malaria – and there are many of them – have missed this reality and have thus interpreted their results in a misleading fashion (Baer, 1998).

Based on what is currently known for Malaysia, nearly ideal genetic inheritance for resistance to malaria would be afforded by having one allele for OV (i.e., inheriting it from one parent and thus being a so-

Table 11
Frequencies of malaria-related alleles in Orang Asli

	G6PD⁻	OV	Hb E	Hb CoSp
Northerners	0.016	0.012	0.024	0
Temiar	0.182-0.218	0.034	0.319-0.323	0
Semai/Perak	0.219	0.033	0.254	0
Semai/Pahang	0.091	0.105	0.217	0
Temuan/US	0.075	0.214	0.011	0
Temuan/other	0.084	0.195	0.016	0.016
Temuan/TL	0.094	0.225	0	0.019
Temuan/AB	0.111	0.138	0.022	0.032
Jakun	0.095	0.048	0.018	0.013

Data from Livingstone, 1985, except Jakun data from Lie-Injo, 1976; AB, TL, and US refer to particular study locations. (Adapted from Table 2 in Baer, 1998.)

called heterozygote), a double dose of Hb E (one dose from each parent), and one or two doses of G6PD- (depending on one's sex and sex chromosomes). So far, no one has been reported to have this unusual genetic combination. If anyone does have it, it is likely to be an Orang Asli living in the area where the states of Selangor, Perak, and Pahang adjoin – an area where all three variants converge toward fairly high population frequencies. If such a person is not a Semai or a Temuan, he or she might be a Che Wong or a Jah Hut, because these latter two groups also live in the suspect area. Unfortunately, no genetical surveys on Che Wong or Jah Hut have ever appeared, although the latter are known to have a high level of G6PD- (see Table 6 and the postscript at the end of the text). Should either of these Aslian-speaking groups turn out to have high frequencies of both OV and Hb E alleles, the genetic connection between the Temuan (OV laden) and their Aslian-speaking neighbors (laden with Hb E) would be greatly strengthened, suggesting – but not proving – a major biological link in the past between the large categories of Malayic-speaking and Aslian-speaking Orang Asli groups.

Looking outward from the Orang Asli world, what might be the biological relationship between them and their more numerous Malay neighbors in West Malaysia? Benjamin (1987) has argued that since many Orang Asli transformed themselves into Orang Melayu (Malays)

in recent centuries, in this sense the Malay population is partly indigenous. Indeed, the fact that the OV, Hb E, and other genetic variants of the Orang Asli exist in Malay populations is congruent with this view. But the frequencies for OV and Hb E, for example, are much *lower* in Malays than in the Orang Asli (Livingstone, 1985), suggestive of a *small* outflow of such Orang Asli alleles into the Malay gene pool. While some of this outflow may have occurred by Orang Asli redefining themselves as Malays, the majority was likely the result of raiding by Malays for Orang Asli child slaves, who then came to speak Malay and accept Islam (note 41). Moreover, massive immigration of "Malays" from Sumatra and other nearby areas is known to have occurred in historical times (note 42). Benjamin hypothesized (1987) that old-time Orang Asli lost their anti-malarial alleles when they took up Malay culture and settled in the low-malaria lowlands, but this notion is problematical. It has long been known that the lowland Temuan have a significant malaria burden and also a strong complex of anti-malarial alleles (Baer et al., 1976). These alleles reach their highest known frequency, in fact, in a Temuan group living in Negri Sembilan that has been "exposed" to Malays geographically for generations, probably for centuries.

If, then, Malays in antiquity were geographically and genetically separate from Temuan and their Orang Asli compeers, we might revisit the question of whether the Aslian- and Malayic-speaking Orang Asli originated from a single prehistoric group. Fix (1995) reviewed this issue, including some genetical aspects. He considered that the various Orang Asli groups are an ancient interlocked population system, rather than static "types." He suggested that the genetic trait ovalocytosis (OV) entered Aslian-speaking groups such as the Semai rather recently and provided several pieces of evidence for this view. One, the frequency of the OV allele is low in Aslian speakers. Two, during the 19th century Rawa slave raiding from Sumatra caused one Selangor Temuan man to flee to Pahang, where he had numerous descendents – many of them having the OV trait today. Three, some Selangor Temuan male slaves of Malays ended up, after becoming free, in Sungkai, Perak, where they married Semai women and founded a village. While data on genetics, archeology, and malaria are too sparse to resolve the question of one or more original populations for the entire Orang Asli world, Fix made a plausible argument for an interacting population.

THE MALARIA MISCONCEPTION

An anthropologist recently asked a geneticist this question: If some genes protect the Orang Asli against malaria, why do they have a high rate of the disease? The answer is a complex one, some parts of it obvious and some not.

Of course, if the Orang Asli lived in Tahiti or Alaska they wouldn't have malaria, because no malaria exists in these places. Given a malarial environment, however, Orang Asli probably would not get malaria if they had a constant supply of anti-malarial drugs, always slept under bednets that had no holes, and lathered themselves with mosquito repellent.

But when a malaria-carrying mosquito bites an Orang Asli, she becomes infected, just as others do. It's what happens later that is critical. For older Orang Asli, with their immune defenses against malaria built up from repeated past exposure, many invading parasites will be "shot down" by antibodies in the bloodstream quickly. The remainder may survive to try to attach to, and invade, red blood cells. If the victim has a protective OV, Hb E, or G6PD- trait, only a few of these parasite survivors will be able to invade red cells and reproduce in them, which they must do to continue their life cycle. For instance (as I discussed in note 40), OV heterozygotes can have low levels of parasites in their red cells, but they almost never have high levels (or have cerebral malaria). When a malarial survey team records only the presence versus the absence of parasites on blood films, numerous adult OV heterozygotes appear to "have" malaria, even though they are not ill. The moral of this story is that parasitemia data may tell us little about the disease burden in a community, especially if the data are not analyzed by age group and by the parasite density on blood films.

When children's malarial rates have been studied separately, a better correlation is discovered between parasitemia and illness, especially at high levels of parasites. The reason is simple. Even if children have OV or another protective gene trait, they have not yet reached a high level of immunity (antibodies). Nevertheless, such gene-protected children are less prone to cerebral malaria. So the biological answer to the question posed initially is that Orang Asli communities have malaria because it pervades their environment, but they have lower rates of malarial morbidity and mortality than city people would have in the same circumstances because of their antibodies and their genes.

PUTTING IT ALL TOGETHER: MALARIA, GENES, AND CULTURE

People die from malarial attacks because they are exposed to the parasite environmentally, have little or no immunity to the parasite, do not receive prompt and proper medical attention, were not lucky enough to inherit a malaria-resistance genetic trait, and (often) have other debilitating conditions.

People survive malarial attacks because they have immunity to the parasite (whether maternal antibodies in infants or immunity acquired through later infections), are generally in good health, receive prompt and proper medical attention, and – in the Orang Asli context – happen to inherit a malarial-resistance genetic trait.

Other factors come into play when we consider an Orang Asli community as a whole that is under continual threat by malaria. Outside of the factors that come easily to mind, such as mosquito nets and care-giving by kin, it is worth mentioning the genetic exclusiveness of the ethnic group (note 43), its methods of house construction, whether it keeps livestock near houses, the length of time spent away from the village (especially as night guard over ripening field crops or tree fruit), the level of genetic-resistance traits in the ethnic group, and various behavioral responses to illness.

For example, many traditional Orang Asli houses are built on tall stilts and are open enough so that the air inside is not still. Temiar

Colin Nicholas

Jehai youth suffering from malaria, Jeli, Kelantan.
The majority of Orang Asli have had experience with malaria. Death can result if they have little or no immunity to the parasite, and if prompt and proper medical attention is not forthcoming.

houses may be 20 feet off the ground (Williams-Hunt, 1952). Semai houses, according to Polunin (1953), are 3 to 6 feet off the ground. However, I have noticed that some are much higher. Smokey hearth fires in stilt houses are kept going after dark, and "warm bodies" of livestock are kept under them – such as a pet wild pig. All these factors may contribute to a cultural form of defense against malaria. At least some mosquito vectors of malaria are known to be low fliers, having a flight ceiling of only 10 feet or so (see May, 1954; Knapen, 1996; Baer, 1998). Indeed, anopheline mosquitos as a rule may not fly more than 60 cm. (2 feet) above the ground (Clements, 1963). When they fly under stilt houses, they often encounter attractive animal bodies, warm and smoke-free, upon which to obtain a blood meal (note 44). Such, at least, is one view; but it seems to have been virtually ignored by mosquitologists in Malaysia. While the findings of Wharton and coworkers (1963) on the mosquitos of Temiar villages seem to fit this view, a study in the Philippines showed that having a water buffalo near a sleeping area increased the level of human biting by the Anopheles mosquito there (Schultz, 1989). However, Vythilingam and coworkers (1992) have shown that the Malayan forest vector of malaria prefers to bite village animals rather than people. More research is needed to resolve this question.

The traditional Orang Asli lifestyle, however, involves nights spent away from the homestead, whether on hunting trips or guarding an ancestral durian tree with ripening fruit from poachers. Under these commonplace circumstances, the chance of a malaria infection may be relatively high. In addition, if a new fever is treated casually – as may be the case – it can develop into full-blown malaria before medical treatment is sought: another negative for a healthy prognosis.

Some traditional herbal remedies may be efficacious against malaria. The bark, leaves, and roots of the small tree known as Tongkat Ali were traditionally used by Semelai for various ailments and by Temuan for malarial and other fevers. Kardono and coworkers (1991) have now shown that constituents of the roots of this tree have strong anti-malarial activity, at least in vitro.

Overall, the Orang Asli survive malaria not well and not in every case, but they do survive under natural conditions of environmental stress. They have done so for millenia. Whether they will survive under the stress of current social-political forces is an open question.

Chapter 8

FOOD, PARASITES, AND HEALTH: THE NEXUS OF BIOLOGICAL SURVIVAL THROUGHOUT HISTORY

NUTRITION

The nutritional status of Orang Asli has been reviewed by Kasim and coworkers (1987), Khor (1994), Chee (1995), and Osman and Zaleha (1995). Most of the data pertain to children.

An overview of energy nutrition is given in Table 12. In general, these studies show that the Orang Asli consume far fewer calories than the recommended daily allowance or intake (RDA), adjusted for age and sex. Other evidence comes from Massita (1992), who estimated that Betau Semai children averaged only 77% of the RDA (cited in Chee, 1995). And Lim (1997) estimated that 34 reproductive-age Jakun women in two Pahang villages averaged less than 60% of the caloric RDA. But the worst off seem to be the Semai children in Perak, who averaged 770 kcal, 42% of the RDA (Khor, 1988).

These food deficits have numerous causes, some of which are subtle. For instance, many Orang Asli economies were traditionally labor-limited, rather than land-limited. In this situation, the weakness associated with malnutrition restricts the amount of food that can be foraged or grown over a season or year, leading to long-term difficulties. More recently, Orang Asli economies have also become land-limited. This introduces another set of health problems and, in some cases, has worsened the nutritional outlook.

Adult Nutrition

It is useful to consider details of adult nutrition before those of childhood nutrition, if only because an infant's nutrition starts in utero.

As mentioned in Chapter 1, Orang Asli women are much more malnourished than other rural women and are also more malnourished

than Orang Asli men, according to the study by Osman and Zaleha (1995). For Lanai and Betau Semai, overall, the figures for protein-energy malnutrition (PEM) are 35.3% for women and 11.6% for men versus 7.5% for rural Malay women and 6.3% for Malay men. Using different methodology, Lim (1997) found that reproductive-age Jakun women averaged 86% of the official protein RDA (and 60% of the caloric RDA), although the diet of all the women was deficient in iron. Deficiencies in vitamins and calcium were also prevalent.

The high level of PEM in Orang Asli adults results from a mix of lost foraging and farm land, the increase of river pollution (decreasing fish catches), dietary inadequacies, the level and frequency of infectious diseases and intestinal infestations, and cultural discrimination. Hard physical labor also contributes to PEM: Osman and coworkers (1991) compared Temuans and Malays, age 7 years and older, living in Kuala Pangsoon, Selangor; while 30% of the Temuans engaged in hard physical labor on a daily basis, only 9% of the Malays did so. For Orang Asli women, additional predispositions to malnutrition include the number and spacing of pregnancies and the extent of breastfeeding. In the 1970's, Orang Asli women ages 16 to 60 years averaged 5 pregnancies, with 7% having 9 or more pregnancies (Sumithran, 1977).

On a longer time scale, according to Osman and Zaleha (1995, p. 322), "Women need nutritious food from birth to maturity, adequate medical care, sufficient rest, more educational opportunities, and less sexual discrimination." In support of this statement, the authors reported that over 95% of Semai women breastfed their infants, for an average of 17.9 months – a considerable boon to their offspring but a metabolic stress to themselves. Khor (1985) found an even longer breastfeeding duration for 189 Semai women, 23 months. Moreover, 64% of the Semai women studied by Osman and Zaleha, as compared to 35% of the men, were goiterous, another metabolic problem. While these authors did not study all possible contributions to maternal ill health and malnutrition (for example, iron and folate deficiency, worm burdens, and malaria), their conclusion that Orang Asli women are unhealthy is a strong one that cannot be ignored. If female reproductive health is an indicator of the quality of a health-care system, as Walsh and coauthors (1993) maintain, then the governmental medical system for Orang Asli is correspondingly dysfunctional.

Table 12

Estimates of energy foods for Orang Asli versus recommended daily allowances (kcal of food/person/day)

Group	Intake	Recommended	Reference
Semai, all ages, resettled, Pahang	1300 provided	1170-2240	Polunin, 1953
Semai, 4-6 yrs., Perak	770	1580-1800	Khor, 1988
Semai, 4-6 yrs., Pahang	1233	1580-1800	Ismail et al., 1988
Semai, >18 yrs., Lanai, Pahang	1430	1550-2220 (for 16-59 yrs.)	Osman et al., 1993a
Semai, >18 yrs., Betau, Pahang	1143	1550-2220 (for 16-59 yrs.)	Osman et al., 1993a
Temuan, >18 yrs., Bukit Lanjan, Selangor	1462	1550-2220 (for 16-59 yrs.)	Osman et al., 1993a
Temuan, > 6 yrs., Kuala Pangsoon, Selangor	1400	1890-2240	Osman et al., 1992
Semaq Beri men, Pahang	2343	2180	Kuchikura, 1988

As published by Kuchikura, 1988, or Ismail et al., 1988. In Polunin (1953), the 1300 kcal provided by the government is a minimum estimate. In all these studies, other components of the diet were also found to be inadequate, such as fats, iron, calcium, protein, thiamine, vitamin A, and niacin.

With the Malaysian ethos increasingly demanding that Orang Asli become dependent on a cash economy, another nutritional problem particular to women (and children) may be growing. As men engage in wage labor, commercial rattan collecting, and other arduous work lasting long hours on a sustained basis, their need for energy foods increases. This may lead to a greater family allocation of food to men, at the expense of women and children. Such a maldistribution of food "would cause or amplify nutritional degradation among the most vulnerable groups in the community, i.e., women of childbearing age and young children" (Kuchikura, 1988, p. 26).

More problems ensue when sedentization villages are newly opened up, that is, when the government displaces a community to a designated (often deforested) area. Home-grown crops are usually not available for months, and if the village is "slated" primarily for commercial cultivation (for example, of rubber) home-grown crops may be deemphasized in relation to store-bought food. For such new villages, civil servants often promise to supply food rations until the community is self-sustaining economically. Sometimes, however, these rations do not arrive on a regular schedule, causing shortages. And often the rations are not enough to satisfy the people's nutritional needs (Kuchikura, 1988). Moreover, if a man is away from his village when government officers arrive to distribute subsidies, he is not paid (Dentan et al., 1997). In my view, this practice could be substantially improved by dispensing subsidites to women, given their key role in family nutrition and the higher chance of them being "at home."

Another metabolic stress can arise in government-run villages (or "schemes") where Orang Asli are directed to commercial activities and disallowed subsistence hunting and foraging. Kuchikura (1988) related how, because of the agricultural cycle, a hunger season occurred for a Semaq Beri village in Trengganu (crops were not yet ripe). During this time the local government representative insisted that the Semaq Beri remain in the village, rather than move into the forest to forage for wild foods as they wished to do. As a result the villagers' energy intake fell 20% in short order.

Orang Asli groups have always foraged for a myriad of food resources in their home environments, even when they were "full-time" farmers (Dentan, 1991). The total Orang Asli diet thus provided high-quality food with interesting variety, if seldom in large quantities. As these groups have been forced to move – by external circumstances – away from foraging toward storable, staple crops and store-bought foods, the quality and variety of food has been sacrified to unbalanced quantity, or sometimes unbalanced scarcity.

Gomes (1989) found that Tapah, Perak, Semai buy at least 88% of their food from the market. Kuchikura (1988) pointed out that for the Semaq Beri of Trengganu cash resources led to purchases of carbohydrates (rice) and fats (cooking oil) but not to better overall nutrition, in part because of "a craving for more costly nonfood goods, such as cassette radios and tapes, more substantial clothing, umbrellas...wristwatches, and so on" (p. 24).

Khoo (1977) also commented on the reliance on store-bought foods, such as sweetened condensed milk, sardines, sugar, cooking oil, and flour. Osman and coworkers (1991) found that Pangsoon Temuan in Selangor, living in a government-invented village, had an average food intake of 1400 kcal/day (possibly referring to adults only, although this was not stated). In contrast, Chan and coworkers (1974) found that 25 Temiar boys in Kelantan who received boarding-school meals ate at least 2250 kcal/day; a smaller sample of Temiar men ate at least 2500 kcal/day. But school meals do not always prevent undernutrition; Ismail and coworkers (1988) found that 23% of Betau Semai recipients of school meals, age 7 to 10 years, were underweight.

While the traditionally eclectic diet of swiddeners was probably low in protein and animal fats, hunter-gatherers such as the Batek may have had ample meat supplies (note 45). In particular, commonly eaten starchy foods such as bananas, yams, rice, sweet potatoes, and cassava tubers are poor in protein. Animal fat has long been considered a special treat by Orang Asli; other than wild pig, most animals hunted for food in the rainforest provide only lean meat (Kuchikura, 1988).

Child Health

Like adult health, childhood health has many dimensions. To introduce this subject, health statistics for West Malaysia as a whole for 1982 show that the crude (live) birth rate was 3.06% in a population of 12,039,000, with the stillbirth rate being 1.35%, the infant mortality rate being 1.9%, and the toddler mortality rate, for 1 to 4 years of age, being 0.17% (Santos et al., 1985). Childhood immunizations were quite comprehensive, with 95% of infants receiving BCG against tuberculosis, for example.

Few publications exist on Orang Asli childhood health statistics.

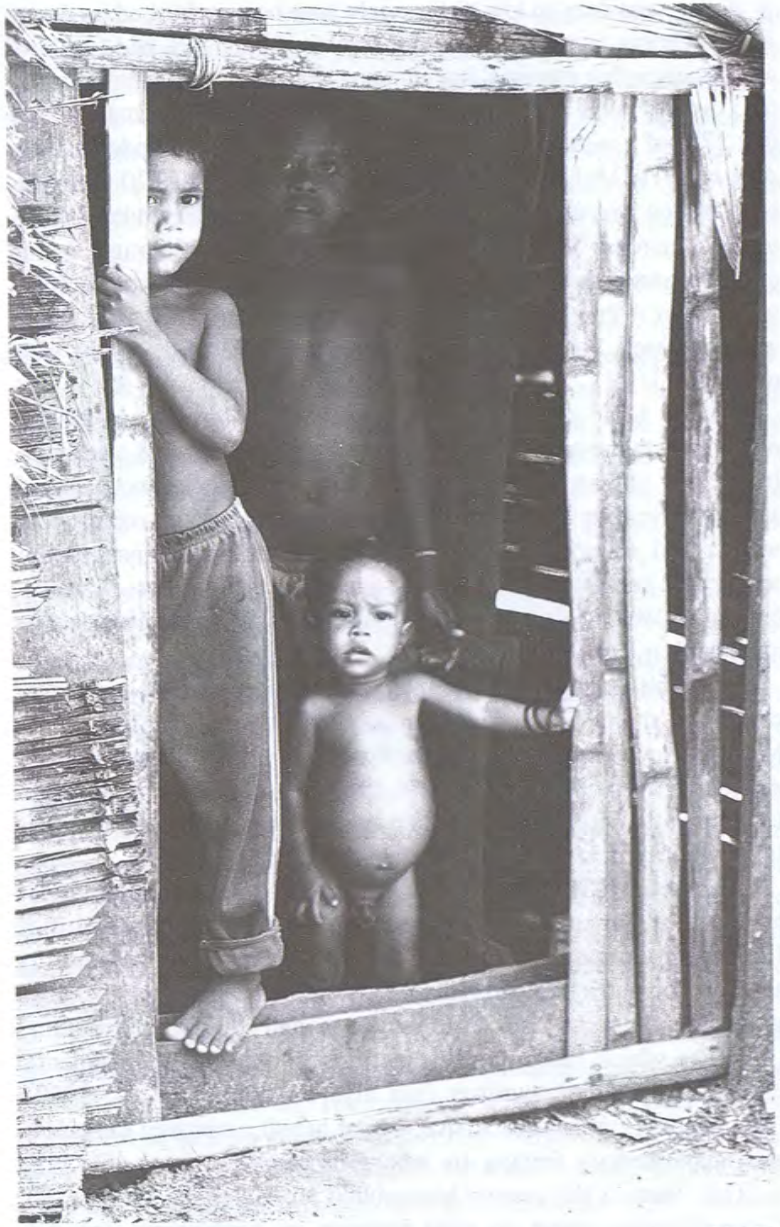
And what has been reported is not promising. Noone (1936) reported a child mortality rate (ages unspecified) of 36% for Temiar. Kinzie and coworkers (1966) reported a child mortality rate of 26% for Bukit Cheeding Temuan and of 54% for Fort Banding Lanoh. Polunin (1953) estimated a child mortality rate of 29% for Orang Seletar in Johore and a 60% rate for lowland Semai. For the 211 Semai mothers that Khor (1985) interviewed in Perak, on average 1 of their children had died before reaching the age of 5 years. Fix (1971) calculated a 20% to 30% infant mortality rate (IMR) for the Semai of Satak, Pahang. Officially, the IMR for all Orang Asli for the years 1984 to 1987 was 5.2%, versus 1.6% for the national average (Ng et al., 1987). In 1987 the IMR for the capitol city, Kuala Lumpur, was only 1% (Wong, 1991b).

For Satak Semai, life expectancy at birth in 1969 was calculated to be 31 years for males and 28 years for females. In comparison, the West Malaysian average for 1970 was 63 years for males and 68 years for females (Chee, 1995), more than twice as long as for the Semai. Matters had improved somewhat by the 1980's, according to comparative data provided by Ng and coworkers (1987), as shown previously in Table 4.

Except for Khor (1985) and Ong (1975a, b), reports have been largely silent about Orang Asli stillbirths. Khor reported that stillbirths were one of the four major causes of Semai deaths within the 0 to 5 years age group. The others were diarrhea, high fever, and severe cough. Ong reported 3.4% stillbirths for a selected hospital sample (N=116) but 2.6% stillbirths and 1.1% miscarriages for a larger sample (N=271).

Malnutrition probably contributes to childhood mortality more than is indicated by government statistics. That is, "downsizing" has both an individual and group component. When the precipitating cause of death is an infection, malnutrition is often the underlying and even ignored cause (Chen, 1986).

Arokiasamy (1990) reviewed childhood nutrition in Malaysia without mentioning Orang Asli. According to this author, governmental programs have given priority to improving child health since the country's independence in 1957. While 10.4% of recorded newborns in 1981 weighed less than 2.5 kg. (the criterion for low birth weight), only 8.8% had low birth weight in 1986. For Malaysia, the average birth weight in the 1980's was about 3 kg (Walsh et al., 1992).



Colin Nicholas

Jehai boy with potbelly, Jeli, Kelantan.
The condition is generally a result of malnutrition or severe worm infestation, or a combination of both. Also, the crude death rate of the Jehai is the highest for any Orang Asli group.

No general data on low birth weight have been published on Orang Asli newborns, but it would be remarkable if only 10% had low birth weight, given that maternal malnutrition and anemia are quite common (as I discuss in the next section of the chapter). In 1975, Ong reported that 27% of a select sample of 114 newborns weighed under 2.5 kg. And in 1971, McLeod reported the average weight of 320 Gombak-born babies was only 2.7 kg (5.9 lbs). Birth weights under 2.5 kg. tend to increase infant mortality and morbidity. Although Bolton (1968) reported that only 5% of Orang Asli births at Gombak Hospital were under 4 lbs. (1.8 kg.), the percentage under 5.5 lbs. (2.5 kg.) was not stated. He did, however, report that 30% of the births were premature.

On the topic of nutrition in preschool children, Arokiasamy recognized that malnutrition still exists, in rural areas and elsewhere. In addition to protein-calorie malnutrition, deficiencies in vitamin A and iron have been found, as well as the chronic parasitemia of intestinal worms and malaria. The author held that childhood malnutrition remained a serious problem in the state of Sarawak in East Malaysia, but not in West Malaysia, where both the severity and prevalence of malnutrition had declined.

In commending the governmental actions that have decreased childhood malnutrition in Malaysia, Arokiasamy focused on the Maternal and Child Health service (MCH) of the Ministry of Health. A health survey conducted in 1978-1980 led to "mobile health teams to improve the accessibility and coverage of basic health services in line with the primary health care approach" (p.69).

Also, the MCH shifted from a clinic to a more community based approach by, for example, implementing the "Strategy for Nutrition Education-Community Approach" and conducting community sessions for nutritional surveillance. Moreover, since the 1970's the government has had an "Applied Food and Nutrition Program" (AFNP), which integrates the actions of various governmental ministries with communities (see also Rajakumar et al., 1980). Breastfeeding, nutritional advice, dental health, improved sanitation, and supplementary feeding are among its emphases (note 46).

This, then, is the general background for Malaysian child health. Given this rosy report on child nutrition by Arokiasamy, one may well ask how the nutrition of Orang Asli children stacks up.

In brief, the nutritional status of Orang Asli children is perilous.

This is not an extreme view. Studying Orang Asli children from all over West Malaysia, Kasim and coworkers (1987) found 54% were underweight and 66% were stunted. In contrast, for Malaysia as a whole 21% of children under the age of 7 years were significantly malnourished in 1986 (Tee and Cavalli-Sforza, 1993). Osman and coworkers (1993c) found 73% of Temuan children 2 to 6 years old to be stunted, and Osman and Zaleha (1995) found 80% of Semai of the same age to be malnourished.

For Semai at Lanai and Sinderut in the age range of 4 to 15 years, at least 78% were malnourished (Wan Nazaimoon et al., 1996) while 31% of Orang Asli children resident in Gombak were malnourished. Khor (1985, 1988, 1994) described the nutritional status of Orang Asli children as poor, both in an acute and a chronic sense. And Chee (1995) compared measurements on Orang Asli children from 1979 through 1993 in different areas; those classified as underweight ranged from 18% to 65% and those classified as stunted ranged from 15% to 73%.

Details of childhood nutrition are available mainly on Semai. By measuring weight for a given age, the frequency of underweight children can be estimated. Using this method, three studies obtained important results. For Semai of Betau, Pahang, Ismail and coworkers (1988) found 30% of 4 to 10 year olds were underweight, while more recently Osman and Zaleha (1995) found 62% of 2 to 6 year olds at Betau were underweight. But for Semai of Lanai, Pahang, Osman and Zaleha's findings were less dire: 28% of 2 to 6 year olds were underweight. For Semai of the Batang Padang district of Perak, Khor (1985, 1988) found 58% of those 0 to 18 years of age were underweight, with the highest percentages being in the 6 to 12 year age group. In my view, these three studies indicate widespread malnutrition among inland Orang Asli children.

Further evidence of generic nutritional problems comes from measurements of height for age, a way to estimate stunted growth due to long-term malnutrition. The frequency of stunting appears to range from a low of 44% in infants (Khor, 1988) to 80% in 2 to 6 year olds (Osman and Zaleha, 1995). Support for these findings comes from Khor's determination of skin-fold measurements (13% to 48% malnourished by this criterion), upper arm measurements (24% to 68% malnourished), and protein deficiency (36% to 41% malnourished).

As shown previously in Table 12, childhood malnutrition as measured by food intake is rampant. For a small sample of Semai children age 2 to 6 years (N=30), Khor (1988) found the average caloric intake was only 42% of the recomemded daily value, the average protein intake only 55% of the recommended value, the average calcium 41%, the average iron 29%, the average vitamin A 68%, and the average thiamine 63%. Unpublished data, quoted by Khor (1994) and Chee (1995), show similar results.

Khoo (1977) found that for Temiar "grouped" a year earlier at Fort Kemar, Perak, 11% to 22% of preschool children were underweight and 29% were stunted. Diets were deficient in calories, protein, iron, and most vitamins.

Kasim and coworkers (1987) studied 566 northern, central, and southern Orang Asli children, 0 to 10 years of age, as a group. For all ages studied, 54% were underweight (note 47). While for those 0 to 6 months of age only 12% were underweight, all older age groups showed much worse malnutrition, averaging 59%. Among toddlers aged 1 to 2 years, 71% were underweight, the highest level for any age group. By ages 8 to 10 the proportion of underweight children had dropped to 39%. Boys were more often underweight than girls (61% versus 48%), with only 30% of girls being underweight by age 8 to 10 years. With respect to stunting, infants 0 to 6 months of age showed the least: 25%. Overall, however, 66% of all ages studied were stunted, with the age range of 6 to 8 years having the highest proportion: 76%. In comparing ethnic group foci by state, the authors found that groups (unspecified) in Pahang had the worst childhood nutrition, followed by those in Perak, Johore, and Kelantan.

Relevant to this dismal state of affairs is an interesting belief held by some Temuan. They say that Temuan were once tall but have become progressively shorter. When they eventually are about one foot tall, the whole world will end and a different one will begin. In it, the dead will live and the living will die; animals will become people and people will become animals. I think it quite possible that the Temuan are making an important point with this scenario.

Associated with research findings of malnutrition have been significant frequencies of intestinal worm infestations. Such worm burdens aggravate malnutrition. Osman and Zaleha (1995) found that 21% of their sample of Pahang Semai children had Ascaris, 24% had Trichuris, and 5% had hookworm. They also found that 18% of their sample had goiter. Khor (1988) reported that 57% of Perak Semai children age 0 to 11 years had Ascaris, 49% had Trichuris, and 14% had hookworm; in addition, 42% were anemic, as measured by age-adjusted hemoglobin levels, and a small percentage was thiamine deficient.

Because of serious childhood malnutrition – in addition to intestinal worms and other problems, Khor (1988) pointed out that Orang Asli children may not be physically very energetic, in order to limit their energy expenditure. If true, this lack of activity would have a detrimental effect not only on the childrens' physical development, but also on their intellectual and social development (Brown and Pollitt, 1996).

MATERNAL HEALTH

The crucial topic of maternal health has received far too little attention by biomedical authors. This neglect is particularly striking in terms of the Orang Asli.

Anemia and Antenatal Care

The dissertation by Khor (1985) provides disturbing evidence of maternal ill health among the Semai of the Batang Padang district of Perak. Khor found, first, that 40% of all women (19 years or older) were anemic and, second, that 64% of pregnant women were anemic.

Likewise, Khoo (1977) earlier had found that among the Temiar newly moved to Fort Kemar in Perak, a third of the non-pregnant women and over half of the pregnant ones were anemic. More recently, Jennings (1995) described in-house childbirth for Temiar women attended by traditional midwives.

Only a few other reports dealing directly with maternal health appear to exist on Orang Asli (Ong, 1973a, b; 1974a, b; 1975a, b). All were on pregnant women at Gombak Hospital – not a random sample by any means. Among 278 "normal" pregnant women studied (Ong, 1973a), 71 or 25.5% were anemic – in contrast to the 64% found by Khor (1985). Almost all of the 71 anemic women in Ong's study were from peri-urban or forest-fringe areas (including many government-mandated "resettlement" villages). According to Ong, these results point to some iron-deficiency anemia in "the average pregnant Orang Asli woman." The practice of giving supplemental oral iron to pregnant women, presumably at Gombak Hospital, was

mentioned in this report (note 48). Ong reported in 1974 (a, b) that among the 71 anemic women, 38% appeared to have folate-deficiency anemia and 6% iron-deficiency anemia, that all 71 were malnourished, that 69% had intestinal worms, and that 13% had malaria. Among the anemics, 55% were in their fifth or higher pregnancy.

Studies on anemia are of particular importance because anemia lowers a woman's ability to resist infection or to survive possible hemorrhaging during pregnancy or childbirth; anemia may increase her chances of dying during labor by a factor of four (Walsh et al., 1993). It can also lead to newborn death. And it lowers work capacity. Anemia, then, can be the proximal cause of tragedy for an entire family or hamlet.

Ong (1974a) also reported on anemia in relation to the hemoglobin E characteristics found in 28 of 124 pregnant women studied at Gombak Hospital (but G6PD deficiency, ovalocytosis, and malaria were ignored in this report). It is striking that 46 of the 96 women who had no Hb E (48%) were anemic by the same criterion as used previously (Ong, 1973a), at which time "only" 25.5% were anemic. The difference between these two findings, not mentioned in this report, cannot be ignored. The sample used in this 1974 study may have been biased by selecting women who appeared to be thin and wan, in the hope thereby of identifying a high proportion having hemoglobin variants. While this nearly two-fold difference in anemia prevalence may be an artifact, it might also be real, reflecting a general deterioration of nutrition, a widespread viral epidemic, or some other important change in the Orang Asli environment. In any case, 55% of the 20 women who were Hb E heterozygotes in this sample (having inherited one dose of Hb E from one parent and one dose of the usual Hb A from the other) had anemia, much like the 48% of their non-E counterparts. Women with other genetic conditions involving Hb E were too few for drawing any conclusions, although the author tried. Also the "thalassemia" mentioned in the report may actually have been Hb Constant Spring, a trait perhaps overlooked by the author. Still, the studies of Khoo, Khor, and Ong establish anemia as a significant force for ill health in Orang Asli women.

Recent work also points to widespread anemia in Orang Asli women. Osman and coworkers (1992) found that Kuala Pangsoon Temuan women had an average hemoglobin concentration of 9.9 g/dl, far below the "acceptable" level.

Salutary procedures were in place for antenatal Orang Asli women in hospital in the 1970's (McLeod, 1971). Blood samples from the mothers were tested for blood type, hematocrit, white cell count, and malaria. An X-ray was taken to check for tuberculosis. Vitamin and iron tablets were given daily. A medical doctor gave a physical exam not only to the mother but all of her accompanying family. Any problem found (such as malaria, TB, anemia, or intestinal worms) was promptly treated. The most common maternal condition was anemia (< 10 g/dl, or a hematocrit of < 30%). Worms, chronic malaria, and amebic dysentery were often implicated in the anemia. After giving birth, a month's supply of iron and vitamin tablets plus a pediatric vitamin syrup was given to each mother upon hospital discharge. All told, these procedures were laudable. However, there is reason to doubt that procedures for Orang Asli women and their families today (in district and other hospitals) are uniformly as comprehensive.

Sexually Transmitted Diseases

The only report focusing exclusively on sexually-transmitted, or venereal, disease in Orang Asli women was by Ong (1973b) on hospitalized pregnant women. The dearth of STD reports is compounded in this case by the remarkable fact that the major STD's were *not* studied; that is, no mention was made of syphilis or gonorrhea (although they were considered by Roslan Ismail, 1997, in a brief report on both men and women). Only vaginal yeast infections (candidiasis) and trichomoniasis were studied by Ong, which are far from life-threatening. Among 94 women tested, 67% had candidiasis and 13% had trichomoniasis, with the vast majority of positive cases being asymptomatic (by questionnaire) (note 49). No causative factor was elucidated for these relatively high levels of vaginal infections.

A rare paper discussed venereal disease historically (Parmer, 1989). While the author made no mention of Orang Asli, he reported that in the 1920's half of the English males and 89% of non-English males in Malaya had some form of VD, principally syphilis. Gonorrhea was in second place. Immigrants then were mainly men and prostitution was rampant.

According to Lim (1993), reliable records on syphilis are not available for Malaysia. Serological testing indicated that 2% of expectant mothers and almost 5% of blood donors in Malaysia had syphilis antibodies in the mid-1970's. Gonorrhea is more prevalent

than syphilis in Malaysia but is widely unreported in this antibiotic era. However, in one month 123 patients were treated for gonorrhea in Ipoh alone, according to a fairly thorough survey in 1958.

It seems to be generally assumed in Malaysia that STD's are rare in Orang Asli. Perhaps this is linked to the erroneous assumption that Orang Asli are remoted and "untouched." Yet it would be quite easy to study important STD's on pregnant women at Gombak Hospital. Such a study of Orang Asli females is urgently needed, including, of course, a study of HIV (the AIDS virus) (but see Roslan Ismail, 1997, for a few hospital data).

Maternal Iron Deficiency and Malaria

Outside of the reports by Khor and Ong, a few others focus on various aspects of maternal health – mainly in terms of nutrition, anemia, malaria, and iodine deficiency. These four aspects can be central to both maternal and infant survival and, in a broader sense, the vitality of Orang Asli communities. As Reuben remarked (1993, p. 478), "[Women] are the people most affected by family illness." A closer look at these four categories of concern will help make this centrality clear.

Dietary iron deficiency – often associated with malnutrition – can lead to anemia. Intestinal worms, malaria, and other blood-destroying infections are common secondary causes of anemia. Food and parasites interact most directly in the case of intestinal worm infestations. The worms increase nutritional needs and interfere with the gut's ability to absorb food. Hookworms cause loss of blood and destroy gut tissue, both of which must be replaced. With intestinal infestations, overall protein levels decline and iron-deficiency anemia often ensues (note 50). Because of menstruation and the metabolic requirements of pregnancy and lactation, women have a general tendency toward iron-deficiency anemia. With all this in mind, iron intake in Orang Asli is an important question.

Osman and coworkers (1992) compared Temuans and Malays in Pangsoon, Selangor, over the age of 6 years, as to iron intake. The Temuans averaged 7.4 grams versus 11.3 for the Malays, a significant difference. Lim (1997) estimated that the average iron intake of reproductive-age Jakun women in Pahang was in the range of 25% to 33% of the required level. By studying transferrin saturation, Khor (1985) found that 25% of boys and 28% of girls in the age range of

Table 13
Goiter in Orang Asli

	N	% of total sample with goiter	% of women with goiter	Reference
Inland groups				
Kensiu, Kedah	63	30	—	Osman et al., 1995a
Lanoh, Upper Perak	163	21	54	Polunin, 1951a
Temiar, Perak	47	32	57	Polunin, 1951a
Semai, Pahang & Perak	361	52	84	Polunin, 1951a
Semai, Pahang	616	40	64	Osman & Zaleha, 1995
Temuan, Selangor & Perak	108	31	77	Polunin, 1951a
Temuan, Bukit Lanjan, Selangor	203	11	—	Osman et al., 1993c
Temuan, Kuala Pangsoon, Selangor	50	27	—	Osman et al., 1992
Coastal groups				
Besisi, Selangor	67	6	—	Osman et al., 1995a
Orang Selatar, Johore	68	1	0	Polunin, 1951a

From data in Polunin, 1951a, where women are defined as females >15 years of age; in Osman and Zaleha, 1995, where I have defined women as females >17 years of age; and in Osman et al., 1992, 1993c, and 1995a.

13 to 18 years were iron deficient. Other Semai age groups were not studied in this regard, but these levels of anemia can be partially blamed on the fact that 14% of younger children were found to harbor hookworms. And in Kuala Koyan, Perak, Greer and coworkers (1989) found 84% of Semai to have hookworm infections. In related matters, Khoo (1977) reported that a significant proportion of Temiar women at Fort Kemar, Perak, was anemic due to iron deficiency.

Malaria presents particular risks to pregnant women. Yet clinical signs of malaria during pregnancy are rare in areas of high malaria transmission, such as those in which the Orang Asli live (Menendez, 1995). This rarity reflects both immunity and genetic resistance to the disease. Malarial symptoms in such areas are most likely to occur in first pregnancies; the combination tends to produce low birth weight babies (Menendez, 1995). And low birth weight is the greatest risk factor for neonatal death (Schultz et al., 1995). Because of these concerns, chemoprophylaxis for malaria in first-pregnancy mothers is widely advised (Reuben, 1993).

Although it seems counterintuitive, malaria in pregnancy rarely produces an infection in the newborn. The explanation may involve the passive transfer of maternal immunity to the offspring through the placenta, the presence of fetal hemoglobin (Hb F) which antagonizes the growth of malarial parasites, or the fact that a milk diet starves the parasites of the required nutrient PABA (part of the vitamin-B complex). Anti-malarial genetic factors may also be involved.

Maternal and Childhood Goiter

Protein-energy malnutrition and endemic goiter often coexist, especially in the metabolically demanding states of pregnancy and childhood. Iodine deficiency leads to the formation of goiter, in which the thyroid gland enlarges in a vain attempt to "capture" a sufficient amount of iodine for the body's metabolism. During pregnancy goiter limits iodine for the growing fetus and leads to neurological damage, in severe cases. The result can be a miscarriage or stillbirth, or a living child with deafness, mutism, or mental retardation (Osman et al., 1995). And goiterous reproductive-age women have a higher than average death rate. So the scattered reports of 40% of "post" Semai in Pahang being goiterous (Osman and Zaleha, 1995), 6% of Besisi and 30% of Kedah northerners being goiterous (Osman et al., 1995),

11% of Bukit Lanjan Temuan being goiterous (Osman et al., 1993c), plus the earlier report of Polunin (1951a) that 41% of inland Orang Asli were goiterous, are ominous signs in terms of possible sequalae. In Polunin's work, Orang Asli women had an overall goiter rate of 73%, compared to 27% for men, 32% for girls, and 24% for boys (Table 13). In Osman and Zaleha's work, the women had a rate of 64%, compared to 35% for men, 33% for girls, and 25% for boys. Based on these data, a reasonable person might well conclude that between 1951 and 1995 improvement has not been remarkable.

Many a Semai family has had a quiet evening with cassava – slowly roasting the tubers over the coals of a dying hearth fire and then enjoying a filling snack. Cassava, a perenniel shrub originally from South America, may have been brought to Southeast Asia centuries ago by the Portuguese or Spanish, probably both. Malaya is said to have had a large cassava-starch industry for 50 years or so before cash cropping of rubber elbowed it out of the colonial trade arena.

Cassava has been successful in many places where it was introduced. It is easy to cultivate on suboptimal soils; a plant's tubers "keep" in the ground for long periods – unlike grain foods that must be harvested immediately when ripe; it is drought-resistant; and it has high yields. One cassava plant has been known to produce 50 pounds of food. Unlike the starchy tubers of cassava, its leaves are rich in protein. And the leaves can be harvested over a period of a year or more – unlike the short harvest period of most green vegetables. According to Osman and coworkers (1992), among the Temuan at Kuala Pangsoon, Selangor, 59% of those age 7 years or older ate cassava tubers or leaves at least once a week. The chance of developing goiter for those eating cassava at least twice a week was 4 times higher than for those eating it less often.

Cassava, then, is an easy, reliable, and important investment toward a stable food supply – a sort of nutritional insurance plan. No wonder then that nutritionists call it an anti-famine crop.

The dark side of this story is that cassava is an important source of goitrogens, in Malaysia and elsewhere. Many cassava varieties contain a plentiful supply of cyanate, which inhibits iodine uptake by the thyroid gland, causing levels of thyroid hormones to be lowered (note 51). While the tubers have enough cyanate to cause trouble for thyroid functioning, the leaves have 3 times the amount found in the tubers (Simwambana et al., 1992). Because cassava is a popular energy crop

for Orang Asli (Khor, 1988), it is thought to contribute to the level of goiter found in their communities. By the same token, it is possible that prior to the introduction of cassava long ago, goiter was less of an Orang Asli problem than it is today.

Goiter occurs among the Orang Asli because of their poverty and the social-cultural discrimination against them. Yet goiter is not inevitable. Only a little preventive "medicine" is needed to overcome this iodine problem – iodized salt, ikan bilis (dried anchovies), spinach, amaranths, and other green vegetables are part of the solution. They all contain beneficial levels of iodine. While it is apparently government policy to combat rural malnutrition by, for example, "nutritional rehabilitation" (Osman and Zaleha, 1995), this policy does not seem to be improving the thyroid function of Orang Asli villagers to any marked degree. Even such a simple item as iodized salt is not freely available to them, despite the fact that it is more expensive than "local" salt and is not carried by many rural food shops. Although iodized salt was once distributed to Orang Asli "in the deep jungle" (Bolton, 1972a), more comprehensive measures are obviously needed today.

A possible problem with iodized salt in West Malaysia is that some so-called iodized salt in East Malaysia was found to be uniodized (Osman Ali, personal communication). No report on iodization in West Malaysia has appeared. Injections of iodized oil have been tried in Borneo (Tee, 1993), since the oil provides iodine to the body for a year or longer; some problems have been reported with this approach, however.

Maternal Survival

Threats to maternal health such as anemia and various disease states may go far to explaining the poor survival of women versus men at advancing ages. As shown for Semai (Khor, 1985), women are less likely to survive through adulthood, in contrast to the all-Malaysian situation in which older women are more numerous than older men (see Table 5).

Maternal deaths in childbirth are relatively frequent among the Orang Asli (The Star, 29 Sept., 1996), according to anecdotal and governmental information, but few studies have been done (note 52). The report by Fix (1977) on Semai is a notable exception. Life-threatening complications during childbirth include obstructed labor, hemorrhaging, and infections (Nowak, 1995). All this suggests that

prenatal care is not sufficient for maternal health; effective obstetric care is also required. Strategic misjudgment has long underrated obstetric care. When women's status is low, their health is not a priority. It seems that well thought out changes are overdue.

ORANG ASLI SURVIVAL IN THE PAST

There is little on which to base an early history of health for Orang Asli, except some semi-theoretical considerations (May, 1983; Fix, 1984). "On the ground" reporting is thin, until long after the Portuguese conquered Melaka in 1511 (note 25ee).

A few guesses can nonetheless be made about the serious infectious diseases in the Orang Asli realm before Indians, Arabs, or Europeans arrived in the region (Knapen, 1996; Owen, 1987). The oldest endemic diseases in the region probably included malaria and dysentery, both of which can have severe, sudden consequences. Less dramatic, but still significant in contributing to disease and death, may have been intestinal worms, microfilarial infections, malnutriton, anemia, and puerperal sepsis following childbirth. In combination, such ills would have markedly lowered Orang Asli vitality and reproductive success (note 54). Leprosy and yaws, while chronic and occasionally disfiguring, may have been too rare to have impacted minority populations in Malaysia. Goiter, while undoubtedly ancient and common in inland areas, is thought to be demographically unimportant because the severe form, cretinism, is exceptional; but resulting mild retardation might be more of a problem (Warren and Mahmoud, 1990). While all of these ills are related to ecological parameters in one way or another, venereal diseases and herpes virus (Cohen, 1989), related more to the social environment, may have presented other ancient problems in the region. Venereal diseases were widely reported in Southeast Asia from the 16th century onward; indeed, in the 1930's, 80% of Bornean Muruts were reported to have gonorrhea (Reid, 1987) (note 55).

Well-known epidemic diseases such as smallpox move easily and quickly from person to person, causing either death or survival with long-term immunity. Such diseases require large populations to continue indefinitely. While smallpox was known in India and China perhaps 2000 years ago, it may not have appeared in Southeast Asia until several centuries later – perhaps arriving in the Philippines, for example, after 300 A. D. (note 56).

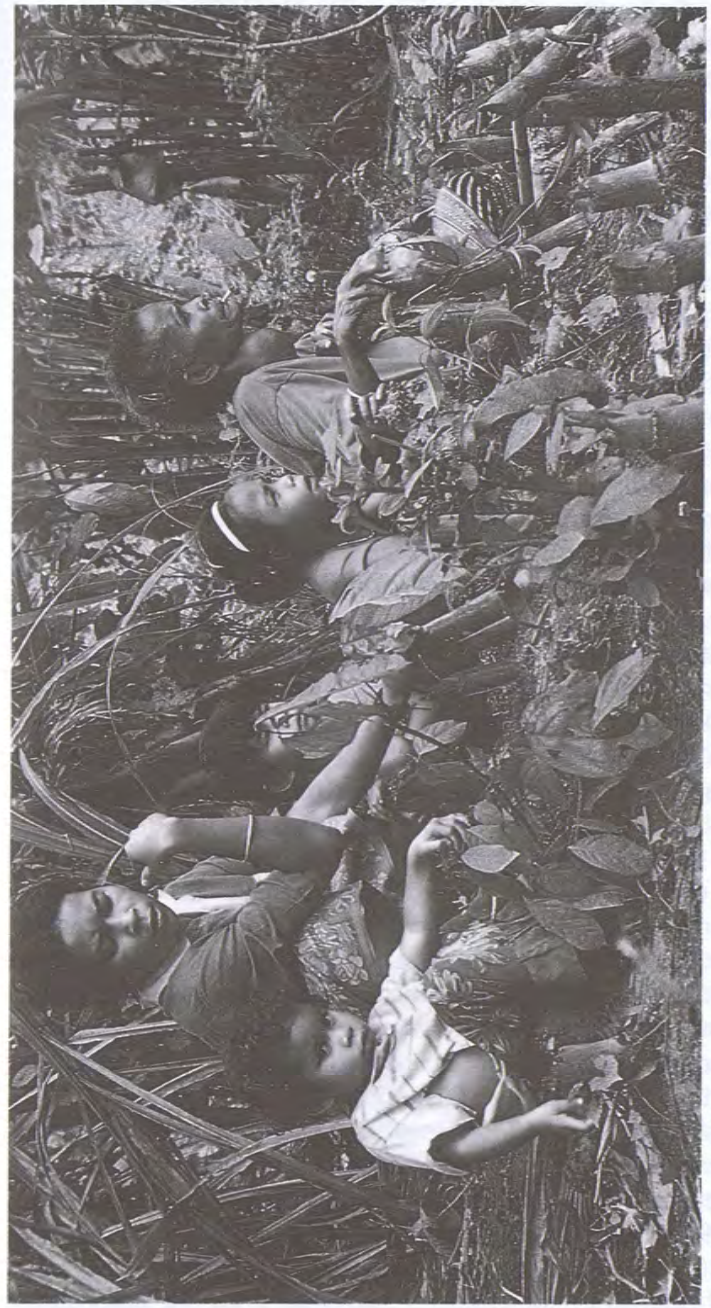
Variolation either via the Chinese or Europeans started in Southeast Asia in the late 18th century, vaccination about 1860. According to Gimlette and Thomson (1971), vaccination started on the Malayan East Coast in 1905. But Polunin (1953) found only 20% of the Orang Asli he surveyed had been vaccinated: 51% of the lowland Semai, 25% of the Seletar, 3% of the Lanoh, and none of the highland Semai. A smallpox epidemic occurred among "aboriginal Malays" in Pahang in 1947 and 1948, producing an official death rate of 68% among 103 known cases (Polunin, 1953). Smallpox was eliminated from Malaysia in 1960.

Orang Asli groups were historically small. As such, during the colonial era their smallpox epidemics necessarily originated through outsiders. The epidemics came in waves, probably at intervals of 15 years or so (Knapen, 1996). The imported epidemics could lead to mass flight, a shortage of community caregivers following adult deaths, neglect of food gathering or food crops, and thus malnutrition, even starvation. Directly or indirectly, then, smallpox probably caused the extinction of some local communities (Polunin, 1953). Population-wide mortality may have been as high as 25%, by all accounts, affecting all ages. In contrast, deaths from "older" diseases, such as dysentery and malaria, mainly affect the very young.

Epidemics may have had little impact on Orang Asli groups until the 18th or 19th centuries, as neo-Malaysian populations grew and regional migrations (and pilgrimages to Mecca) increased (Cohen, 1989, p. 137) (note 57). Batek have reported that before World War II their population was much larger than at present and that great numbers died in disease epidemics brought into their homeland by immigrant Malays (Endicott, 1997). But epidemics also occurred after the Japanese surrendered, one or more occurred about 1950 and another in 1978, this one a lethal intestinal infection.

Just as for smallpox, the "crowd diseases" of cholera, typhoid, flu, and measles can have high mortality rates. While the death rate for cholera might reach 50% (Knapen, 1996), mortality from more chronic conditions, such as tuberculosis, can also be substantial (Owen, 1987). Yet tuberculosis may be a more severe disease now than it was in prehistory (Cohen, 1989, p. 86).

Wars, insurgencies, and the like have produced another layer of hazards. The roundup of Perak Semai in the 1950's by British colonial strategists resulted in widespread morbidity and mortality, probably



Colin Nicholas

Semai female elder with goiter, Ulu Woh, Perak.
Goiter is common among inland Orang Asli groups due in part to the iodine-deficient diet as well as iodine-depleting goitrogens found in cassava, a common staple. Also, Orang Asli women have a higher goiter rate than men. Iodine-deficient pregnancies can also result in the mental retardation of newborns.

due to infectious hepatitis according to Dentan (1988). Other changes in recent centuries in the ecology and social setting of the Orang Asli undoubtedly brought about other, largely unknown, changes in disease patterns and disease severity (note 58). For example, slave raiding prior to 1920 undoubtedly had both social and health detriments (Endicott, 1983; Dentan, 1991).

In traditionally dispersed Orang Asli populations, zoonotic diseases that could be maintained in non-human hosts, producing animal reservoirs for human infection, may have often been fatal to people. Indeed, they sometimes still are. Such zoonoses include dengue, scrub typhus, and malaria.

For most of these infections, success in averting individual or group morbidity or mortality would largely depend on general nutrition and health, the level of social cohesion, and the proportion of adult providers in the population.

Carey (1976) summarized the health condition of the Orang Asli as he knew it in the 1970's. Since he was writing on his experiences as a government official, but not a biomedical specialist, his outlook was both positive and derivative. Accordingly, he reported that the government's aim of "raising standards of health and hygiene has largely been fulfilled." This statement is not a comparative one, vis-a-vis other Malaysian ethnic groups. And it is undoubtedly based somewhat on visible improvements in some villages, such as new wells and new buildings. It is also based on what Carey described as "the excellent medical facilities" by that time available to Orang Asli and to his belief that the infant mortality rate was declining (note 59).

As the reason for this assumed decline, he maintained that "nearly all" Orang Asli women had their babies at Gombak Hospital, where deliveries were safer and more hygienic than in villages. He proffered no evidence for the belief about "nearly all" deliveries being at this hospital, and, indeed, other observations suggest a contrary situation.

Khor (1985), for instance, found that 70% to 91% of Semai women in various areas had their babies at home. And McLeod (1971) estimated that only 25% of Orang Asli births occurred in hospital. Further, few published data exist on the infant mortality rate (IMR) for the Orang Asli; those that do exist are far from comprehensive. Fix (1971) reported the highest IMR for Orang Asli: a rate of more than 20% for Pahang Semai during the years 1955 to 1969. Khor (1985) reported a range of zero to 7 offspring dying per Semai family

before reaching the age of 5, suggesting that infant and toddler mortality remains a considerable problem. The official IMR for Orang Asli for 1984-1987 was 5.2%, or 3.2 times the West Malaysian average (Ng et al., 1987). Even then, this rate for Orang Asli may be an understatement, due to underreporting.

Carey further commented (1976, p. 332) that Orang Asli women previously thought they must have large numbers of children because half would die before reaching maturity. But "this...was no longer true." However, pre-adult death rates have rarely been studied in detail.

As I discussed earlier, the population growth of the Orang Asli after World War II does not per se require low childhood mortality if fertility rates are high and/or surviving members of communities live longer lives. Hypothetically, if those who previously died at age 50 lived to age 70, this change alone could increase the existing population by perhaps 5%.

About 20 years ago the government's medical service unit for the Orang Asli put forth a grand plan (Khoo, 1979). It also has relevance today. The first-priority program in the plan was community education. Second was maternal and child health, given the high death rate of infants and toddlers, rampant malnutrition, "maternal depletion," neonatal tetanus, and other problems. Third was the control of communicable diseases, given that TB and malaria were widespread and caused many deaths. Fourth was environmental sanitation, including the provision of potable water. Fifth was the improvement of "curative services" and the referral system. Sixth and last was the gathering of health statistics and related health information.

SURVIVAL NOW

It is difficult to estimate the major medical problems of the Orang Asli at present, not least because now – as previously – many conditions are not seen by, or reported to, governmental medical personnel (note 60).

According to Hamid (1990) the most common complaint recorded in recent years for Orang Asli at medical clinics was non-malarial infections or infestations, followed by diarrhea and then malnutrition. Between 1981 and 1985 the most common illness recorded for Orang Asli patients at Gombak Hospital was malaria, followed by gastroenteritis and tuberculosis. Conditions such as dental caries, alcoholism, and goiter would not be recorded in such tabulations.

It is likely that the overall stress of the Orang Asli's medical problems today – encompassing all the new, waning, and persistent ailments – has not lessened much in recent decades, except that yaws may have been eradicated (Chee, 1995), filariasis may be rarer, and smallpox is extinct. No health survey as provided anything like a comprehensive report on a single Orang Asli community in recent years.

Today, many Orang Asli individuals – and other Southeast Asians – are malnourished from an early age and are vulnerable to infant diarrhea, intestinal worms, tuberculosis, and malaria. While some notable infectious diseases are now easily curable and increasingly rare, malaria, dysentery, and intestinal worms have not been "cured" as much, among the Orang Asli.

Although Orang Asli mortality has declined and populations have grown over the last century, morbidity has not been reduced to the same extent. While it is true that as villages grow and crowding increases, parasites circulate more rapidly, Owen (1987) speculated that this modern disjunction between death and illness is due largely to the fact that "modern" tropical medicine ignores the role of poverty and malnutrition in causing disease. "Modern" political systems also have something to do with this situation. The principal, appreciable risks to Orang Asli health are poverty and malnutrition, although the ultimate causes of these risks lie elsewhere (note 61).

Urbanites may not think of poverty and malnutrition as major risks to Orang Asli health; rather they may mistakenly focus on the stress of (erroneously assumed) "nomadism;" the lack of a stable supply of polished rice; the erroneously assumed "dirty" living conditions (Carey, 1976) and indifference about health; life in mosquito-ridden areas; and other factors. Such misconceptions have not helped improve Orang Asli health. In other matters, the curative facilities available to Orang Asli at the hospital in Gombak are hardly supportive of the image of Malaysia embodied in the new Twin Towers in Kuala Lumpur, now the world's tallest building. As Veeman (1986/87) first pointed out, the "temporary" wooden structures at Gombak Hospital built in 1959 by Orang Asli workers still house patients (even in 1998). And, among other inadequacies, hospital staffing there still lacks specialists in various medical disciplines. Nor is health education properly addressed by the current medical bureaucracy for the Orang Asli. Improvement is long overdue.

Chapter 9

THE CHANGING BIOCULTURAL WORLD:

THE FUTURE HEALTH OF ORANG ASLI ETHNIC GROUPS

In this chapter I discuss Orang Asli health in the future from the perspectives of community action, of "civilization," and of government programs. Two questions seem critical in thinking about this future. For Orang Asli, the medical and university specialists, and others concerned about Orang Asli health, one question is this: Is today's information sufficient for promoting an informed consensus on the need to improve health? I believe that, despite gaps, the information is sufficient. The previous chapters of this book provide a wealth of data. The follow-up question then is whether existing conditions permit a consensus to emerge on workable methods for such improvement. With good will, this could be an easier step forward than the initial one of becoming informed!

A textbook on community medicine in Southeast Asia declares, "The WHO statement 'Health for all by the year 2000' is familiar to all who work in the health field" (Phoon and Chen, 1986, p. 313). If this is a goal on which diverse interest groups can agree, a good way to achieve this goal is to start with community empowerment. This is a view that is apt to be misunderstood. In this view, a "top down" approach is upside down. With respect to hookworm anemia, for example, work on surveys, prevention, and control should start at the village level, according to WHO, the World Health Organization (Pawlowski et al., 1991, p. 53):

> "Such an approach ensures that each community is fully involved from the beginning in investigating its own hookworm anemia problem, in choosing the most suitable measures for prevention and control, in finding at least some of the resources required, and in playing an active role in control measures.Community members may contribute in many ways,

for example by enlisting further public support, by keeping records, by distributing iron tablets to schoolchildren and pregnant women, and by providing labor and materials for latrine construction and drainage."

WHO's description of a successful program against hookworm anemia is detailed. It could easily be adapted to combat other medical problems, given a flexible agenda by government agencies.

Osman and coworkers (1993c) studied the effectiveness of training and using village health workers for surveys and services for Orang Asli. They emphasized that community participation in primary health care helps develop self-reliance and self-determination, since the success of health programs ultimately depends on the community. It knows its own needs, problems, and priorities best. Government agencies, universities, and nongovernmental organizations can facilitate community decisions on how to meet these health needs. Good mediators in this process are in-village health volunteers, or cadres. Such people provide an inexpensive way to extend health care to the village level. Their presence encourages wide participation in health programs; indeed, in this particular study participation was higher in villages where the volunteers worked than in "control" villages where no volunteers were used. Cadres were selected by the head of the village; they were 20 to 25 years old, had had a secondary education, and were well motivated. They were trained at the university for one month in interviewing skills, measuring height and weight, and the like. Upon returning home, they collected demographic data, food consumption information, and stool specimens. Osman's team urged that universities offer training for village health workers, as part of their role in providing community-level services. When a university and community work together, more can be accomplished than when each works alone.

Some health improvement can be obtained at relatively low cost. Jamison and coauthors (1993) have published an authoritative list of such health strategies. Many are applicable to Orang Asli concerns. Here are some items on their list:

1. promotion of breastfeeding (community health effort);
2. DPT, polio, measles, and anti-TB (BCG) immunizations;
3. iodization of salt (mass chemoprophylaxis and community health effort);
4. fortification of sugar with vitamin A;

5. hepatitis B immunization;
6. targeted mass antihelminthics (frequent mass chemoprophylaxis and community health effort);
7. use of condoms to prevent excess births and sexually transmitted diseases (community health effort);
8. daily oral iron during pregnancy (mass chemoprophylaxis and community health effort).

Beyond such medical strategies, cultural factors are most important for the future health of Orang Asli. If culture is a "kind of blueprint for survival" (Polunin, 1986), the dissolution of a culture may be a blueprint for disaster. And dissolution may be happening.

Cultural erosion has been perceptible for some time. It's been a bad century for the Orang Asli (note 62). From slavery and smallpox epidemics to someone else's war, to being cooped up in government-organized villages with no forest in sight. Presumably the ills of "civilization" will increase in Orang Asli enclaves over time: poverty, drug addiction, suicide, alcoholism, AIDS, diabetes, hypertension, and so on. Already in 1991 Osman and coworkers reported that, for those 7 years and older, a third of the Pangsoon Temuan smoked cigarettes and 30% consumed alcohol (usually during festivals or other special occasions). However, many Orang Asli groups are well known to be unaccustomed to alcohol. While rare cases of opium addiction were reported by Schebesta (1973) for the Kintak in the 1920's, today a dope pusher may relentlessly "push" at a local Orang Asli community with impunity, to support his habit.

This outlook of heightened ill health for the Orang Asli may appear too gloomy for those Malaysians who think of modern life as full of health benefits and Orang Asli life as full of health risks. Modernity should improve the Orang Asli lot, one might argue. If Orang Asli were prosperous and citified, this might be true, but it is unlikely they will occupy this niche any time soon. Rather they will be increasingly bereft of their ancestral way of life while increasingly subject to the risks of so-called development, that is, internal exile in administrative villages perhaps surrounded by logged-over wastelands or on the perimeters of predatory "developments" such as golf courses.

Khoo (1979) listed 6 negative features of forced relocations to one potentially positive feature, in terms of Orang Asli health:

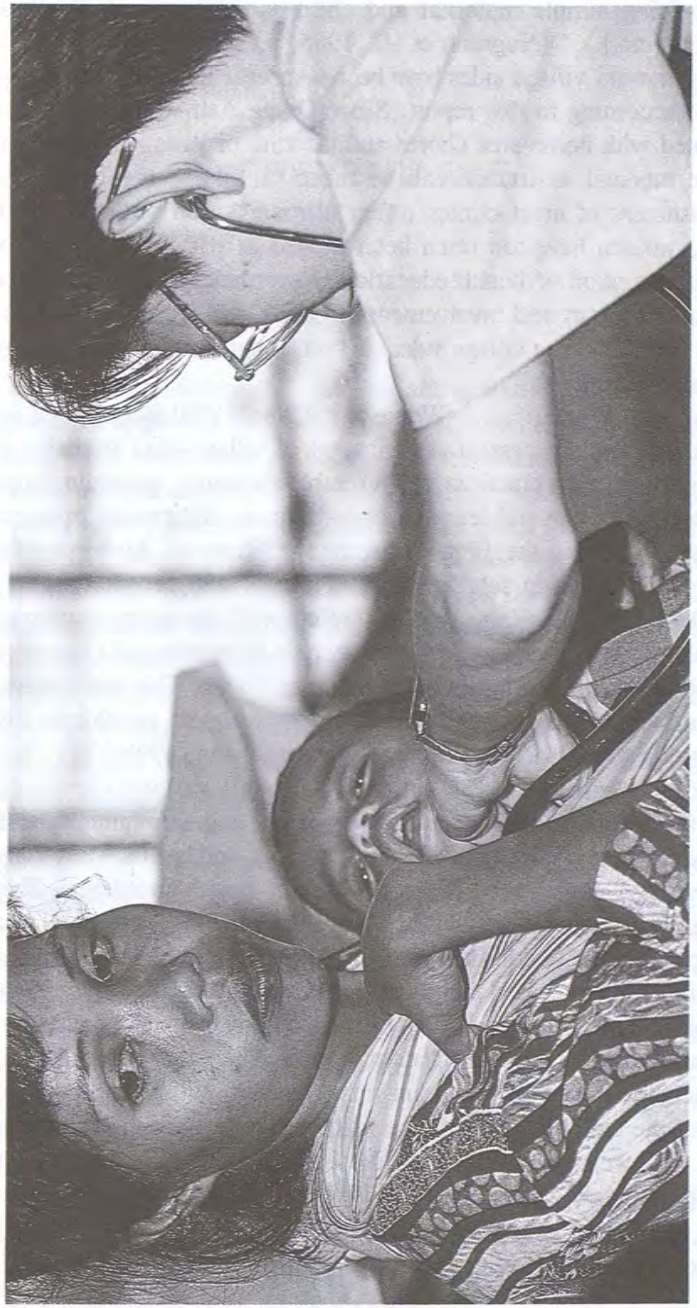
1. the dislocation of Orang Asli groups;
2. crowding at the relocation site;

3. rapid depletion of natural resources (especially food) at the new site;
4. pollution of the new environment, primarily of soil and water;
5. ecological degradation at the new site;
6. accelerated social change;
7. more cost-effectiveness of health-care delivery.

Top-down programs for health services of the sort imposed on the Orang Asli have been found to be unsatisfactory in many countries. The experience in Malaysia does little to suggest more justification exists for them there than elsewhere. By changing from an "in locus parentis" approach to an appropriate one, officials and non-officials will be able to improve medical services, especially preventive services, for Orang Asli. This will entail a paradigm shift from bureaucracy to community, from office-based medical practice to a grassroots educational approach and community-based decision making.

This being said, it is well to acknowledge that health is only one facet of life's fortune. One might reasonably argue that the maintenance of health is not the primary goal of most human behavior – including social-political behavior. Still, on a national scale Malaysia may pride itself on improvements in health and nutrition. Yet on closer inspection, this may only be a partitioning of health stresses, with successful groups enjoying access to good health and nutrition in contrast to the Orang Asli in particular – as a result of their loss of traditional resources, crowding in "modern" villages, and bottom-rung social status. For example, the head of Gombak Hospital recently noted that Orang Asli health was not well served by the "staff attitude" at medical facilities (Roslan Ismail, 1997). Moreover, it is noteworthy that as early as 1957 Gerald Templer pointedly remarked (Holman, 1958, p. xi) that Orang Asli had a right to effective medical services from the government which "should never be forgotten."

Bolton (1973b) gave details of Orang Asli participation in improving their health in the 1970's, from the optimisitc viewpoint of Gombak Hospital experiences. According to Hamid (1990), the Medical and Health Services Division for Orang Asli still trains the staff at Gombak Hospital and holds refresher courses for traditional midwives and field assistants at its medical stations (note 63). He did not mention the training of "village aides," as has been done in Sarawak, East Malaysia. Village aides there reportedly work part-time, are selected by the village for the village, and "are trained to assist mobile teams



Colin Nicholas

A member of the medical team from Ipoh Hospital attending to Temiar at Kampung Bawong, Upper Perak. The success of health programs for the Orang Asli ultimately depends on the community itself – since it knows its own needs, problems, and priorities best. Community participation in primary health care not only promotes self-reliance and self-determination, but is frequently cost-effective as well.

in providing simple maternal and child care, treatment of minor ailments [etc.]..." (Nugroho et al., 1986, p. 342).

In Sarawak, village aides have been a success in improving village health, according to this report. Since Orang Asli women are often burdened with home-area chores and the care of young children, in-village medical assistance can be more useful for them than the establishment of areal clinics (often in more urban settings). Even worse, women have too often been treated as passive recipients of medical attention or health education. Nevertheless, mobilization of the active support and involvement of Orang Asli women in medical issues (such as being village aides) is fundamental to the improvement of Orang Asli health.

Wong (1991a, b) and Wong and Chen (1991) described an imaginative and successful combination of village-level initiative in fostering sanitation practices and vegetable gardening, providing good preschool nutrition and learning opportunities, and raising maternal self-esteem among the Berawan women in Sarawak. Moreover, the cost of this self-help scheme for a village of 450 people was only $4000 Malaysian dollars. This outlay covered the costs of training workshops, transport, and support materials for nutritional education, as well as the furnishing of a kindergarten room. This amount was one-thirteenth of the expenditure by the government to relocate just one family for a land development scheme (Wong, 1991b).

Wong's pilot program started with all-village meetings, identification of village needs by the women, and a community self-survey on demography, housing, food supplies, and health. The village women then constructed a priority list of health problems. They unanimously ranked their children's health as the top priority. A plan to improve childhood health was evolved that led to the establishment of a kindergarten, based mainly on maternal participation. Nutritious school-time meals were provided by the mothers, utilizing "dedicated" vegetable and fruit gardens. District government workers in agriculture and health assisted the program upon request. Whereas previously 35% of the children proved to be malnourished, a year into the plan many were wearing sandals and almost all had noticeable weight gain; also, breastfeeding increased from 70% to 91%. This fieldwork shows what can be accomplished when one goes beyond off-the-shelf procedures and the "value-laden assumptions" of central planners.

Postcript

SEBOI: A PREVENTABLE TRAGEDY?

One evening in February, 1997, an anti-malarial team of two health personnel arrived in one section of Seboi, a Jah Hut village in Pahang state. They went house to house. At each house they called out the names of residents to be given anti-malarials, from a list they carried. Adults were given tablets. Children were given three types of syrup (milkish, orange, or purple in color), 10 ml. of each syrup to each child, irrespective of age. The team asked no questions, not even whether anyone was ill or if people had already eaten their supper.

Shortly after the team left the first-visited house, the family's four children became violently ill, being nauseated and vomiting. In turn, children in neighboring houses also became violently ill. The father from the first house bicycled after the mobile team three times in all to inform them of the problem. Initially he was told, "Tak apa, jangan bimbang" (Don't worry, it's nothing). By the father's third trip, other parents were also complaining to the team. No first-aid treatment was provided, but the affected children were taken by landrover to a government clinic and then by ambulance to hospital, involving an hour or more for the total journey. Of these 11 children taken away, two died – in transit to hospital.

Weeks later, an investigative team came to the village to sample vegetable-garden leaves and the water supply, and to check the place where herbicides, pesticides, and fertilizer were stored for the rubber-tree plantations. They did not interview the villagers or take blood samples.

In the meantime, piecemeal reports of the incident appeared in the press. These stated, among other things, that a police report had been filed. Later the affected families sought legal aid, and the Bar Council Legal Aid Center took up their case.

One government spokesman opined that the children who died had already contracted malaria and it triggered their deaths; he also held that the anti-malarial syrups could not have caused their deaths because not all the medicated children died (The Star, 22 Feb., 1997) – perhaps based on the assumption that all humans respond uniformly to drugs. Later it was suggested to press reporters that pesticide spraying in a nearby agricultural area, an unclean water supply in the village, or an accidental drug overdose might have caused the Seboi tragedy (The Sun, 28 Feb., 1997). Several days earlier, one non-medical commentator had already brought up other possibilities (The Star, 25 Feb., 1997): plain negligence or the involvement of a genetic condition called G6PD deficiency (discussed in Chapter 7). Indeed, independent testing of nine Jah Hut hospitalized survivors of this Seboi tragedy later revealed that two of them did have the G6PD condition, as did others in the village. Two other children with the condition were brothers of a child who had died. But not all of the nine child survivors were G6PD deficient, suggesting that other factors were involved in their acute attacks of illness.

Following the press publicity, the federal health ministry appointed a 15-member ad hoc committee, including pharmacists and anti-malaria experts, to review the incident and provide a report on it. Although reports on some medical tragedies have been made public only weeks after investigations were carried out, no report on the Seboi case surfaced over a period of many months (Nicholas, 1997), or longer.

***Commentary*:**

Whatever the actual events at Seboi in February, 1997, it appears that serious problems exist in health-care delivery for Orang Asli, if this case is in any way typical. These problems range over a number of areas.

The first area to be mentioned is information flow. It was not at its best in this instance. Press reports were neither complete nor entirely accurate, suggesting that relevant information was not fully and freely available to the public or to government commentators. As the Malaysian Medical Association has stated, "all health services information except those [medical data] pertaining to individuals should be in the public domain" (Rajakumar et al., 1980, p. 91). In the Seboi case, gaps in information were quite noticeable. An early press story (New Straits Times, 21 Feb., 1997) reported that all 117 villagers

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Jah Hut family grieves for a wasted life, Seboi, Kuala Krau, Pahang.
As a result of a mass anti-malarial program in the village of Seboi in 1997, this family lost a two-year-old daughter. In the same incident, another relative (an 8-month-old baby boy) died while another ten children were hospitalized. The authorities maintained they were not at fault.

received the medicine, but this was not the case. The next day the Malaysian health minister was reported as saying (The Sun, 22 Feb., 1997) that the Seboi children received pills, although they actually received liquid drugs.

Some reports said two brothers had died, but in fact one boy and one girl died, and they were from different families. No mention was made in press statements about the civil servant responsible for anti-malarial policies and practices, how the quality of this program was monitored, or what standard measures were taken in cases where errors or mishaps occurred. Nor was the public informed of the names of all the "experts" on the ad hoc review committee and what the responsibilities of this committee were to the public and to the families involved.

In addition, the citizens of Seboi were not informed as to why certain individuals were to be administered anti-malarial drugs, and individuals were not asked for their consent for this drug administration. They were also not told the contents of the medications or their possible harmful effects. Finally, it has been well known medically for decades that the Jah Hut subgroup of Orang Asli has the highest recorded frequency of the G6PD condition in West Malaysia (23%, see Table 6) and, just as importantly, that G6PD-deficient people are susceptible to life-threatening conditions if they take primaquine (a common anti-malarial in Malaysia) or a few other chemical compounds.

Inexplicably, no questions apparently were asked as to whether any Seboi people had this condition, nor were any of them tested for it before the drugs were to be administered. Indeed, it seemed to be assumed in government circles that no medical evidence existed that primaquine, for example, could ever precipitate death (The Sun, 22 Feb., 1997).

And the Seboi case was not the first one in Orang Asli communities where deaths occurred following administration of anti-malarials, according to information in the press (The Star, 25 Feb., 1997; Uthmani, 1993). Moreover, in a 1993 case with one childhood death and the hospitalization of 19 other Orang Asli – also Jah Hut – from Sungai Merjok, Pahang, no governmental follow-up report was ever made public (Nicholas, 1997).

The second area to be mentioned is closely related to the problem of information flow; it is the question of trust and rapport. Some

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Taking blood samples for G6PD tests, Seboi, Pahang.
The Jah Huts are known to have the highest recorded frequency (23%) of the G6PD condition in Peninsular Malaysia. Blood samples taken in Seboi and tested independently confirmed this frequency. Intake of primaquine, one of the anti-malarial drugs dispensed, can be life-threatening to G6PD-deficient individuals, a factor overlooked in the fatal mass-dispensation of anti-malarials.

health-care providers for Orang Asli have been admonished in print for their "attitude" in this regard.

Part of the problem here is lack of trained staff and inadequate funding (for training, maintaining ample staffing levels, and so on), as stated by Veeman (1986/87). And Fadzillah Kamaluden (1997) held that medical practitioners are unaware of the social and cultural practices of Orang Asli, thus undermining much chance for rapport with them. Similarly, Khoo (1979) emphasized the need for community education in Orang Asli areas, presumably including consensual planning and execution of health-related practices. Hii and colleagues (1996) went further; they described an anti-malarial program in Sabah that owes its success in large part to "cooperative networking between the government health sector and the community" (p. 512).

More generally, the Malaysian Medical Association years ago strongly advocated "health activities at the grassroots level with participation by the community" (Rajakumar et al., 1980, p. 156). In addition to these problems, strenuous efforts have not been made to recruit, train, and retain Orang Asli themselves, rather than members of other ethnic groups, as primary health-care providers, thereby missing a bulls-eye chance for improving trust and rapport with Orang Asli health-care clients.

The third area to be mentioned is that of standard procedures used by medical personnel in the face of untoward events. It is not clear if all health-related personnel dealing with the public are trained to provide first aid, that is, if they are qualified as paramedics. If this is not the case, this lapse could be rectified in a short period. The question here is whether medicine is being practiced optimally by those who work first-hand on health issues in rural communities, especially those communities remote from hospitals, telephones, and other avenues for sharing decision-making with urban medical experts.

The fourth area to be mentioned is much broader in scope: it involves the question of lines of authority and responsibility for health policies and practices for Orang Asli. The issue of malaria control among the Orang Asli provides one example, but much that can be said about this issue also applies to others that affect Orang Asli health. To start with the Seboi case, the minister of health was reported as saying that the government's main concern was to prevent such an incident from recurring (The Sun, 22 Feb., 1997). He also said that the Malaysian health ministry might need to centralize the production

of drugs so that "the mixture of the anti-malaria medicine is not based on individual judgment" (The Sun, 28 Feb., 1997), despite the fact that age-calibrated, prepackaged tablets have already been used in Sabah (Hii et al., 1996).

But no government official was reported as stating, when the issue of G6PD deficiency arose outside of government circles, that all high-frequency groups would henceforth be tested for this condition before primaquine was advised for them, nor did any official evidently state that individuals would henceforth be asked about their G6PD status before drug administration.

It follows that also no mention was made of providing standard written information to those who are found to be G6PD deficient as to which drugs are contra-indicated for them. The health minister was only reported as saying that if it was "established" that "10 to 12 percent" of Orang Asli had the G6PD blood disorder, then they would "have to ensure that screening [for G6PD deficiency] is done before treatment" (New Straits Times, 28 Feb., 1997).

(Yet in spite of these press statements, it seems that all West Malaysian infants born in hospital are indeed tested for G6PD deficiency, and the parents of the G6PD deficient infants are given a card to this effect which, according to one medical practicioner, lists contra-indicated drugs.)

In a related context, no official apparently thought to mention that the anti-malarial campaign in Orang Asli areas could profit by more adequate funding for training, mobile clinics, monitoring of effectiveness, reviewing and updating policies, carrying out needed research, and so on. Yet as Diggs (1992) has pointed out, most national anti-malarial campaigns suffer from a lack of sufficient personnel, especially personnel with an adequate understanding of all aspects of devising and maintaining such a complex field program.

Questions of authority and responsibility extend to broader arenas as well. The first set of questions in this regard include the following: Who in government is ultimately responsible for the anti-malarial campaign among the Orang Asli? How is this responsibility exercised in terms of formulating policies, the bureaucratic chain of command, public accountability, and quality control?

A second set of questions include the following: What are the standards set for the duties of anti-malarial workers – and indeed for all health-service providers and their supervisory personnel for Orang

Asli? If a report on these standards is not publicly available, what is the reason underlying this decision?

The fifth area to be mentioned involves ways that might be considered for improving the existing anti-malarial program for Orang Asli. I have already referred to the possibility of training more Orang Asli as health workers. The distant prospect of anti-malarial vaccines has a certain allure, but other procedures may be of more immediate, practical importance. The current program relies greatly on "modern" drugs and insecticides, particularly DDT fogging of Orang Asli living areas and chemically-treated bed nets. The "traditional concerns for drainage, shading, oiling, and other techniques" are largely neglected today in West Malaysia (Aiken and Leigh, 1992, p. 107).

In addition, not all Orang Asli are supplied with a bednet. For over 95,000 Orang Asli, 13,000 treated nets were supplied at some unspecified time (The Star, 23 Feb., 1997). Even in areas where they are supplied, more than one net needs to be given per household. As might be expected, in a typical multi-generational household, not all the members sleep together under one net. Older children, grandparents – let alone visitors – sleep separately. Four untreated bednets per household might very well be more efficacious against malaria than one per household that is chemically treated – but no tests of this possibility seem to have been conducted or reported in the biomedical literature.

On the question of DDT spraying of houses or the fogging of surrounding areas, this practice has already been discontinued in some Orang Asli, and other, locales by medical personnel because it proved not to be effective (Vythilingam et al., 1995; Hii et al., 1996). But for many locales, the practice continues for reasons that are unclear. Already mosquitos in some parts of West Malaysia have developed resistance to DDT (favored for indoor spraying), malathion (used in outdoor fogging), and permethrin (an insecticide used to impregnate bednets) while "in most areas...malaria transmission has continued despite DDT spraying" (Rohani et al., 1995, p. 44). Since DDT has been part of the anti-malaria program in Malaysia since 1967 (Loong et al., 1989), a build-up of DDT resistance in mosquitos might have been recognized as inevitable.

And it is well known, world wide, that DDT (among other insecticides) is environmentally harmful. Most nations have banned any use of DDT and many also severely restrict the use of other

potent insecticides (including their use in agriculture). It also needs to be emphasized that Orang Asli, among others, do not live their entire lives inside houses. They are occasionally away from home at night: for example, on forest expeditions or guarding their farms from noctural predators. And, as many investigators have discovered, mosquitos deterred by insecticides of any kind can indeed change their behavior, by going to untreated houses or simply biting people outdoors before they retire to bed.

Hii and colleagues (1996, pp. 512-515) have reviewed the general problem of anti-malarial work in rural Sabah. They mention the "considerable difficulties" of a "vertically-oriented malaria control program" that resorts to semi-annual DDT spraying and mass drug administration on an ad hoc basis. The lack of success of such a program is compounded by "inadequate access to health facilities" and the "limited availability of over-the-counter antimalarial drugs," among other factors. They emphasize that the key concepts of primary health care include, foremost, "the continued active role of the community for whom the program is intended and who are the final beneficiaries." In their work they combined elements of a bottom-up with a top-down approach, with notable success. It involved village health volunteers and networking between the health sector and the community in running a community-managed malaria control program. Their first step in establishing a sustainable program was to obtain consent from villages included in the plan, after an explanation and discussion of the details of the program. In consenting villages a village health volunteer (VHV) – unpaid – was then selected, as was also a "standby" VHV. People who were married, functionally literate in Malay, and motivated to participate were preferred as VHV's. The VHV's received a series of 2-day training courses on-site, including illustrated talks, discussions, and practical sessions about the malaria life cycle, disease symptoms, diagnosis and treatment of malaria, and so on. Each VHV received a medical kit containing antimalarial drugs, analgesics, and vitamin and iron supplements provided by the health department. Since Hii and his colleagues had found that malaria can kill children within only a few days after the onset of symptoms, the VHV's were instructed to treat fever attacks even before blood-slide diagnosis of malaria was available, with – as mentioned previously – age-calibrated, prepackaged single-dose tablets (containing chloroquine and primaquine). (I should note that the authors did not mention

G6PD deficiency at all, despite the fact that Khoo reported in 1981 that some G6PD-deficient malaria patients in Sabah developed acute renal failure or required blood transfusions after primaquine administration.) If malaria was later confirmed by blood-slide evidence, the VHV gave further treatment. All work by the VHV's was supervised by visits from a malaria specialist at least twice a month, with the specialist also providing ongoing training and program evaluation. In addition, supervision was provided by mobile-health-clinic personnel on their visits to the villages, as well as by visiting sanitarians involved in environmental health projects. The result of all this effort was that the incidence of severe and complicated malaria declined dramatically in villages participating in the program (relative to non-participating villages). This program is a shining example of what can be done elsewhere in Malaysia, not the least being in Orang Asli communities.

As a hopeful finale to recent events, in May, 1997, three months after the Seboi tragedy, the Malaysian health ministry announced it would "re-examine" the anti-malarial program for Orang Asli and indeed review the entire health-care system for them. The purpose of this review is "to ensure that all procedures are safe" and to improve the system (The Sun, 31 May, 1997). The report on this review "will detail what the current system is like, what the defects are, and how we can improve on it," the health minister announced (New Straits Times, 31 May, 1997). The minister also said a board of inquiry had been set up on the Seboi case and disciplinary action was to be taken on those responsible. A non-governmental observer thereafter was quoted as saying that the Orang Asli community, or its association leaders, must be involved in conducting this health-care review, both to ensure that health-care workers for Orang Asli are competent and that they treat Orang Asli with courtesy and respect (The Sun, 2 June, 1997).

ENDNOTES

CHAPTER 1

Note 1: Polunin (1953) quoted a Kelantan government report for 1933 that mentioned three Temiar being trained at Kota Bahru for a week on methods for treating skin diseases, malaria, etc., and being given medical stocks to use in their villages.

On yaws, Williams-Hunt (1952, p. 35) remarked that it was "an unpleasant disease not unlike syphilis in its course" and that it was widespread, such that "there is hardly an Aboriginal community which has not had at least one case in the past few years." Yaws is a non-venereal disease of hot, wet climates, spread through skin lesions. These ulcerate, slowly affecting muscle and then bone, but skipping the nervous system. Yaws is easily curable by antibiotics.

On scabies, Williams-Hunt told Pamela Gouldsbury (1960, p.38) of a "miracle cure," a soap called Tetmosal:

> "...it was portable, and no one was likely to drink it or eat it by mistake...a cake of soap could be left with the patient and there was no further treatment required except a bath and good soaping twice a day and a good washing with it of any garments the patient might possess. In about four...days the scabies had completely gone."

Scabies is caused by a mite and is similar to mange in dogs. Among 312 Orang Asli patients and their relatives at Gombak Hospital recently, 1% had clinical signs of scabies (Normaznah et al., 1996).

Note 2: The Orang Asli speak languages belonging either to the Austroasiatic or Austronesian family. The Aslian languages spoken by Orang Asli groups are in the Mon-Khmer division of Austroasiatic.

That is, Aslian languages are related to many languages in the Burma-Thailand-Cambodia-Vietnam sector. Aslian speakers live in the central and northern parts of West Malaysia. Austronesian-speaking Orang Asli live in the central and southern parts; although most speak dialects of Malay now, they may have spoken Aslian languages in the past. Aslian languages have been in West Malaysia much longer than Malay has (Benjamin, 1989).

As Benjamin noted (1989, pp. 13-14):

> "Languages...are not the same thing as 'societies,' 'cultures,' or populations. This makes it futile to try and [fit] all data on the Orang Asli onto a unidimensional ethnological classification...If...you happen to be interested in the biology of Orang Asli populations, you would be ill-advised to accept the conventional classification without closer inspection of what it implies."

Benjamin's point is well taken, especially in terms of outmoded speculations about Orang Asli prehistory versus genetic evidence about their relationships – topics I will address in this review.

Note 3: Number 1 (of the 10 listed differences for the Orang Asli): The finding that the Orang Asli's tuberculosis rate was twice the national average prompted Bolton and Snelling (1975) to write, "A vigorous attempt is being made to control tuberculosis in the Orang Asli," by which they meant that a campaign was in progress to inoculate the Orang Asli community with the well-known BCG vaccine. The authors noted that tuberculosis was commonest in older Orang Asli, who often "caught" new TB infections. On a global scale, TB is the top killer of adults, according to the World Health Organization (Colwell, 1996).

Bolton and Snelling went on, "In a few years time it will be easier to judge whether these [anti-TB] efforts have been successful..." Dentan (1988), citing these authors, regarded this campaign as effective. Nevertheless, the protective effect of BCG has ranged from 0% to 80% around the world, for unknown reasons (Strickland, 1991). Notably, Lokman and Baharuddin (1996) briefly reported that among the 89 tubercular Orang Asli children they had studied, 80% had received a BCG vaccination and only 20% had not.

Although no follow-up studies have been published on this anti-TB effort for the Orang Asli community, Hamid (1990) reported that

the number of tubercular Orang Asli admitted to Gombak Hospital increased from 61 to 86 per year between 1981 and 1985. This compares with an average of 61 cases per year for the period 1951 to 1971. Similarly, Bolton (1973) suggested 78 cases per year for 1963 to 1973. At first glance, the TB rate did not change much from 1951 to 1985. But this conclusion is probably unjustified since the Orang Asli population increased markedly in the 1970's and 1980's. (The 1949 census totaled almost 53,000 people. By 1988 there were about 69,000.) The average life expectancy of Orang Asli has also slowly increased.

In any event, TB remains a major problem for the Orang Asli, despite the fact that relatively inexpensive therapies exist. Some indication of this problem can be glimpsed from the following: In 1986 the number of TB cases detected in West Malaysia was 3945 (Lim, 1993). Based on the 86 Orang Asli cases reported in 1985, they had 2.1% of all West-Malaysia cases at that time – far more than their proportion in the population might lead one to expect.

Moreover, according to Veeman (1986/87), among the 450 Orang Asli employed by the government health services for the Orang Asli community – presumably a select group – 70 were tubercular (15.5%). And for 1995, Fadzillah Kamaludin (1997) found 200 registered tuberculosis cases among the Orang Asli, as well as far fewer Orang Asli children than others who had received the anti-tubercular BCG vaccine (58.7% versus 99.6%). Finally, according to one report (New Straits Times, 12 May, 1997), in Perak alone TB cases were three times higher in 1995 for Orang Asli children than for others (ages 1 to 14 years).

Number 2: In 1977, Thomas and Dissanaike showed that 89% of a general Orang Asli sample had had the falciparum form of malaria at some time in the past. That is, 89% had antibodies against this malaria parasite, the frequency rising from 71% for young children to 94% by age 20. (Other forms of malaria also occur in Orang Asli.) These falciparum frequencies are high by any standard, much higher than for other Malaysian groups (see, for example, Mak et al., 1987). For the years 1993-1996, Orang Asli malaria cases were over 70% of all such cases in West Malaysia (Roslan Ismail, 1997).

While it might be true that over 50% of malarial cases in Malaysia occur in people 14 years of age or older and that 70% of the cases occur in males, as asserted by Arasu (1972), this is certainly *not* the

case for the Orang Asli. Orang Asli children are much more susceptible to malaria than are adults, and both sexes seem equally vulnerable.

Number 3: According to Mak (1994), for the years 1991 and 1992 the Orang Asli averaged 48% of all malaria infections recorded in West Malaysia, even though they accounted for less than 1% of the population. And since Orang Asli bouts of fever are relatively unrecorded, their proportion of malarial infection was undoubtedly higher than the official 48% figure.

The 48% figure is higher than that for Pahang for the first half of 1992: 38% of recorded cases there were in Orang Asli (486/1282), according to the news media (Berita Harian, 8 Sept., 1992).

Numbers 4 and 5: Crude death rates were well discussed by Gomes (1982, 1990) and will be reviewed in Chapter 2. Ng et al. (1987) gave further information. Few general data are available on the life expectancy at birth for Orang Asli; for Semai women in Pahang, this value was 35 years in 1987, amounting to 72% of the national average (Fix, 1989b; Chee, 1995). However, using data in Santos et al., 1985, the life expectancy of 35 years was only 52% of the all-Malaysian 1982 life expectancy at birth (67 years).

Number 6: This information comes from a statment by the Malaysian Minister of Health (Sunday Star, 29 Sept., 1996). The proportion of births at home in West Malaysia is not known exactly but is likely to be far higher for Orang Asli than for others.

Some related historical information was provided by Fix (1977): in 1969 the female crude death rate in Malaysia was 0.8% per year, but for Pahang Semai females the rate was 3.5%. In comparison, for the year 1987 Wong (1991b) reported that the maternal mortality rate for remote rural areas of West Malaysia was 0.2% overall, versus 0.01% for the capitol, Kuala Lumpur.

Number 7: Nutrition is discussed in Chapter 8. To cite two studies here, Chan et al. (1974) found no obesity in Temiar males, and Osman and Zaleha (1995) found Post Lanai and Post Betau Semai women to have three times more protein-energy malnutrition (PEM) than men and almost five times more PEM than rural Malay women (35.3% vs. 7.5%). Recent nutritional studies on children include those by Osman and Zaleha (1995), Khor (1988), and Ismail et al. (1988).

Number 8: Dengue is an endemic disease, not well studied in Orang Asli. The cited data are from Rudnick et al. (1986).

Number 9: The cited data are from Fadzillah Kamaludin (1997).

Number 10: For examples of cultural factors versus malaria, see Chapter 7 and also Chapters 8 and 9.

Number 11: See also Chapter 9 and the postscript.

Number 12: See Chapters 5 and 7 for various details.

Note 4: The 27 "exotic" diseases in Southeast Asia:

angiostrongyliasis	giardiasis	rabies
anthrax	gnathostomiasis	schistosomiasis
ascariasis	hookworm infection	scrub typhus
capillariasis	Japanese encephalitis	shigellosis
cholera	leprosy	strongyloidiasis
clonorchiasis	malaria	taeniasis
dengue	melioidosis	trichinosis
E. coli enteritis	opisthorchiasis	trichuriasis
fasciolopsiasis	paragonimiasis	tuberculosis

Most readers will be familiar with the major diseases in this list. More obscure diseases on which pertinent information exists will be briefly described.

Note 5: The head of Gombak Hospital discussed a strong anti-malarial program for the Orang Asli that had been started in the 1960's, with periodic DDT spraying of houses, administration of drugs, patient care, and blood surveys for parasites (Bolton, 1972a). In areas where the program was faithfully carried out, malaria survey rates declined dramatically – e.g., from 66% in 1960 to 0% in 1967 in the Temuan village of Bukit Manchong, in Selangor. But in Selangor overall, the survey rate was 12% in 1967, indicating persistent problems. And other surveys (Baer et al., 1976) indicated that the average rate for Temuan villages was 29% in 1972, with a range of 16% to 43%. Moreover, as late as 1986 among Temiar at Legap, Perak, the rate was 64% for children 0-9 years of age, with three-fourths of the infections being of the falciparum type. The rate in older Temiar was also relatively high, bringing the average for all age groups to 49% (not the 46% printed in the report) (Lee et al., 1988).

These and other findings raised questions in the minds of some observers about the long-term effectiveness of such an anti-malarial campaign, one based largely on the work of outsiders rather than on

village-resident, Orang Asli health initiatives. While Carey stated in 1976 that 120 Orang Asli were then being paid a monthly allowance to assist in the distribution of medicines in villages, including anti-malarials, this number hardly sufficed to cover the existing settlements. They numbered 217 in the state of Pahang alone and 629 for the entire country, according to the 1969 census.

Lim (1993) discussed the futility of the 1970's malaria eradication program for West Malaysia. Diggs (1992, p. 114) has pointedly remarked that while many countries have nominal malaria-control programs, "most suffer from lack of personnel with an adequate understanding of all the factors required to initiate and maintain such an effort, and all suffer from a lack of sufficient funds for the purpose."

Note 6: According to one authoritative source in Malaysia (Rajakumar et al., 1980, p. 53), "Pressure from an elite for a fully equipped coronary care unit is more effective than that for a comparably equipped health center."

There is a flip side to this neglect of Orang Asli health. Attempts to kill malaria-carrying mosquitoes resting indoors after a blood meal by spraying DDT inside houses, a practice still legal in Malaysia, kills other bugs. If no DDT is sprayed, Orang Asli roofs last longer. Palm thatched (atap) roofs deteriorate at a faster rate than usual in DDT-sprayed houses because the insecticide kills homely insect-eating insects, the ones that feed on bugs that eat atap or hide inside roof beams – while the bugs inside the beams are untouched by the DDT (Huehne et al., 1966; Polunin, 1986).

Also, if DDT is not sprayed, it cannot accumulate in soils and the food chain and concentrate in breast milk. Small, malnourished children are qute vulnerable to the toxic effects of pesticides such as DDT, and malnutrition is a large problem among the Orang Asli.

Note 7: Little enough of Malaysian prehistory is known (see, e.g., Endicott and Bellwood, 1991; Baer, 1995). Benjamin (1989) maintained that archeological studies do link today's Orang Asli with a definite prehistoric past on the Malay Peninsula. Future interdisciplinary studies in this area are possible, such as combining new techniques for studying ancient human DNA (from fossil bones or teeth) with studies on the DNA of living persons.

Note 8: "Now nowhere in the world can be found a group so racially pure that all its members look alike...There is no such thing as a typical Negrito, a typical Senoi or a typical Jakun any more than one can say a fair haired Englishman is more typical than a dark haired" (Williams-Hunt, 1952, p. 15).

This reasoned statement by Peter Williams-Hunt has two flaws. One is his off-hand reference to "race." (Are the Minangkabau a race? The Balinese? Laotians? Kurds? You tell me.) The other flaw is the related notion that human groups, or "races," were once pure, so that the intra-group variation seen today is the result of mixing between groups. [This speculation seems to misinform the writing of Carey (1976) at several points.] No population biologist subscribes to this purity notion because all of many findings show just the opposite. I mean, all human groups – and also zebra herds and wolf packs – are genetically diverse.

Note 9: Benjamin (1985) considered Jehai and Batek to "have become more nomadic in recent years than they were formerly" (p. 244), with their "secondarily intensified nomadism...if such it be...very likely to have developed as an abreaction from the Neolithic sedentization of their proto-Senoi neighbors" (p. 262).

CHAPTER 2

Note 10: Bulbeck (1982) studied skeletal remains from Gua Cha, West Malaysia, that date to 3,000 to 10,000 years ago. He estimated the adults of these ancient people to be taller than Semai or northern Orang Asli adults today. If the northerners were eternally "short," then who were these ancient people?

Note 11: Schebesta (1973, p. 263) quoted a description of a northern group seen by King Chulalongkorn of Siam:

> "They are of middle height, have broad noses, their lips are not too thick; their hair is frizzy...The complexion is not so dark as that of the Siamese. The people are powerfully built."

Note 12: In "unmaking the myth" of Semai being economically backward and "untouched," Gomes (1989) pointed out that they have been trading forest products for centuries and that a number of Semai

villages in Perak have forsaken farm work (swiddening) gradually over several decades. They now produce commodities for the outside market.

Note 13: Filariasis is endemic in Malaysia. It is a non-fatal but chronic infection of the lymph system by filarial worms (nematodes). The parasite is carried by mosquitos, just as in the case of malaria. If not treated, filariasis can cause recurrent inflammation, fevers, and weakness over many years, occasionally leading to elephantiasis or other sequalae. The report by Kinzie et al. (1966) that 10% of Jehai had elephantiasis supports Williams-Hunt's earlier remark (1952) that filariasis, as evidenced by lower-limb elephantiasis, has not been rare among the Orang Asli.

Ramachandran and Dondero (1976) cautioned that results of different filariasis surveys could not realistically be compared because sampled blood volumes were variable and often small. The larger the blood sample studied, the greater the chance of detecting parasites, it turned out. So filariasis probably was more prevalent among Orang Asli up to that time than previously thought. Still, the authors maintained that chemotherapy had for some years largely controlled clinical cases of filariasis throughout West Malaysia.

Nevertheless, in the 1960's Onyah showed that the Orang Asli had substantial filariasis. Temiar had 16% showing microfilarial parasitemia. Semai had 10%, Semelai had 20%, Temuan had 9%, and Jakun had 15%. Besisi had 7%, while Orang Kuala had none. Inland areas had more filariasis than did coastal areas.

The reduction of filariasis for some Orang Asli groups since the 1950's amd 1960's has been substantial. By and large, this appears to be a notable success story for Orang Asli health. As shown in Table 2, groups with previously high rates experienced a decline by 1978 of 60% or more. For northerners, the decline was from 33% filarial parasitemia in 1951 to 3% in 1978, a 67% drop.

The Semelai provide the best evidence for a filariasis decline because the four surveys on them probably occurred on the same general population, in the vicinity of Post Iskandar near Tasek Bera, Pahang.

And yet, a recent paper (Hakim et al., 1995) raises doubts about the generality of the filariasis decline. For Jehai in Banun and Temiar/ Lanoh in Dala, Perak, microfilaremia rates were 25% and 23%, respectively. (Numbers of persons surveyed were not mentioned.)

Table 2
Microfilarial surveys on inland groups

	N	No. positive	% positive	Reference
Northerners				
Lanoh, Perak	121	40	33	Polunin, 1951b
Jehai, Banding	12	2	17	Kinzie et al., 1966
miscellaneous	27	3	11	Mak, 1978
Temiar				
Ulu Plus, Perak	61	13	21	Polunin, 1951b
Ulu Kelantan	239	38	16	Wharton et al., 1963
4 sites, Kelantan & Pk.	506	79	16	Onyah, 1967
miscellaneous	300	10	3	Mak, 1978
Semai				
Ulu Jelai, Pahang	33	21	64	Polunin, 1951b
7 sites, Pahang & Perak	742	76	10	Onyah, 1967
miscellaneous	866	64	7	Mak, 1978
Semaq Beri				
miscellaneous	39	4	10	Mak, 1978
Semelai				
Post Iskandar, Pahang	186	28	15	Wharton et al., 1963
Post Iskandar, Pahang	74	15	20	Onyah, 1967
Post Iskandar, Pahang	54	6	11	Else et al., 1976
miscellaneous	73	3	4	Mak, 1978
Temuan				
Ulu Langat, Selangor	18	0	0	Polunin, 1951b
12 sites, Sel. & Pahang	579	112	19	Wharton et al., 1963
Bukit Lanjan, Selangor	167	29	17	Ramachandran et al.,1964
Bukit Cheeding, Sel.	53	9	17	Kinzie et al., 1966
11 sites	317	29	9	Onyah, 1967
miscellaneous	601	28	5	Mak, 1978

Abbreviations for the first column: Pk., Perak; Sel., Selangor.

Following a 19-month study of treatment with diethylcarbamazine (DEC), the parasitemia rates were 7% for Banun and 10% for Dala. In most parts of the world such rates would be considered unacceptably high. For comparison, the overall rate for Malaysia was 0.35% in 1994, according to this paper. Another consideration is that Mak (1992) found that non-DEC drugs, having milder side effects, appear to be more effective or convenient in treating filariasis.

While the reported declines in filariasis may be continuing, the cause of the declines is unclear. Possible causes include DDT spraying,

mass drug administration, use of impregnated bed nets, chemotherapy of clinical cases, swamp drainage or oiling (where applicable), or some unknown factor.

Note 14: Hookworm infection and its consequent anemia are underdiagnosed. According to an authoritative review (Pawlowski et al., 1991, p. 29), hookworm anemia is often ignored as a basic factor in "high maternal morbidity and mortality, apathy and poor health in children, and easy fatiguability and impaired work capacity in adults. Its effects are insidious."

However, deciding the relative importance of hookworm, malaria, and a poor diet in the case of a nursing mother who is anemic or malnourished may be difficult (Polunin, 1967).

Preventive measures against hookworm took a leap forward as early as the 1920's in Malaya when a Chinese businessman agreed to manufacture cheap shoes at the urging of the Rural Sanitation Campaign (Parmer, 1989). The accompanying propaganda touted: "Better shoes than shrouds."

Note 15: By the 1970's Carey was aware that, at least for northerners, government-run "resettlement schemes have had only a very limited success" (1976, p. 65). He cited adverse cultural effects of the schemes, such as thwarting the preference for a traditionally mobile-forager lifestyle. But as Gomes has shown, there are also adverse biological effects. Gomes' data suggest that a high birth rate can be linked to a high death rate for reproductive-age women. Presumably the surviving women also have reproduction-related illnesses short of dying, although such information is unavailable. An important point is that maternal mortality and morbidity are not only hard on the female population, but hard on families and the community as a whole. Children are the immediate victims, being deprived thereby of emotional support, as well as maternal "medical" services such as monitoring cleanliness and providing regular meals. Tan (1986) gave a general discussion of this issue.

Some of Gomes' data can be compared to those of Schebesta (see Evans, 1937, p. 16) and of Polunin (1953). While Gomes estimated Jehai completed fertility to be 4.5 live-born children, Schebesta estimated it to be 4.3 for northerners prior to World War II. While Gomes estimated the fraction of live-born Jehai offspring who had

died to be 36% in 1988, Polunin estimated the comparable Lanoh fraction to be 48% in the 1950's and Schebesta estimated the fraction for northerners to be 39% even earlier.

Note 16: Carey (1976) mentioned a fall in the Orang Asli infant mortality rate (IMR) throughout his text, without citing evidence. He seemed to consider that population growth required a fall in the IMR. But this does not "compute." As Gomes' work showed, the fertility rate may increase, thereby offsetting high childhood mortality.

CHAPTER 3

Note 17: Whereas Khor found a maximum of 17% childhood malaria in the 1980's, Andre et al. (1972) reported a 51% rate for Batang Padang school children in 1969. For another Batang Padang sample, ages not specified, Bolton (1972a) reported a 55% rate in 1970, despite an anti-malarial program then in progress.

Note 18: As Table 3 shows, Semai health has remained problematical for a long time.

Table 3
Time track of some Semai health problems (percent with each condition)

	Filariasis	Malaria	Hookworm	Ascaris	Trichuris	Low Weight
Nevin, 1937	10	19	26	70	14	nd
Polunin, 1953	64	18	86	7	2	45
Dunn, 1972	nd	nd	66	40	51	nd
Khor, 1985	nd	7-17	14	57	49	58*
Ismail et al., 1980	nd	nd	nd	nd	nd	27-52*
Greer et al., 1989	nd	nd	84	79	93	nd
Massita, 1992	nd	nd	nd	nd	nd	33*
Mak, 1992	<7	47	nd	nd	nd	nd
Osman & Zaleha, 1995	nd	nd	5	21	24	80*

Parasite data are for population samples lacking prophylactic measures or for which none were reported. Data on children only are starred (*). nd = no data.

According to Gouldsbury (1960, p. 25), for the Semai and others who build stilt houses on hillsides:

> "[they]...can throw out the remains of a meal, spit through the split bamboo flooring, pot their children at the side of the house; and lo the Malayan rains come down almost every day in torrents and wash it all away, leaving the ground round the houses sweet and clean. With the houses well away from the river or stream, the refuse never gets washed that far and the water supply remains unpolluted."

However, as Table 3 shows, intestinal worms are not eliminated by this common-sense siting of houses.

Between 1985 and 1992 (Mak, 1992; 1994) Semai at Betau, Pahang, had malaria parasitemia prevalence rates that changed as follows: for 1985, 47%; for 1986, 37%; for 1987, 26% and 32%; for 1988, 26% and 15%; for 1989, 21%; for 1991, 23%. Following determination of the 47% rate in 1985, cases were routinely treated, and DDT was sprayed in houses at 6-month intervals. In 1989, permethrin-impregnated bed nets were provided for one group while a control group received ordinary bed nets. In 1991, DDT spraying was stopped. Data for 1991-1992 on the bednet test (Mak, 1994) showed that the overall parasite rate declined from 23% in March, 1991, to 7% in August, 1992. The final rates for treated and untreated nets was the same. Despite this result, in 1997 the government was still using treated nets for Orang Asli, even though resistance to permethrin can and does occur in mosquitos (Rohani et al., 1995).

Note 19: Traditionally the Semai studied by Fix did not practice contraception, abortion, or infanticide.

Note 20: In a sample of 22 men over 50 years of age, 6 (27%) had had no children (Fix, 1982a), largely because finding and keeping a wife was difficult, given the relative scarcity of women.

Note 21: Among Pahang (Satak) Semai, 29 women died between 1950 and 1969. This was 13% of those aged 15 to 44 years. Among the 29, 45% died in childbirth (Fix, 1977). Infections following delivery were common and reported to be much feared.

Note 22: This discussion of genetic surveys on the Semai and other groups is based on Saha et al., 1995; Cavalli-Sforza et al., 1994; Sofro, 1982; Omoto et al., 1993; and Baer, 1998.

Note 23: A Semai village of 272 in 1969 had a turnover of 10% of its population in 9 years, as the result of in- and out-migration (Fix, 1982a).

Note 24: For the village with 35% hemoglobin E, the high level was largely due to a kin-group fusion event some 40 years earlier. Without that event the frequency would have been 24% (Fix, 1982a).

CHAPTER 4

Note 25: The only other report on scrub typhus in the Orang Asli, by Cadigan et al. (1972), studied "deep jungle" (remote), "jungle fringe," and peri-urban categories. These categories were ethnically heterogeneous. All three categories contained Semai (Satak, Woh, and Denak sites). The authors found that adults (>19 yrs.) showed more evidence of exposure to scrub typhus serologically than did children. They also found that remote areas had more exposure than fringe or peri-urban ones but offered no explanation for this difference. Curiously, their reported high rate (73%) in adults in remote areas was similar to that found later for Bukit Lanjan Temuan, who are not "remote."

Note 26: For 220 Semaq Beri, 40% were 0 to 14 years of age and 21% were 40 or older. For this latter group, the sex ratio was 30 males to 16 females, or 1.9, providing one of several examples of poor survivorship in post-reproductive women. However, it is likely that many men (ages 15-49) were not censused, perhaps being absent from villages for wage-labor work.

CHAPTER 5

Note 27: Carey (1976, p. 5) distanced himself from the term "aborigine," although he used the phrase "aboriginal tribes" in the subtitle to his book.

Note 28: In Malaysia, dengue viruses cause various acute conditions. Dengue is generally a disease of fevers, chills, headache, and rash,

but it may proceed to hemorrhages, shock, and sudden death. Dengue is endemic in Malaysia and is carred by Aedes mosquitos from human to human. The virus is also found in forest monkeys, but it is still unclear if they are reservoir hosts for human infection.

Note 29: Kinzie et al. (1966) reported the opposite finding: a higher pre-adult death rate among Jehai (54%) than among Temuan (26%); data supporting this conclusion were not provided.

Note 30: Polunin (1953) also noted "half-Chinese" children in some areas: 2 in a group of 174 Semai (plus 1 all-Chinese girl) and "4 or 5" half-Chinese among 149 Seletar.

CHAPTER 6

Note 31: According to Soong (1972), not all cultural practices of Temuan are positive in terms of health. This author stressed that although Temuan utilize "modern medical care," their "erroneous beliefs lead to undesirable practices" that foster ill health.

Note 32: Nah's grandfather appears in two photos and his house appears in one photo in a paper published by Self (1970). This paper was based on a flying visit by an American zoologist interested in pentastomiasis. Unfortunately, the paper contains some biocultural misinformation.

Note 33: Razha Rashid (1995b) reported five cases of fever for several days and then death for two children, two women, and one man, all during one fruit season in a Kintak village. A number of endemic diseases are possible candidates for such fatalities: dengue, Japanese encephalitis, scrub typhus, and even malaria – but often information on such tragedies is too scanty to hazard a disease label.

CHAPTER 7

Note 34: Many malarial data are questionable. For example, authors may not specify if their data refer to clinical cases or simply to parasitemia detected during field surveys. For the year 1995, Roslan Ismail (1997) reported 8106 Orang Asli malaria "cases" in one table, more than reported for all of West Malaysia in another table (7752).

Note 35: Chloroquine was introduced in 1964, but its prophylactic administration to Orang Asli was unsystematic and its effects small (Lie-Injo, 1969). In the 1970's, for example, in one peri-urban Temuan settlement in Negri Sembilan, new cases of parasitemia were occurring at the rate of 6% per month for falciparum and 5.7% per month for vivax malaria (Lewis et al., 1973). More recently, other drugs – plus insecticide-impregnated bednets – have been provided, but not comprehensively. Gordon and co-workers (1991) commented that Temiar at Legap, Perak, "do not use malarial prophylaxis," but when recognized as being ill, they are treated at local health stations. These comments leave open the question of whether anti-malarial drugs are made available to Legap Temiar for prophylactic purposes.

Lambros and co-workers (1989, p. 6) also considered this situation. They wrote that anti-malarial prophylaxis "...is infrequent if not nonexistent. Fansidar and chloroquine have not been freely available to the Orang Asli. Medication is only available when they become so sick they must leave their forest dwelling and travel long distances to seek medical care at a clinic or hospital." This helps explain why malarial parasites in some Orang Asli areas are (or recently were) still quite sensitive to chloroquine, as the Lambros team discovered, while in other areas, where drugs are more readily available, plasmodia (malaria parasites) tend to be genetically resistant to chloroquine.

In a 1992 review on malaria in Malaysia, Lim wrote that mass drug therapy is used "when necessary," without defining this criterion. Rather, he emphasized the treatment of cases as they are found to occur by government workers. But it is not clear how often "home visits are made by anti-malaria workers" to detect cases of malaria in Orang Asli villages. Also, a reader of Lim's review might get the impression that mass blood surveys are carried out "regularly" among Orang Asli living "in the interior." Not only does other evidence suggest this is a rosy statement, but it begs the question of the high malarial rates known to exist in more accessible Orang Asli, such as the Temuan.

Note 36: With the advent of malaria studies early in the 20th century in Malaya, entomologists became adept at finding and identifying the mosquito vectors of the disease. Since mosquitos also transmit filariasis, that disease also became a popular target of study. Hence, far more historical data are available on filariasis than on, for example,

scrub typhus or dysentery. Filariasis is much less of a health problem for Orang Asli than malaria is, although both diseases are endemic in the region.

Note 37: Unfortunately, some malarial data are not useful for this review. Collins et al. (1968), for example, only provided pre-drug treatment data for Perak and Kelantan Orang Asli together, combining Temiar, Semai, and perhaps other ethnic groups. In tables giving data on their post-drug surveys they identified the areas studied but not the ethnic group surveyed in them. And samples from most areas were, in any case, too small to provide reliable information about parasite rates. Finally, they failed to provide infection data on children vs. adults. In these respects, this paper is a good example of how *not* to study malaria epidemiologically.

I have excluded data from Table 7 that were not collected in villages. While Mak (1978) analyzed many groups at Gombak Hospital, the population profile he presented shows a deficit of older children, an age cohort highly vulnerable to parasitemia that is well represented in village surveys.

Note 38: Contrarily, a few authors have held that iron-deficiency anemia may protect against malaria, given that the parasite requires iron for its own growth. But one cannot look at a particular condition by itself: many facets of health need to be considered together. Indeed, anemia lowers resistance to many infections.

Note 39: Nagel (1990) discussed possible effects on plasmodia of red cells that contain Hb E, Hb CoSp, high levels of fetal hemoglobin (Hb F), thalassemic phenotypes, or that are ovalocytic; he did not discuss G6PD deficiency. Livingstone (1983, 1985) discussed a variety of evidence, pro and con, on "malaria-related" alleles, including G6PD deficiency.

Two genetic variants providing resistance to malaria in Africa are not found in the Orang Asli. They are sickle-cell hemoglobin (Hb S) and Duffy-negative blood group (Fy). Data presented in Lewis et al., 1988, can be used to calculate the frequencies of the two Duffy alleles that do exist in the Orang Asli: Fy-a and Fy-b. For Semai (N=120), the Fy-a allele frequency is 0.963. For Temiar (N=70), it is 0.971; for Semelai (N=31), it is 0.935; for Jakun (N=32), it is 0.953; and for Temuan (N=37), it is 0.932. Earlier data in Polunin and Sneath (1953) suggested an Fy-a allele frequency of 0.553 for northern Orang Asli (N=35) and of 0.689 for Temuan (N=31; called "Orang Darat" by the authors). Despite the low reliability of genetic findings on small samples, it is clear that Fy-a is common among Orang Asli and Fy-b is rare (4%, according to the work of the Lewis team).

Note 40: Among hemoglobin variants, Hb E is centered in mainland Southeast Asia, It probably diffused throughout its range by means of kin-group migration (Fix, 1978). It is due to a simple (base) change in the DNA that determines a change in one amino acid in the beta chain of the protein. E chain synthesis is low, but EE homozygotes are at most mildly anemic. In vitro, EE cells strongly hinder the growth of falciparum parasites, but cells with one dose of E (E heterozygotes) only weakly affect them. Other evidence also supports an anti-malarial effect of the EE condition. It can also interfere with pregnancy (Lie-Injo, 1964).

Hb CoSp is a rarer Hb variant, also localized in Southeast Asia. It produces, by a simple mutational change, an elongated alpha chain of the hemoglobin protein. In vitro, it impairs the growth of falciparum parasites (Yuthavong et al., 1988). Although it is probably underreported in laboratory studies because of technical problems in its detection, it may have little selective impact on survival in malarious areas because of its rarity, despite its anemia-like effects (Hsia et al., 1989; Higgs et al., 1989). One Jakun family had three mildly anemic children homozygous for Hb CoSp (Lie-Injo, 1972).

While thalassemias occur widely in Southeast Asia (Wasi, 1983), and are known to have anti-malarial effects, they have not been well surveyed among Orang Asli. Lie-Injo (1976) mentioned that they are rare in Orang Asli, without providing quantitative data. I therefore exclude thalassemias from discussion, for lack of relevant information.

Hb F (fetal hemoglobin) deserves mention here because it retards the growth of falciparum parasites. This turns out to benefit, indirectly, some infants and young children infected with malaria (discussed in Nagel, 1990).

G6PD-deficient males (who have one dose of the variant since they have only one X chromosome) have low levels of malarial parasitemia, as do heterozygotic females (who have one dose of the

variant, on one of their two X chromosomes) (Baer et al., 1976; Baer, 1998) Many G6PD- variants exist throughout the world, including several that are quite common in Southeast Asia. But the kinds and proportions of variants in Orang Asli have not been analyzed. A large amount of raw data on G6PD deficiency in Malaysian newborns exists in hospital records and could profitably be studied in terms of population frequencies and resistance to malaria.

Many G6PD- individuals are prone to develop anemia as neonates (resulting in jaundice), after infections (e.g., hepatitis and pneumonia), or after ingesting certain drugs. Indeed, Lie-Injo and Chin (1964) provided data that, by my analysis, indicate Orang Asli male hospital patients were more likely to be G6PD- than were healthy Orang Asli males. Planners of malarial control measures need to be aware of the existence of G6PD- in Orang Asli populations. An important complication in malaria chemotherapy is drug-induced hemolytic anemia in G6PD- individuals, particularly anemia caused by primaquine (as Khoo, 1981, reported for Sabah). And chloroquine – seemingly not a major problem for Orang Asli (Lewis and Ponnampalam, 1975; Lambros et al., 1989) – has been flagged as a problem elsewhere in Southeast Asia. The "double trouble" with G6PD- is that malaria itself leads to hemolytic episodes when a wave of parasites burst out of red cells, so that when a G6PD- patient is treated with primaquine even more hemolysis occurs. Thus while the malaria may be stopped, the patient's anemic condition worsens.

In 1970 Sandosham warned that the then-currently planned mass treatment of Orang Asli with primaquine as an antimalarial drug was dangerous and illconceived, given their high frequency of G6PD-. Luckily, other drug treatments were then in vogue and new drugs soon became available. But Orang Asli have been subjected to widespread administration of primaquine since that time. In one fairly recent case, Gordon and co-workers (1991) administered primaquine to 275 Temiar at Legap, Perak. Since this group was not studied genetically, the researchers were presumably unaware of the G6PD status of individual Temiar and hence of the possible toxicity of primaquine for them. No mention was made in the authors' report about the health effects of drug administration on the volunteers.

Mak (1992), however, discussed this question in detail and reported that no antimalarial drug used in Malaysia is contraindicated for G6PD- individuals, if given at the recommended dosage. He recommended

that if jaundice or hemolysis did occur, delayed and lower doses over longer periods were to be used.

Ovalocytosis, once called elliptocytosis, refers to oval red cells. The oval shape results from a loss of a small part of a protein located in the outer membrane of red cells (Anstee et al., 1995; Jarolim et al., 1991). The OV heterozygotic condition does not harm red cell viability, but it is abnormal in biochemical terms (Nagel, 1990; Schofield et al., 1992). The OV membrane defect causes red-cell rigidity. This change may decrease the attachment of plasmodia to the red cell, decrease its invasibility, or interfere in some other way with parasite survival (Jarolim et al., 1991; Schofield et al., 1992). In addition, OV homozygosity is lethal before birth (Liu et al., 1994).

The OV allele is found mainly in the lower half of Southeast Asia east to New Guinea (Livingstone, 1985; Sofro, 1986). It is less common at high, than at low, altitude both in Malaysia and New Guinea (Baer et al., 1976 on Temuan vs. Semai; Serjeantson et al., 1977), a difference that may reflect a selective disadvantage at high altitude due to poor oxygen transport by OV red cells (Schofield et al., 1992). In Temuan and Semai, the OV allele frequency is higher in adults than in children (Baer et al., 1976; Foo et al., 1992). Four Semai were found who were homozygous for Hb E and heterozygous for OV; none were anemic.

OV heterozygotes are resistant to all three types of malarial parasites found in Malaysia (Nagel, 1990; Serjeantson et al., 1977). Significantly, OV is the only gene known with such broad resistance to malaria. With the "booster" of acquired immunity, OV heterozygotes rarely have clinical malaria as adults. In childhood they are not prone to cerebral malaria (Genton et al., 1995). In addition, OV seems to provide a selective advantage in terms of female fertility and longevity (Baer, 1988).

Orang Asli groups that have experienced high malaria pressure in the past tend to have high levels of malaria-resistant genetic traits. The net effect of these traits in the Orang Asli is to provide them with strong resistance to life-threatening malarial attacks. Being a Hb EE homozygote, a G6PD- male or G6PD- heterozygous female, an OV heterozygote, or a Hb CoSp heterozygote has definite advantages in a malarious world.

Note 41: A classic example of a trickling of genes between populations

that has produced a marked effect is the case of Afro-Americans having about 30% of their genes being of European origin, due to interbreeding over the past several centuries.

Note 42: The Temuan say there are six kinds of Malays, all from elsewhere: three groups from Sumatra, one from southern Borneo, one from Banka Island in the Malacca Straits, plus Javanese.

Note 43: In the past Orang Asli have outbred to neighboring groups as the result of Malay slave raiding of women and children, from about 1860 to 1920 (Endicott and Bellwood, 1991), or, more recently, by an Orang Asli woman "marrying out." In addition, Williams-Hunt (1952, p. 44) noted that about 3-4% of Orang Asli of various ethnic groups were living in what he called "semi-slavery" adjacent to Malay villages in the 1950's. That situation may also have produced some outbreeding.

This exodus of genes, including malaria-resistant genes, has probably grown in recent decades. Yet the integrity of the various Orang Asli gene pools remains largely intact, since few outsiders marry in. The Besisi might be an exception, as they have been reported to "frequently adopt Chinese and Indian children, usually girls, and these adopt...the culture completely" (Carey, 1976, p. 169).

The genetic exclusiveness of Orang Asli aids in the integrity of their cultural "pools," tending to keep them viable, despite outside social, economic, and political factors. But the most critical cultural variable, language, may not withstand the current internal colonization of Orang Asli territorial lands by outsiders. As one observer put it, "[L]anguage continues to be the most pervasive symbol of social cohesiveness [for the Orang Asli], reinforcing other commonly shared sentiments" (Karim, 1995, p. 23), such as those about origins, history, land, and religion. Yet as long as Orang Asli groups are able to keep their genetic-cultural distinctiveness, they will maintain two important defenses against malaria: a high frequency of malaria-resistant traits and cultural characteristics (such as smokey hearth fires) that buffer disease threats.

Note 44: Semelai sometimes drag smoldering logs from recently burned swiddens underneath their stilt houses. The log's white smoke can keep mosquitos away for a week or more (Gianno, 1990). Hunting-gathering northerners keep smokey fires going throughout the night in front of their sleeping platforms. The fires provide warmth, deter mosquitos, and attract snakes (Williams-Hunt, 1952).

CHAPTER 8

Note 45: Karen Endicott (1980) estimated that hunting provided the Batek over 45 grams of animal protein per person per day, possibly a sufficient amount. [During the same era, Kuchikura (1988) found that Semaq Beri also had a sufficient amount of animal protein.] The Batek also had about 2.4 pounds of raw vegetable foods, a slim majority of which was foraged by women and children in near-camp work parties, with the rest being procured by men. She noted wryly that while hunting was done quietly, usually by men, "...gathering can be done by large, noisy groups, since tubers cannot be scared off" (p. 636). Among all foods eaten, meat, fish, fruit, and yams figured prominently, with palm heart, mushrooms, legunes, and other items being secondary. Interestingly, Batek do not hunt pigs, deer, tapir, or snakes (Endicott and Bellwood, 1991). And Kuchikura (1988) noted that Semaq Beri also do not hunt pigs or other large animals, having no weapons or traps for them.

In contrast to the Batek, Osman and co-workers (1992) estimated that Pangsoon Temuan over the age of 6 years averaged 57 grams of protein in their diet. (The recommended protein daily allowance is 60 grams, according to Tan, 1982.)

However, Bolton (1972b), considered that food taboos lowered protein nutrition in northern women and children. During pregnancy Jehai and Kensiu women (and their husbands) traditionally obtained animal protein only from small mammals, fish, and frogs. That is, they abstained from eating venison and pork, hoping thereby to foster a healthy pregnancy. While Batek and Mendrik pregnant women had no special food taboos, they traditionally did not eat meat or fish for one month postpartum. Furthermore, children of all these groups had more food taboos than did adults, according to Bolton, raising questions of malnutrition for them. Other observers, however, maintained that children did not have extra food taboos (Williams-Hunt, 1952; Gouldsbury, 1960).

Among the Temiar, an additional group with stringent food taboos is village midwives, usually older women (Jennings, 1995). Yet while many Orang Asli proffer a belief in avoiding tabooed foods in order

to prevent illness, the taboos are elastic. As Dentan (1988) recalled, one Semai mother commented that both she and her children were healthy, so she didn't pay much attention to taboos.

Note 46: Malaysia submitted a "Country Paper" for the FAO/WHO International Conference on Nutrition held in Rome in 1992 (Tee and Cavalli-Sforza, 1993). The paper described the free distribution of whole milk powder to malnourished children as well as to some pregnant and lactating women, at the rate of 1 kg/month. So-called "food baskets" also were given to families with malnourished children. The baskets contained rice, flour, dried anchovies, green gram (mung beans), sugar, oil, biscuits, milk powder, and multivitamin supplements, valued at about 60 Malaysian ringget/month/child (24 U. S. dollars). No information appears to be published on how many Orang Asli children were aided by these programs. However, Siti Noor (1996) reported that Jakun at Langkap, Pahang, recently were receiving such food baskets. As noted in Chapter 4, at least in this instance, they did not get the whole list of basket items: they received only 2/3 the official amounts of sugar and biscuits and got no dried beans or oil. They were not given multivitamins, only vitamin C.

Note 47: Typographical errors exist in table 1 of Kasim et al. (1987); 259 children were not underweight and 307 were. Also, males totaled 271 and females 295, according to the table – but not according to the text. In table 2, males totaled 282 and females 284, supposedly on the same 566 children as studied in table 1.

Note 48: Some local foods have high iron contents, including whole-grain millet, beans, red palm oil, betel nut, and jackfruit (Strickland, 1991, p. 939).

Note 49: Worldwide, 50% of women infected with sexually-transmitted diseases have no external symptoms (Nowak, 1995).

Note 50: In a study of non-Orang Asli Malaysian children, Li (1990) showed that those with hookworm infection were shorter, thinner, and more likely to be anemic than those who were hookworm-free, when age and other variables were controlled. Thus hookworm infection may have a longstanding effect in terms of growth retardation.

Note 51: Schebesta wrote (1973, p. 166), on Jehai at "Teledn":

> "Not all species of hubi can be eaten straight away. Many of the tubers are poisonous and the poison must first be removed...To make [the tulegn tuber (hubi kapor)] edible they burn the leaves of the gobn tree and mix the ashes with the tulegn, which is first pounded to a pulp. This dish is edible and tastes like paste. The gadogn root (hubi gadong) is...left to soak a long time in the river before it is boiled. The hakai tuber (lekeh) is also crushed, its flesh dried and then boiled. Each root demands different treatment, just as each has its own flavor."

Note 52: Lim (1993) reported that the maternal mortality rate for West Malaysia in 1989 was 0.02 per thousand, largely due to hemorraging, toxemia, or infection.

Note 53: Polunin (1967) pointed out that most of the illness load in populations like the Orang Asli results from a small number of diseases or conditions, in contrast to the situation for "sophisticated" populations where many diseases each add a small part to the total morbidity.

Note 54: Skeat and Blagden (1906, vol. 1) thought that the Temuan of Negri Sembilan "...do not appear to possess much stamina for resisting fever and other internal ailments" (p. 103) and that the "Jakun" (or others?) of Johore "...die for the most part of fever caused by the dampness and insalubrity of the places they inhabit" (p. 104).

Note 55: The only early "report" on serious venereal diseases I have found is the remark by Williams-Hunt (1952, p. 35) that they are (were) rare, although a brothel containing Orang Asli had been discovered in a town: a business enterprise run by a non-Asli. Also, one case of gonorrhea had been identified. But he had an impression that venereal diseases had already reached the "aboriginal Malays" and lowland Semai. Ong (1973b) reported only on minor venereal diseases in pregnant women. More recently, Roslan Ismail (1997) presented some remarks on HIV carriers among the Orang Asli, relevant to the question of AIDS. Among 14 carriers detected, one was a prostitute.

Dentan et al. (1997) mentioned prostitution as one of the few avenues open to Orang Asli women for unskilled "jobs," whether at a multi-ethnic "scheme" or in a city. Can a VD explosion be far behind?

Note 56: "Diseases are, the Behrang people told me, thought to be caused by spirits which come from the direction of the sea, and, in the case of epidemic disease at any rate, the idea is partially supported by reason, since smallpox especially – one of the most dreaded ailments – reaches the [Semai] through the Malays" (Evans, 1970, pp. 218-219).

Skeat and Blagden (1906, vol. 2, pp. 247-248) wrote, "Questioned as to smallpox..., they said that it was a 'Nyani gob' or Malay spirit, which was very hostile to the [Semai] while it treated the Malays kindly. The chieftain of the Sahun tribe remarked that he used to estimate his tribe at about 300 souls, but within the last year 24 had died of smallpox."

The same two authors also wrote (1906, vol. 1) that the northerners were "in mortal terror" of smallpox, from which many of their communities "greatly suffered from time to time" (p. 100). Senoi (the generic term that includes Temiar and Semai) had "the same dread of smallpox" (p. 102). For Jakun, "...smallpox was the one disease that they most dreaded, and upon the appearance of which they would flee from the district" (p. 103).

Note 57: A cholera-like disease was described in Sanskrit about 2500 years ago. The cholera pandemic that started in 1961 continues today in Asia and elsewhere, although low-tech measures for prevention are known (Colwell, 1996). In the 1960's half of the Semaq Beri in a "resettlement center" died from cholera (Morris, 1997). Cholera epidemics still occur among Orang Asli, as observed during the 1980's for the Semai in Betau, Pahang (Nicholas, 1994). Also, a cholera epidemic among the Jehai grouped at Sungai Rual, Kelantan, killed 6 people in 1993 (Nicholas, 1993).

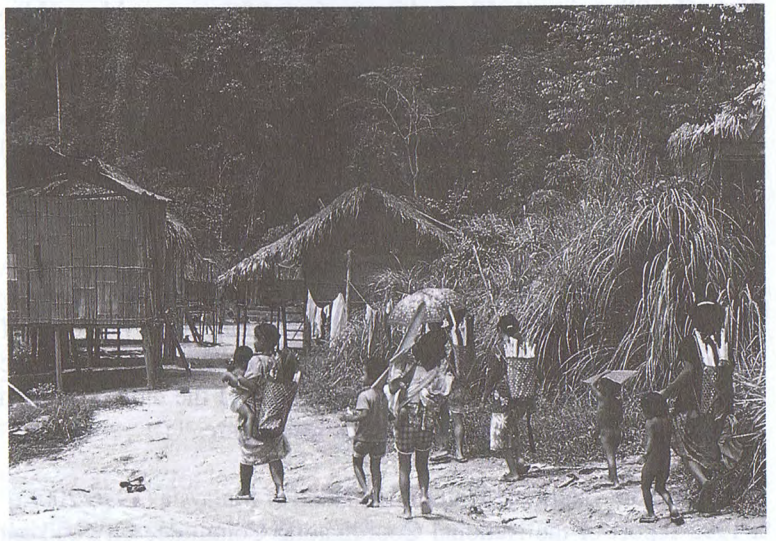
Kintak at Kupang told Schebesta (1973) in the 1920's that because of a "pneumonia" epidemic (influenza?), their group had decreased by 170, from 231 to 61 people. Polunin (1953) related that the 1918 flu pandemic killed 27 of the small Kintak Bong group and two-thirds of the Che Wong. That pandemic is also known to have devastated

Semai communities (Dentan, 1988).

A whooping cough epidemic was reported among Orang Asli in the border region of Pahang and Negri Sembilan in 1967 (Haug et al., 1969). The death rate was 5%, all deaths being in the 0 to 5 year age group. A previous epidemic of whooping cough, around the year 1928, was recalled by one elder. DPT immunization was initiated during the 1967 epidemic for children 6 years of age or less. Evidently, DPT had not been made available previously.

According to Parmer (1989), the unhealthy conditions in Malaya in the early part of the twentieth century were largely the result of British capitalization in plantation agriculture, mining, and infrastructure: ecosystems were disrupted, foreign laborers were imported, and medical personnel were ill-trained and inexperienced.

Note 58: Fix (1977) mentioned that the good manners of traditional food sharing among Semai may produce "disease sharing." By hypothesis, in the cash-economy Semai villages of Perak, where food is shared far less among households, infectious hepatitis and other such diseases may be shared less. This hypothesis has not been tested.



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Semai women and children returning from the swidden, Ulu Slim, Perak. It has been contended that medical services were not meeting the needs of the Orang Asli, especially in the interior areas, for various reasons. These include a lack of trained staff, antiquated facilities, and lack of community participation.

At the same time, food sharing is an adaptive trait ensuring the everyone gets nourishment. In the cash-only Semai situation, one might expect greater disparities in nutrition between households based on income. This conjecture has also not been tested.

Note 59: While the medical facilities might possibly have been excellent for the Orang Asli, in comparative terms they were cheap. The daily cost for a patient at Gombak Hospital in the 1970's was only 28% of that at other Malaysian government hospitals (Bolton and Snelling, 1975).

Note 60: Veeman (1986/87) listed (1) "mainly curative" visits to remote areas by the flying doctor service, (2) medical posts in remote areas, and (3) field trips from Gombak Hospital by road to accessible areas for outpatient services, immunizations, health education, and maternal and child care. In addition, malaria teams are sent out to villages at times. She contended that these services were not meeting the needs of the Orang Asli for various reasons. She cited a lack of trained staff, antiquated facilities, politics, cultural discrimination, inadequate funding, and lack of community participation as factors in this morass.
Note 61: Slimming (1958, p. 46) mentioned "the inevitable problem of the unkindness of other races."

Physical conditions also matter. Dentan et al. (1997) reported that the enormous number of logging roads in Orang Asli areas have caused widespread erosion that, among other things, damages Orang Asli food crops in their swidden fields.

CHAPTER 9

Note 62: Dentan (1988, p. 875) cited an unpublished 1983 report by Jimin Idris and others that referred to "relocation" after World War II killing about 14% of the total Orang Asli population at that time, about 7000 people – most of them children and the elderly. Dentan did not specify the time period of this loss of life.

Note 63: The "National Plan for Nutrition of Malaysia (1996-2000)," a 1995 report of the National Coordinating Committee on Food and Nutrition, envisions training programs for the health sector, but some details were not yet (in 1997) finalized.

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Appendix

THE ORANG ASLI BIOMEDICAL BIBLIOGRAPHY

Biomedical and genetic studies on Orang Asli have been intermittent since Ivan Polunin published his wide-ranging reports in the 1950's. Current research appears to be fragmented, thereby providing one rationale for cataloging what was accomplished so far. Although no other biomedical bibliography has been published on Orang Asli, a few have been compiled on Orang Asli lifeways, including those published in the Orang Asli Studies Newsletter (see, e.g, No. 10, September, 1992) and those published by Azizah Kassim (1979), Corfield (1990), Bonta (1993), and Hood (1993). They provide essential background information on the social world of Orang Asli.

In "Achievements and gaps in Orang Asli research," Benjamin (1989) listed some areas of ethnological interest that have been little studied. This bibliography will serve to shorten his list, by illustrating the many fundamental aspects of biology that impinge on Orang Asli cultures.

Biomedical research published in Malaysia is inadequately known to those outside the country. And such research published elsewhere may be incompletely known to Malaysians. The Medical Journal of Malaysia is probably read as little in the United States as the American Journal of Human Genetics is read in Malaysia, but both journals provide a broad biomedical perspective on Orang Asli life.

Few studies cited here have a longitudinal component. It is thus difficult to estimate how, how much, or why health problems have changed over time, whether due to changes in nutrition, ecology, or other factors. And many studies have been narrowly focused, leading to interpretations of medical problems as being the result of narrow causes. Yet infections, malnutrition, and anemia, for example, are

known to be closely related. In addition, genetic traits broadly affect health but are often disregarded by specialists in other fields.

Subjects such as cancer, dengue, geriatrics, leprosy, maternal health, mental illness, tuberculosis, typhus, and venereal disease have been little studied for any Orang Asli group. Malaria, nutrition, and genetics have been best studied, but not comprehensively so.

The bibliography is organized into three sections. The first section contains subject headings with the reference numbers that pertain to them. The second section lists the various Orang Asli groups with pertinent reference numbers. The third contains the references.

Section I

General and Miscellaneous Subjects: 6, 9, 11, 13, 21-23, 26, 27, 30, 33-36, 37a, 38, 40, 41, 43, 47, 48, 50, 57-59, 61, 62, 66, 69, 95, 96, 104, 108, 109, 112, 114-117, 120-122, 127, 130, 131a, 132a, 137, 142, 145, 146, 165, 167, 169a, 171, 176, 183a, 186, 186a, 188, 191, 194, 195, 197, 199, 200, 202, 203a, 204, 294a, 205, 208, 224, 228-231, 233-236, 238, 242-246, 251, 254a, 258-260, 262, 264, 265, 267-270, 276, 280, 283, 284, 286, 291a, 299-304.

Cancer: 246, 271a, 272-274.

Demography: 5, 60, 71, 73-77, 81-86, 93, 97-102, 142, 192, 195, 196, 263, 275.

Dengue: 253, 264a.

Dentistry: 1-4, 26, 142, 180.

Filariasis: 7, 45, 59, 69, 110, 142, 147, 172-174, 178, 181, 191, 206, 227, 241, 248, 298.

Genetics: 14-20, 28, 39, 42, 46, 77-80, 87-89, 91, 92, 92a, 105, 118, 125, 126, 131, 132, 143, 144, 153-164, 168, 170, 183, 185, 187, 190, 203, 203b, 207, 225, 232, 239, 252, 254, 266, 270, 278, 285, 287, 288, 292-297.

Goiter: 209, 210, 214-221, 223, 226, 237, 291.

Intestinal Worms: 59, 63-65, 67, 68, 94, 107, 133, 184, 191, 198, 222, 247, 255, 271.

Leprosy: 72.

Malaria: 8, 10, 12, 13a, 16-19, 24, 45, 49, 51, 59, 69, 82, 91, 103, 111, 119, 123, 124, 128, 129, 135, 148, 149, 151, 152, 155, 164, 168, 175, 177, 179, 185, 185a, 191, 193, 225a, 231, 240, 250, 251, 256, 257, 281, 282, 288a, 289, 298.

Mental Health: 53-56, 113, 141, 223, 277.

Nutrition: 25, 31, 44, 52, 70, 71a, 90, 129, 134, 136, 138-140, 144a, 166, 182, 201, 203, 209, 211-215, 222, 249, 291.

Schistosomiasis: 11a, 106, 107, 150, 189, 261.

Tuberculosis: 29, 72, 169, 189.

Typhus: 32, 37.

Section II

Some authors classify Orang Asli groups by language affiliation while others emphasize other criteria. All the classifications have some merit, even if they are related to assumptions about history that may be false. But older group terms such as "Sakai" and "Semang" are pejorative, while the term "Negrito" is used–and misused–in the international literature for groups in Malaysia, the Philippines, and even elsewhere. With these difficulties in mind, I classify groups here by geography.

As the citation index below shows, the small northern Orang Asli groups have been least studied biomedically, perhaps the most ignored being Batek and Mendrik. Other small groups have also gained little attention. Jah Hut, Che Wong, and Semaq Beri come to mind. Among larger groups, Jakun are the least studied, while Semai and Temuan and the best studied.

A. **Orang Asli in general**, cases in which an ethnic group was not clearly identified, or multi-group cases: 7, 9, 11-13, 14-15, 17, 18, 21-27, 29-31, 33, 34, 36-39, 41, 43, 44, 46, 47, 49, 50, 55-59, 61, 62, 64, 65, 68, 71, 72, 78, 87, 90, 92a, 94, 95, 98, 104, 105, 108, 109, 111-116, 118-120, 122, 124, 127, 130-134, 137, 140, 141, 145-148, 150, 153-159, 161-165, 167, 168, 169a, 171-174, 176, 177, 181, 183a, 185, 185a, 187, 189, 190, 192-194, 197, 199, 201-210, 216, 218, 219, 223, 225-228, 230-235, 237-243, 245-247, 249-251, 253, 254a, 255-257, 260-262, 264, 264a, 266, 267, 271a, 272-274,

276-278, 280, 282, 284-286, 288a, 291, 291a, 292, 294. 295, 299-304.

B. **Northern Groups**

1. In general (including Malaysian "Negrito"): 63, 65, 190, 252, 258, 266, 277.
2. Kensiu (including Kintak): 42, 126, 170, 220, 239.
3. Jehai: 20, 97, 100-102, 110, 142, 239, 290.
4. Batek/Bateq: 70, 71a, 106, 110.
5. Others (Lanoh, Mendrik): 157, 226, 227, 228, 231, 239, 275.

C. **Central Groups**

1. Senoi, in general: 132a, 143, 252, 272-274, 288, 290.
2. Temiar: 20, 25, 30, 35, 40, 51, 65, 96, 103, 110, 125, 128, 131a, 136, 148, 149, 153, 157, 169, 195, 226, 239, 266, 275, 277, 290, 296, 298.
3. Semai: 8, 10, 13a, 20, 25, 28, 37a, 45, 48, 52-55, 65, 73-77, 79-86, 88, 89, 91, 92, 96, 107, 129, 134, 138, 139, 153, 154, 157, 159, 162, 163, 170, 175, 177, 182, 183, 191, 196, 213, 215, 217, 221, 222, 224, 226-229, 231, 239, 244, 254, 266, 269, 277, 278, 289, 291-293, 296, 297.
4. Others (Jah Hut, Che Wong, Semaq Beri): 5, 144a, 153, 157, 186a, 188, 193, 200, 236, 277.

D. **Southern Groups**

1. Besisi/Mahmeri: 153, 220, 239, 268, 277, 298.
2. Semelai: 32, 60, 65, 69, 90, 93, 106, 121, 153, 157, 186, 266, 277, 287, 293, 297, 298.
3. Temuan: 1-4, 16, 19, 25, 32, 65-67, 89, 99-101, 106, 118, 123, 142, 151-153, 157, 160, 162, 163, 178, 179, 184, 198, 211-215, 217, 221, 224, 226, 227, 239, 248, 253, 259, 265, 266, 270, 271, 277, 281, 283, 293, 297, 298.
4. Jakun: 6, 20, 65, 117, 153, 157, 160, 162, 166, 172, 206, 227, 239, 263, 266, 277, 293, 297, 298.
5. Others (Orang Kanaq, Orang Seletar, Orang Kuala): 226, 228, 231, 239, 277.

Section III: References

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Betau, Pahang, Semai; 12% of supposedly malaria-negative persons were in fact infected, as shown by this new methodology.)

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28. Bolton, J., and L. Lie-Injo. Hb E-beta thalassemia in West Malaysian Orang Asli (aborigines). *Med. J. Malaysia* 24: 36-40, 1969. (Study of an extended family in Gopeng, Perak: Semai x Chinese.)

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30. Bonta, B. *Peaceful Peoples: An Annotated Bibliography.* Scarecrow, Metuchen, NJ, USA, 1993. (Includes Batek, Che Wong, Semai, and Temiar.)

31. Brearley, A. Serum proteins, hematocrits, heights and weights of aborigine subjects in W. Malaysia. *Med. J. Malaysia* 24: 183-186, 1970. (Study of 109 Orang Asli, 4 to 45 yrs. of age, accompanying patients to Gombak Hospital, ethnicity not specified; 88 were from the forest fringe, 21 from deep forest; none were from resettled sites. Serum protein levels were healthy, implying that nutrition was adequate, at least for these hospital visitors.)

32. Brown, G., D. Robinson, and D. Huxsoll. Serological evidence for a high incidence of transmission of Rickettsia tsutsugamushi in two Orang Asli settlements. *Amer. J. Trop. Med. Hyg.* 27: 121-124, 1978. (On scrub typhus in Bukit Lanjan Temuan and Post Iskandar Semelai.)

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34. Bulbeck, F. A reevaluation of possible evolutionary processes in Southeast Asia since the late Pleistocene. *Bull. Indo-Pacific Prehistory Assoc.* 3: 1-21, 1982. (Based on skeletal remains in West Malaysia dating to 3,000-10,000 years ago, ancient Malaysians were taller than the Semai or Malayan hunter-gatherers of today.)

35. Burns-Cox, C., and J. Maclean. Splenomegaly and blood pressure in an Orang Asli community in West Malaysia. *Amer. Heart J.* 80: 718-719, 1970. (On 85 River Nenggiri Temiar, male and female; found little risk of coronary heart disease.)

36. Burns-Cox, C., Y. H. Chong, and R. Gilman. Risk factors and the absence of coronary heart disease in aborigines in West Malaysia. *Brit. Heart J.* 34: 953-958, 1972. (Study of 73 Orang Asli men visiting Gombak Hospital; 1 case of elephantiasis was found; 34% had enlarged spleens; all had low blood pressure and were adequately nourished.)

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40. Chan, O. L, et al. The cardiorespiratory fitness and energy expenditure of the Temiars. *Med. J. Malaysia* 28 (4): 267-272, 1974. (On Temiar boys and men in the Nenggiri area of the Kelantan River; no malnutrition or yaws found; N=35.)

41. Chee, H. L. Health and nutrition of the Orang Asli: the need for primary health care amidst economic transformation. In: *Indigenous Minorities of Peninsular Malaysia*. Razha Rashid, ed. Intersocietal and Scientific Sdn. Bhd., Kuala Lumpur, 1995. Pp. 48-71. (A general review; cites and discusses some unpublished work.)

42. Chen, H., R. Sokal, and M. Ruhlen. Worldwide analysis of genetic and linguistic relationships of human populations. *Human Biol.* 67: 595-612, 1995. (Includes data on "Kensiu," but this may be a mislabeling of older "Negrito" data; suggests Kensiu may be as genetically related to Malays as, for example, Turks are to Koreans.)

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44. Chen, P. C. Food habits and malnutrition. *Med. J. Malaysia* 31: 170-174, 1977. (Passing reference to Orang Asli, citing work by others.)

45. Chiang, G. L., et al. Effectiveness of repellent/insecticidal bars against malaria and filariasis vectors in Peninsular Malaysia. *Southeast J. Trop. Med. Pub. Hlth.* 21 (3): 412-417, 1990. (Studied mosquito repellents at Betau, Pahang, in a Semai village.)

46. Chin, J. Absence of Di^{a+} in Malayan aborigines. *Nature* 201: 1039, 1964. (Study of 270 "aboriginal Malays" and Senoi.)

47. Chooi, O. H. The ethnobotany of Citrus and their relatives. *Korean J. Plant Taxonomy* 24 (3): 157-171, 1994. (On Orang Asli traditional medical uses.) (Note: the author has also been cited in databases as Ong Hean Chooi.)

48. Chong, Y. H., and C. W. Pang. Blood lipids in the Malayan aborigines. In: *Food and Nutritional Biochemistry*. P. Kanagasuntheram, ed. Proc. 2nd Symp. Fed. Asian and Oceanian Biochemists, Kuala Lumpur, 1980. Pp. 165-168. (Determined that periurban Semai adults had a higher risk of coronary heart disease than did forest-fringe or deep-forest Orang Asli.)

49. Collins, W., et al. Studies on the relationship between fluorescent antibody response and ecology of malaria in Malaysia. *Bull. World Health Organization* 39: 451-463, 1968. [Studied Batu 55 Jerentut Rd. resettlement village in Pahang and 9 sites in Upper Perak/Ulu Kelantan (Fort Kemar, Banding, Kuah, Sira, Peltu, Fort Betau, Fort Chabai, Kuala Yai, and Gamala).]

50. Corfield, J. *A Comprehensive Bibliography of Literature Relating to the Orang Asli of West Malaysia*. Working Paper 61, Centre of Southeast Asian Studies, Monash University, Clayton, Victoria, Australia, 1990. (Emphasizes the social sciences.)

51. Delorme, D., et al. Identification of sporozoites in Anopheles maculatus from Malaysia by enzyme-linked immunosorbent assays. *Trop. Biomed.* 6: 21-26, 1989. (On Post Legap Temiar.)

52. Dentan, R. Some Senoi Semai dietary restrictions. Ph. D. dissertation, Yale University, New Haven, Conn., USA, 1965.

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59. Dissanaike, A. S., et al. Studies of parasitic infections in Orang Asli (Aborigines) in Peninsular Malaysia. *Med. J. Malaysia* 32: 48-55, 1977. (Studied mainly hospital patients; ethnic groups not identified; 25% had amebic antibodies; 21% had E. coli infections, over 90% had intestinal helminths; 11% had falciparum malarial parasitemia and 89% were seropositive for the falciparum parasite; 14% had microfilariae; 16% were seropositive for toxoplasmosis.)

60. Dobbins, J. Life expectancy in an aboriginal Malaysian population. *Southeast Asian J. Trop. Med. Pub. Hlth.* 10 (1): 106-114, 1979. (Reports Semelai life tables, infant mortality, and life expectancy.)

61. Dugdale, A., J. Bolton, and A. Ganendran. Respiratory function among Malaysian aboriginals. *Thorax* 26 (6): 740-743, 1971. (On Orang Asli at Gombak Hospital; mentions that gout and tuberculosis were common.)

62. Dunn, F. The current status of ethnographic, genetic and other biomedical research among the primitive ethnic groups of Southeast Asia. In: *The Biology of Human Adaptability*, P. Baker and J. Weiner, eds. Oxford University Press, London, 1968a. Pp. 533-563. (Note: This chapter was not included in the first printing of the book.)

63. Dunn, F. Epidemiological factors: health and disease in hunter-gatherers. In: *Man the Hunter*, R. Lee and I. DeVore, eds. Aldine, Chicago, 1968b. Pp. 221-228. (Summarizes intestinal parasitism of "Negritos"; reports 22 species of such parasites, higher than for desert dwellers.)

64. Dunn, F. The TIF direct smear as an epidemiological tool. *Bull. World Health Organization* 39: 439-449, 1968c. (Laboratory evaluations, based on Orang Asli fieldwork.)

65. Dunn, F. Intestinal parasitism in Malayan aborigines (Orang Asli). *Bull. World Health Organization* 46: 99-113, 1972. (Found 92% of 1036 persons–"Negritos," Temiar, Jah Hut, Semai, Semelai, Temuan, and Jakun–had parasites.)

66. Dunn, F. *Rain-Forest Collectors and Traders.* Monographs of the Roy. Asiatic Soc., Malaysian Branch, No. 5, Kuala Lumpur, 1975. Ed. 2, 1982. (Lists some medicinal plants used by Temuan in northern Selangor.)

67. Dunn, F. Secular changes in Temuan (Malaysian Orang Asli) settlement patterns, subsistence, and health. *Malayan Nature J.* 31 (2): 81-92, 1977. (Studied N. Selangor area; associated intestinal parasitism with crowded conditions after 1945; reported improvement in malarial control in the 1960's.)

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70. Endicott, K. Batek Negrito sex roles. *Second Internat. Cong. on Hunter-Gatherers*. Lavalle, Quebec, 1980. Pp. 625-670. (Contains food consumption data.)

71. Endicott, K. M. The effects of slave raiding on the aborigines of the Malay Peninsula. In: *Slavery, Bondage, and Dependency in Southeast Asia*. A. Reid and J. Brewster, eds. University of Queensland Press, Brisbane, 1983. Pp. 216-245. (Discusses demographic and other consequences of pre-1920 slave raiding.)

71a. Endicott, K. M. Batek history, interethnic relations, and subgroup dynamics. In: *Indigenous Peoples and the State.* R. Winzeler,

ed. Yale University Southeast Asia Studies Monograph 46. New Haven, Conn., 1997. Pp. 30-50.

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81. Fix, A. Endogamy and settlement populations of Semai Senoi: potential mate pool analysis and simulation. *Social Biol.* 28: 62-74, 1982b.

82. Fix, A. Kin groups and trait groups: population structure and epidemic disease selection. *Amer. J. Phy. Anthropol.* 65: 201-212, 1984. (Uses Semai data to test models of variant-gene selection, driven by malaria.)

83. Fix, A. Evolution of altruism in kin-structured and random subdivided populations. *Evolution* 39 (4): 928-939, 1985. (Uses a Semai model for theory testing.)

84. Fix, A. Semai Senoi fertility and population dynamics: two-census method. *Amer. J. Hum. Biol.* 1: 463-469, 1989a. (Reports a 2% growth rate, with a lower rate before 1969.)

85. Fix, A. Semai Senoi mortality: two-census method. *Amer. J. Hum. Biol.* 1: 471-477, 1989b. (Reports that lower mortality accords with health care improvement.)

86. Fix, A. Changing sex ratio of mortality in the Semai Senoi. *Human Biol.* 63: 211-220, 1991. (Reports that prior to 1969 Semai women had higher mortality than men.)

87. Fix, A. Malayan paleosociology: implications for patterns of genetic variation among the Orang Asli. *Amer. Anthropologist* 97 (2): 313-323, 1995.

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89. Fix, A., A. Baer, and L. E. Lie-Injo. The mode of inheritance of ovalocytosis/elliptocytosis in Malaysian Orang Asli families. *Hum. Genetics* 61: 250-253, 1982. (Contains Temuan and Semai data.)

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92a. Gajra, B., et al. Genotype associations among seven apolipoprotein B polymorpohisms in a population of Orang Asli of western Malaysia. *Hum. Biol.* 69 (5): 629-640, 1997.

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94. Gilman, R., C. Davis, and F. Fitzgerald. Heavy Trichuris infection and amoebic dysentery in Orang Asli children. *Trans. Roy. Soc. Trop. Med. Hyg.* 70: 313-316, 1976. (On Gombak Hospital patients and visitors.)

95. Gilman, R., and K. Prathap. Acute intestinal amoebiasis-proctoscopic appearances with histopathological correlation. *Annals Trop. Med. Parasit.* 65: 359-365, 1971. (On Orang Asli.)

96. Gilman, R., et al. Seroepidemiology of amoebiasis in the Orang Asli (Western Malaysian aborigines) and other Malaysians. *Amer. J. Trop. Med. Hyg.* 25: 663-666, 1976. (On Satak Semai and Belatim Temiar; Satak had high and Belatim low levels of ameba antibodies.)

97. Gomes, A. G. A social demography of Jahai Negritos at Rual Post, Kelantan. University of Malaya, Kuala Lumpur, B. A. thesis, 1977.

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99. Gomes, A. G. *The Paya Lebar Temuans: A social demographic study.* Dept. of Anthropology and Sociology, Working Paper No. 3, University of Malaya, Kuala Lumpur, 1979b.

100. Gomes, A. G. *Ecological Adaptation and Population Change: Semang Foragers and Temuan Horticulturists in West Malaysia.* East-West Environment and Policy Institute, Research Report No. 12, East-West Center, Honolulu, HI, 1982. (Study of Jehai at Sungai Rual, Kelantan, and Temuan at Paya Lebar, Selangor.)

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102. Gomes, A. G. Demographic implications of villagisation among the Semang of Malaysia. In: *Hunter-Gatherer Demography: Past and Present,* B. Meehan and N. White, eds. Oceania Publications, Sydney, 1990. Pp. 126-138. (On Sungai Rual Jehai governmental settlement.)

103. Gordon, D., et al. Significance of circumsporozoite-specific antibody in the natural transmission of Plasmodium falciparum, Plasmodium vivax, and Plasmodium malariae in an aboriginal (Orang Asli) population of central Peninsular Malaysia. *Amer. J. Trop. Med. Hyg.* 45 (1): 49-56, 1991. (Studied 275 Temiar; 56% of the 0-4 yr. age group had malarial parasitemia, but the over-40 yrs. group had 0%; study design on acquired immunity ignored human genetic resistance to malaria.)

104. Gouldsbury, P. *Jungle Nurse.* Jarrolds, London, 1960.

105. Green, R. Anthropological blood grouping among the "Sakai." *Bull. Raffles Mus.*, Series B, No. 4, pp. 130-132, 1949.

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107. Greer, G., et al. Malaysian schistosomiasis: description of a population at risk. *J. Trop. Med. Hyg.* 92 (3): 203-208, 1989. (At Kuala Koyan, Pahang, 79% of 56 Semai had roundworms, 93% had whipworms, 84% had hookworm, and none had schistosomes. By serology, < 10% had anti-schistosome antibodies.)

108. Hakim, S. L., et al. Seroprevalence of Toxocara canis antibodies among Orang Asli (aborigines) in Peninsular Malaysia. *Southeast Asian J. Trop. Med. Pub. Hlth.* 23 (3): 493-496, 1992. (Human toxocariasis is caused by a dog nematode and is associated with visceral larval migrans; of 480 Orang Asli

studied at Gombak Hospital, 32% had relevant antibodies.) (Note: the lead author is cited as Lokman Hakim, S., or Lokman, H. S., in various databases.)

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113. Hartog, J. Institutions for the mentally and socially deviant in Malaysia. *Asian J. Medicine* 8: 170-177, 1972. (Includes incidental remarks on Orang Asli.)

114. Haug, N., et al. Studies on bacterial disease in West Malaysian Orang Asli (Aborigines): An epidemic of whooping cough. *Med. J. Malaya* 23: 192-198, 1969. (The epidemic covered villages in Negri Sembilan and western Pahang, among Temuan and Semelai children, as well as a few Jah Hut; over 300 children were affected, but the mortality rate was low.)

115. Haug, N., et al. Studies of bacterial disease in West Malaysian Orang Asli: distribution of bacterial enteropathogens. *Med. J. Malaya* 24: 24-31, 1969. (Found deep-forest groups differed little from forest-fringe groups in diarrhea level; those under 12 yrs. of age were most commonly affected.)

116. Haug, N., T. W. Lim, and J. Anandon. Studies of bacterial disease in West Malaysian Orang Asli: previously unrecorded Salmonella serotypes. *Med. J. Malaya* 23: 269-271, 1969.

117. Hill, A. Notes on traditional healing and modern medicine among the Orang Hulu (Jakun) of Johore. Department of Anthropology, Sydney University, Australia, 1974. (Johore Utara, Bekok, Segamat, Johore.) (Note: in 1996 the Dept. in Sydney had no record/copy of this report.)

118. Hirayama, K., et al. Molecular analysis of HLA-B in Malaysian aborigines. *Tis. Antigens* 48: 692-697, 1996. (On Gapoi, Pahang, Temuan and a mixed sample of Temiar and Semai.)

119. Ho Keong Bin. Current status of malaria and anti-malaria programme in Malaysia. In: *Proc. Asia and Pac. Conf. on Malaria.* W. Siddiqui, ed. Dept. Trop. Med., Sch. of Med., Univ. Hawaii, Honolulu, 1985. Pp. 63-74.

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121. Hoe Ban Seng. Aboriginal Community at Tasek Bera, Southwest Pahang–A Study on the Structure of Semelai Society. Report to Commissioner of Aborigines, Dept. of Aboriginal Affairs, Kuala Lumpur, 1964 (typescript). (Pp. 243-246 give a health overview.)

122. Hood, M. S. Senarai Hasil kajian Mengenai Orang Asli 1824-1980. In: *Mereka yang Terpinggir,* by Hood Salleh, K. Said, and Awang H. Mois. Department of Anthropology and Sociology, Universiti Kebangsaan Malaysia, Bangi, 1993. Pp. 40-67.

123. Huehne, H., et al. A comprehensive account of the malaria eradication pilot project in Malaya. *Med. J. Malaya* 21: 3-25, 1966. (A few Selangor Temuan areas were studied: Bukit Manchong, Bkt. Kelubi, Bkt. Legong, Rantau Panjang, Sungai Choh Estate, Sg. Jinjang, Ulu Kuang, and Ulu Serendah; Orang

Asli were not included in the project until 3 yrs. after it was started.)

124. Huehne, W. H., M. D. Ahmad, and D. S. Ling. Malaria, a primary health problem in rural West Malaysia. *Med. J. Malaya* 22: 60-71, 1967. (Studied a few Orang Asli areas but mainly other areas; data analyzed at state level; cited in Lie-Injo, 1969, as showing an erroneously low malarial rate in W. Malaysia.)

125. Hughes, D. Senoi Temiar: dermatoglyphic data. *Man* 63: 74 only, 1963. (On Pasir Riang and Gemalah, Kelantan.)

126. Hughes, D. Kensiu Negritos: dermatoglyphic data with comparative notes. *Man* 64: 82-85, 1964.

127. Hughes, D. The physical anthropology of south-east Asia. University of Cambridge, England, doctoral dissertation, 1965.

128. Institute for Medical Research, Kuala Lumpur, Malaysia. Annual Report, 1987. (Pp. 181-182 record a 37-40% prevalence of malarial parasitemia in Post Legap Temiar, with nearly 60% in children < 10 yrs. of age, according to Lambros et al., 1989.)

129. Ismail Mohd. Noor, T. S. Wong, and Zawiah Hashim. Anthropometric and food intake studies among Semai children. *J. Malaysian Soc. Hlth.* 6 (1): 19-25, 1988. (For 13 administrative villages in Betau, Pahang, a majority of the preschool children and over a third of the 7-to-10 year olds studied were underweight or stunted, despite the fact that school attendees in the latter group received meals at school.)

130. Jabatan Hal Ehwal Orang Asli. Appendix: Research on the Orang Asli. *Federation Museums J.* 24: 188-208, 1979.

131. Jarolim, P., et al. Deletion of erythrocyte band 3 gene in malaria-resistant Southeast Asian ovalocytosis. *Proc. Natl. Acad. Sci. USA* 88: 11022-11026, 1991. (Includes data on one Orang Asli family, although not so stated in the report.)

131a. Jennings, S. *Theatre, Ritual and Transformation: the Senoi Temiar.* Routledge, London, 1995.

132. Joysey, V., et al. Study of a Malay population. In: *Histocompatibility Testing 1972.* J. Dausset and J. Colombani, eds. Munksgaard, Copenhagen, 1973. Pp. 251-260. (Includes HLA data on 38 Orang Asli patients from Gombak Hospital; the HLA-A antigen called "Long Malay" in this report is Aw34, which is common in the Pacific.)

132a. Kamath, S. Hepatitis B surface antigen subtypes in Malaysia. *Amer. J. Epidem.* 102 (2): 191-195, 1975. ("Senoi" were like Sarawak "Dayaks," but unlike other Malaysian groups studied, in having a certain antigen subtype in high frequency.)

133. Kan, S. P. Malaysia and other Southeast Asian countries. In: *Ascariasis and its Prevention and Control.* D. Compton et al., eds. Taylor & Francis, London, 1989. (Not specifically on Orang Asli, but a good overview on intestinal worms.)

134. Kasim, Mohd. S., Zulkifli Ismail, and Lailanor Ibrahim. Nutritional status of children of various Orang Asli communities in Peninsular Malaysia. *J. Singapore Paediatr. Soc.* 29, Suppl. 1: 96-100, 1987. Reprinted in: *Akademika* (Malaysia) 35: 69-74, 1989. (Studied Cameron Highlands; Post Jernang area, Sungkai, Perak; DARA project in S. E. Pahang; W. Pahang; Semai at the resettlement Land Development Scheme, Betau, Pahang; interior Kelantan; and Sungai Temuan, S. Johore.)

135. Khoo, A., et al. Nested polymerase chain reaction for detection of Plasmodium falciparum infection in Malaysia. *Trans. Roy. Soc. Trop. Med. Hyg.* 90: 40-41, 1996. (On Betau, Pahang, Semai; 12% of supposedly malaria-negative persons were in fact infected, as shown by this new methodology.)

136. Khoo, T. E. Some aspects of the nutritional status of Temiar in Kemar. University of Malaya, Kuala Lumpur, Masters of Public Health thesis, 1977. (On the Fort Kemar, Perak, resettlement community bereft of territory for the acquisition of forest food resources; children were found to be underweight and stunted; women were found to be iron-deficient anemic.)

137. Khoo, T. E. Health priorities in the resettlement of the Orang Asli. *Federation Mus. J.* 24: 177-184, 1979. (Lists 6 negative impacts on forced relocations to 1 potentially positive one.)

138. Khor, G. L. A study of the nutritional status of the Semai. Universiti Malaya, Kuala Lumpur, Ph. D. dissertation, 1985.

(Semai of Batang Padang District, Perak, had shorter lifespans than Malaysians in general; point prevalence rates for malaria parasitemia in the 1980's ranged from 1% to 21%, with over 90% of parasitemia being in those under 19 yrs. of age; major child-mortality causes were diarrhea and fevers; the diet was found to be deficient in protein, calories, calcium, and iron.)

139. Khor, G. L. Malnutrition among Semai children. *Med. J. Malaysia* 43 (4): 318-326, 1988. (Semai of Batang Padang District, Perak, had 24-44% iron-deficiency anemia, 42-78% stunting, and 20-65% underweightness, by age group; N=1180; for those 0-12 yrs. of age, 14-57% had various intestinal worm infestations.)

140. Khor, G. L. Resettlement and nutritional implications: the case of Orang Asli in regroupment schemes. *Pertanika J. Soc. Sci. and Hum.* 2: 123-132, 1994. (A review, with international comparisons.)

141. Kinzie, J., and J. Bolton. Psychiatry with the aborigines of West Malaysia. *Amer. J. Psychiatry* 130 (7): 769-773, 1973. [Thoughtful discussion of lack of suicide or physical aggression among Orang Asli, but the data set in Tan and Armstrong (which see) is more comprehensive.]

142. Kinzie, J., K. Kinzie, and J. Tyas. A comparative health survey among two groups of Malayan Aborigines. *Med. J. Malaya* 21: 135-139, 1966. [Reports on weight, height, dental problems, goiter, malaria, filariasis, anemia, intestinal parasitism, diabetes, respiratory infections, and pre-adult mortality for Temuan at Bukit Cheeding, Selangor, and Jehai at Fort Banding, Upper Perak River. Found 26% Temuan and 54% Jehai pre-adult mortality and a 17% microfilarial rate for adults in both groups; 1 of 53 Temuan and 4 of 40 Jehai had elephantiasis; hematocrit levels were normal in adult men (indicating little iron deficiency) but were lower in women.]

143. Kirk, R. Genetic differentiation in Australia and the Western Pacific. In: *The First Americans*. A. Harper and W. Laughlin, eds. G. Fischer, NY, 1979. Pp. 211-237. (Presents a genetic distance analysis showing "Senoi" outside of the Western Malayo-Polynesian cluster.)

144. Kirk, R., and L. Lai. The distribution of haptoglobin and transferrin groups in South and Southeast Asia. *Acta Genet.* 11: 97-105, 1961. (On "Proto-Malays.")

194a. Kuchikura, Y. Food use and nutrition in a hunting and gathering community in transition, Peninsular Malaysia. *Man and Culture in Oceania* 4: 1-30, 1988. (Mainly on Semaq Beri.)

145. Kutty, M. K., A. M. Das, and A. S. Dissanaike. Sarcocystic infection in an Orang Asli: the second human case from Malaysia. *Southeast Asian J. Trop. Med. Pub. Hlth.* 6:400, 1975. (On incidental parasitism.)

146. Laderman, C. Destructive heat and cooling prayer. *Soc. Sci. Med.* 25 (4): 357-365, 1989. (Relates Malay pre- through post-natal practices in large part to those of the Orang Asli.)

147. Laing, A., and R. Wharton. Filariasis investigation. In: Ann. Report, 1960. Institute for Medical Research, Kuala Lumpur, 1961. Pp. 114-131. (First report of filariasis foci in Orang Asli.)

148. Lambros, C., D. Davis, and G. Lewis. Antimalarial drug susceptibility of Plasmodium falciparum isolates from forest fringe dwelling aborigines (Orang Asli) of Peninsular Malaysia. *Amer. J. Trop. Med. Hyg.* 41 (1): 3-8, 1989. (Parasites from Temiar at Post Legap, Perak, and from Pahang and Selangor patients at Gombak Hospital were studied; 93% of falciparum isolates were susceptible to chloroquine.)

149. Lee, M., et al. Interaction of Malaysian sera with Plasmodium vivax sporozoite antigen. *Amer. J. Trop. Med. Hyg.* 39 (6): 535-539, 1988. (Found 49% of Post Legap, Perak, Temiar had malarial parasites; 76% of 0.5-4 yr. group had parasitemia, but adults had lower parasitemia, about 40%, and high acquired immunity; human genetic resistance variation was ignored.)

150. Leong, S. H., R. Murugasu, and K. C. Chong. Schistosomiasis in the Orang Asli (A report of 9 cases). In: *Proc. 10th Malaysian-Singapore Congr. of Med.*, Stamford College Press, Singapore, 1975. Pp. 184-186.

151. Lewis, A., T. Dondero, and J. T. Ponnampalam. Falciparum malaria resistant to chloroquine suppression but sensitive to

chloroquine treatment in West Malaysia. *Trans. Roy. Soc. Trop. Med. Hyg.* 67: 310-312, 1973. (Found a monthly rate of 6% for new falciparum and 5.7% for new vivax parasitemia in Negri Sembilan Temuan.)

152. Lewis, A., and J. T. Ponnampalam. Suppression of malaria with monthly administration of combined sulphadoxine and pyrimethamine. *Annals Trop. Med. Parasit.* 69: 1-12, 1975. [On Temuan; pre-treatment parasitemia rates were 25% for Senebai (children) and 32% for Tekir Labu (all ages).]

153. Lewis, G., et al. Duffy phenotypes in Malaysian populations: correction of previous unusual findings. *Trans. Roy. Soc. Trop. Med. Hyg.* 82: 509-510, 1988. (Reports on Semai, Temiar, Jah Hut, Semok Beri, Semelai, Mahmeri, Temuan, and Jakun; no Duffy-negative phenotypes were found, correcting the report by Ooi, 1979.)

154. Lie-Injo, L. E. Hereditary ovalocytosis and hemoglobin E-ovalocytosis in Malayan aborigines. *Nature* 208: 1329, 1965. (Subjects unspecified, but mostly Semai.)

155. Lie-Injo, L. E. Distribution of genetic red cell defects in Southeast Asia. *Trans. Roy. Soc. Trop. Med. Hyg.* 63: 664-674, 1969. (Discusses genetic red-cell variants and malarial history in W. Malaysians.)

156. Lie-Injo, L. E. Genetic relationships of several aboriginal groups in South East Asia. In: *The Origin of the Australians*, R. Kirk and A. Thorne, eds. Australian Institute of Aboriginal Studies, Canberra, 1976. Pp. 277-306. (Contains some data not available elsewhere, e.g., monomorphism of lactate dehydrogenase, phisohexose isomerase, catalase, and carbonic anhydrase in Orang Asli.)

157. Lie-Injo, L. E., and J. Chin. Abnormal hemoglobins and glucose-6-phosphate dehydrogenase deficiency in Malayan aborigines. *Nature* 204: 291-292, 1964. (Reports on Semai, Temiar, Semelai, Temuan, and Jakun for G6PD and hemoglobins and on Jah Hut for G6PD and Lanoh for Hb; N ranged from 1 to 181.)

158. Lie-Injo, L. E., et al. Unusual albumin variants in Indonesians and Malayan aborigines. *Hum. Heredity* 21: 376-383, 1971. (Two "Gombak" variants were found in a sample of 165 Orang Asli, but Baer et al., 1976, found no variants in a sample of 189 Temuan.)

159. Lie-Injo, L. E., et al. Haemoglobin E-hereditary elliptocytosis in Malayan aborigines. *Acta Haematologica* (Basel) 47: 210-216, 1972. (Reports on a mixture of Orang Asli, but mainly Semai.)

160. Lie-Injo, L. E., et al. Hemoglobin constant spring (slow-moving hemoglobin X components) and hemoglobin E in Malayan aborigines. *Amer. J. Hum. Genetics* 25: 382-387, 1973. (Reports on Temuan and Jakun.)

161. Lie-Injo, L. E., J. Bolton, and H. Fudenberg. Haptoglobins, transferrins and serum gamma-globulin types in Malayan aborigines. *Nature* 215: 777 only, 1967. (On a mixture of groups.)

162. Lie-Injo, L. E., J. Ganesan, and C. Lopez. The clinical, hematological, and biochemical expression of Hemoglobin Constant Spring and its distribution. In: *Abnormal Hemoglobins and Thalassemia–Diagnostic Aspects.* Academic Press, NY, 1975. Pp. 275-291. (On Semai, Temiar, Temuan, Jakun.)

163. Lie-Injo, L. E., and Q. Welch. Electrophoretic variants of 6-phosphogluconate dehydrogenase (6PGD) and phosphohexose isomerase (PHI) in different racial groups in Malaysia. *Hum. Heredity* 22: 338-343, 1972. (On Temuan, Semai, and miscellaneous others.)

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165. Lim, H. F. *Orang Asli, Forest and Development.* Forest Research Institute Malaysia, Kepong. 1997.

166. Lim, H. W. Nutritional status and reproductive health of Orang Asli women. B. S. Thesis, Fac. of Hum. Ecol., Universiti Putra Malaysia, Serdang, 1997. (Studied 34 reproductive-age Jakun women in Sungai Soi and Batu-15 villages in Pahang;

on average, they showed significant nutritional deficits in calories, thiamine, vitamin A, riboflavin, and–especially–iron.)

167. Lim, K. G. *A Review of Disease in Malaysia.* Pelanduk, Petaling Jaya, Malaysia. 1993. (Orang Asli receive minor mention.)

168. Livingstone, F. *Frequencies of Hemoglobin Variants: Thalassemia, the Glucose-6-Phosphate Dehydrogenase Deficiency, G6PD Variants, and Ovalocytosis in Human Populations.* Oxford University Press, NY. 1985. (A thorough review of genetic red-cell variants relevant to malarial resistance in humans, including all Orang Asli groups studied before 1985.)

169. Lokman, M. N., and Z. Baharuddin. Tuberculin sensitivity in Malaysian children: lessons from the Orang Asli. *Malaysian Med. Sciences* 3 (1) (suppl.): 51, 1996. (On Legap Temiar.)

169a. Looi, L. M., and K. Prathap. Amyloidosis in Malayan aborigines (Orang Asli). *Pathology* 11 (4): 575-582, 1979. (Nine of 334 autopsied Orang Asli had amyloidosis.)

170. Lugg, J. Taste thresholds for phenylthiocarbamide of some populations. *Annals Hum. Genetics* 21: 244-253, 1957. (Reports 18% of 50 Kintak Bong and 4% of 50 Semai were nontasters; see also PTC data in Baer et al., 1976.)

171. MacLean, J., and K. R. Kamath. Infantile scurvy in Malaysia. *Med. J. Malaysia* 24: 200-207, 1970. (On 4 Orang Asli children in Gombak.)

172. Mak, J. W. Studies on filariasis amongst Orang Asli in Peninsular Malaysia with special reference to chemotherapy. M. D. thesis, Univ. Singapore, 1978. (Studied 2645 Orang Asli at Gombak Hospital; found a microfilarial rate of 6.3% with no sex difference, but the rate increased with age, to late teens, then stabilized; only 27 "Negritos" were tested; 6% of the 2645 had malarial parasites.)

173. Mak, J. W., ed. *Filariasis.* Bulletin No. 19, Institute for Medical Research, Kuala Lumpur, 1983. (Contains information on Orang Asli and others in W. Malaysia and a map of filariasis on p. 83, updating that published by Wharton et al. in 1963.)

174. Mak, J. W. Problems in filariasis control and the need for human behavior and socioeconomic research. *Southeast Asian J. Trop. Med. Pub. Hlth.* 17 (3); 479-485, 1986.

175. Mak, J. W. Review of seroepidemiological tools for control program of parasitic diseases in Malaysia. *Trop. Biomedicine* 5 (suppl. 1): 28-32, 1988. (On Ulu Jelai, Perak, and Betau, Pahang, Semai; malarial parasitemia rates were high, higher at Betau, with a 62% rate in the 0-9 yr. age group there.)

176. Mak, J. W. Medical research in Malaysia: parasitic diseases. In: *90th Anniv. Scientific Seminar: Health Research.* (No editor.) Institute for Medical Research, Kuala Lumpur, 1992. Pp. 29-69. (Reports on malaria, filariasis, or other conditions in Temuan, Temiar, Jehai, Batek, Semai, Semelai, and Johore groups.)

177. Mak, J. W. Current malaria research activities in Malaysia. *J. Bioscience* (Malaysia) 5 (1 & 2): 73-79, 1994. (Notes half the reported malarial infections in W. Malaysia were in the Orang Asli in 1991-1992; gives parasitemia data for Betau, Pahang, Semai.)

178. Mak, J. W., et al. Studies on the epidemiology of subperiodic Brugia malayi in Malaysia. *Acta Tropica* 39: 237-245, 1982. (On Sungei Lui, Selangor, Temuan.)

179. Mak, J. W., et al. Parasitological and serological surveys for malaria among the inhabitants of an aborigine village and an adjacent Malay village. *Acta Tropica* 44: 83-89, 1987. (On Sungai Lui Temuan; authors took into account some genetic resistance to malaria in the Orang Asli later: see Foo et al., 1992.)

180. Mak, J. W., et al. Epidemiology and control of malaria in Malaysia. *Southeast Asian J. Trop. Med. Pub. Hlth.* 23 (4): 572-577, 1992. (A review.)

181. Marzhuki, M., A. Tham, and S. Poovaneswari. Current status of filariasis in Malaysia. *Southeast Asian J. Trop. Med. Pub. Hlth.* 24, Suppl. 2: 10-14, 1993. (Includes surveys of Orang Asli areas, as shown in maps, but contains no discussion by ethnic group.)

182. Massita Mohd. Sin. Penilaian taraf pemakanan kanak-kanak Orang Asli di Rancangan Pengumpulan Semula (RPS), Betau, Pahang. B. S. thesis, Fac. of Human Ecology, Universiti Pertanian Malaysia, Serdang, 1992. (Cited in Chee, 1995; among 129 Semai children 8 yrs. of age or younger, more than a third were underweight or stunted; energy nutrition averaged only 77% of the recommended daily allowance.)

183a. McLeod, F. Midwifery among the aborigines. *Nursing Mirror News* 132 (11): 28-31, 1971. (Observations on Gombak Hospital care.)

183. Melton, T., et al. Polynesian genetic affinities with Southeast Asian populations as identified by mtDNA analysis. *Amer. J. Hum. Genet.* 57: 403-414, 1995. (On Semai.)

184. Mohamed Kamel, A., Mohamed Sham Kasim, and K. P. Lai. Parasitic infections among Orang Asli community in Pangsun, Hulu Langat. In: Annual Report, Institute for Medical Research, Kuala Lumpur, 1994. Pp. 46-47. (Found 82% of the Temuan studied had helminthic or protozoan infections.)

185. Mohandas, N., et al. Rigid membranes of Malayan ovalocytes: a likely genetic barrier against malaria. *Blood* 63: 1385-1392, 1984.

185a. Moorhouse, D. Some entomological aspects of the malaria eradication pilot project in Malaya. *J. Med. Entomol.* 2 (2): 109-119, 1965. (On Selangor Temuan at Klubi, Legong, Manchong, Mandol, and Ulu Lui.)

186. Morley, D. C. Some notes on the health of the Semelai in Southern Pahang. *Bull. Raffles Mus.,* Series B, 4: 133-134, 1949. (About 200 people were studied after a food emergency occurred.)

186a. Morris, K. Forest utilization: commodity and subsistence among the Semaq Beri of Peninsular Malaysia. *Civilisations* 44: 194-219, 1997.

187. Mourant, A., A. Kopec, and K. Domaniewska-Sobczak. *The Distribution of the Human Blood Groups and Other Polymorphisms.* Ed. 2. Oxford University Press, NY, 1976.

(A world survey containing data on Orang Asli groups studied before 1976; see also Tills et al., 1983.)

188. Mummery, C. F. The teeth of the Che Wong. *Brit. Dental J.* 84: 69-72, 1948. (Caries increased with age; fever was common; 2 of 51 persons had yaws.)

189. Murugasu, R., F. Wang, and A. S. Dissanaike. Schistosoma japonicum-type infection in Malaysia–report of the first living case. Trans. *Roy. Soc. Trop. Med. Hyg.* 72: 389-391, 1978. (On an Orang Asli tuberculosis patient who had a rare form of schistosomiasis.)

190. Nei, M. Evolution of human races at the gene level. In: *Human Genetics*, Part A: The Unfolding Genome. B. Bonne-Tamir, ed. Alan Liss, NY, 1982. Pp. 167-181. (Includes data on Malayan "Negritos" and "aboriginal Malays.")

191. Nevin, H. Annual report of the Institute of Medical Research, Federated Malay States, for the year 1937. Pp. 145-147. (A health survey on Semai at Kuala Denak, Perak, revealed 85% of those less than 10 yrs. of age, but only 6% of adults, had malaria parasitemia; after 2 yrs. of anti-malarial treatment, only 5% of all ages had parasitemia; over 10%, all ages, had filarial infections and 74% had intestinal worms.)

192. Ng, M. S., K. Van, and J. Pala. Demographic situation of the aborigines in Malaysia. Dept. of Statistics, Kuala Lumpur, 1987 and Quarterly Review of Malaysian Population Statistics, No. 18, pp. 9-18, 1992. (Cited on p. 13 in: *Indigenous Minorities of Peninsular Malaysia*, Razha Rashid, ed., Intersocietal and Scientific, Kuala Lumpur, 1995. The crude death rate median values for 1984-1987 were 10.4 for all Orang Asli combined vs. 5.2 for all Malaysians; synchronic infant mortality rates were 51.7 vs. 16.3, respectively.)

193. Nicholas, C. Medicines are for curing, not killing. *Aliran Monthly* 17 (3): 7-8, 1997. (Criticizes the malaria-control program for Orang Asli, following the deaths of Jah Hut children in February, 1997, upon being administered anti-malarial drugs.)

194. Nicholas, C., A. Williams-Hunt, and Tiah Sabak. *Orang Asli*

in the News; the Emergency Years: 1950-1958. Center for Orang Asli Concerns, Petaling Jaya, Malaysia, 1989.

195. Noone, H. D. Report on the settlements and welfare of the Ple-Temiar Senoi of the Perak-Kelantan watershed. *J. Federated Malay States Mus.* 19: 1-85, 1936. [Describes stereotypes of physical appearance of Orang Asli groups and of Malays; appendix III, p. 80, mentions yaws in the Temiar and skin diseases uncommon in them but common in the nomadic "Negritos;" the Temiar had no goiter; appendix IV, pp. 81-83, discusses various ills found in Orang Asli in Ulu Plus, especially malaria and skin diseases; appendix V, p. 84, presents a rough census of "Plus" for 1931; appendix VI, p. 85, gives estimates of infant mortality rates (29-45%) and other demographic data.]

196. Noone, H. D. Some vital statistics of the Lowland Senoi of Perak. *J. Federated Malay States Museums* 15: 195-217, 1939. (Sparce demographic data.)

197. Norazah, A., et al. Streptococcal impetigo among aboriginal children in Malaysia. *Southeast Asian J. Trop. Med. Pub. Hlth.* 26 (4): 803-804, 1995.

198. Norhayati, M., et al. Hookworm infection and reinfection following treatment among Orang Asli children. *Med. J. Malaysia* 50 (4):314-319, 1995. (Among 193 Temuan children, 31% had hookworm infections; only 8% had them after 4 mo. of treatment, but reinfection rates were high.)

199. Normaznah, Y., et al. Seroprevalence of Sarcoptes scabiei var. canis antibodies among aborigines in Peninsular Malaysia. *Southeast Asian J. Trop. Med. Pub. Hlth.* 27 (1): 53-56, 1996. (On Orang Asli at Gombak Hospital.)

200. Ogilvie, C. Che Wong word list and notes. *Bull. Raffles Mus.* Series B, No. 1, p. 11, 1949. (Reports two-thirds of the Che Wong died as a result of the 1918 influenza pandemic, according to Polunin, 1953.)

201. Ong, H. C. Hematological values in pregnancy in Orang Asli (Aboriginal). *Med. J. Malaysia* 27: 240-242, 1973a. (At Gombak Hospital, 26% of pregnant Orang Asli women of unspecified ethnic group were anemic; N=278.)

202. Ong, H. C. Vaginal candidiasis and trichmoniasis in pregnancy. *Asian J. Med.* 9: 93-95, 1973b.

203. Ong, H. C. Hemoglobin E variants and pregnancy in Malaysian aborigines. *Acta Haematologica* 52 (4): 220-222, 1974a. (On 126 pregnant women at Gombak Hospital; found 48% of Hb E women were anemic; no statistical tests given; other genetic factors not assessed.)

203a. Ong, H. C. Anemia in pregnancy in an aboriginal population. *J. Trop. Med. Hyg.* 77: 22-26, 1974b. (On Gombak Hospital patients.)

203b. Ong, H. C. Maternal and fetal outcome associated with hemoglobin E trait and hemoglobin E disease. *Obst. and Gynecol.* 45 (6): 672-674, 1975a.

204a. Ong, H. C. Migration in Malaysian aborigines: clinical observations in pregnancy. *Southeast Asian J. Trop. Med. Pub. Hlth.* 6 (3): 407-412, 1975b. (On Gombak Hospital patients.)

204. Ong, H. T. Medical services for the Orang Asli (Aborigines) of West Malaysia. *Med. J. Malaysia* 30: 30-37, 1975.

205. Ong, H. T. Total health care for the Orang Asli (Aborigines) of Malaysia. Jabatan Hal Ehwal Orang Asli, Kuala Lumpur. 1976.

206. Onyah b. Itam. Filariasis among Malayan aborigines examined at the Gombak Hospital during the period 1961-1966. *Med. J. Malaya* 21: 384-385, 1967. (This paper reports an average of 11% filariasis among Orang Asli groups, with N=1964. It is probably the first biomedical report published by an Orang Asli. Note: the correct spelling of the author's name is Unyah.)

207. Ooi, W. L. Red cell polymorphisms and malaria in Malaysia. Yale University, New Haven, CN, Masters of Public Health thesis, 1979. (A small survey on Duffy blood group and hemoglobin E in a combination of Orang Asli and others; results were inconclusive; see Lewis et al., 1988, for correction about the "finding" of Duffy-negative phenotypes.)

208. Orang Asli Studies Newsletter, Kirk Endicott, ed. Department of Anthropology, Dartmouth College, Hanover, NH 03755,

USA. (Contains articles and periodic bibliographies on various aspects of Orang Asli life.)

209. Osman, A. The relationship between malnutrition and endocrine disorders among Malays and Aborigines in Malaysia. Ph. D. thesis, Universiti Kebangsaan Malaysia, 1992.

210. Osman, A. Iodine deficiency disorders: a public health challenge in developing countries. *Nutrition* 11 (suppl. 5): 517-520, 1995. (A review.)

211. Osman, A., Zarina Shamsuddin, and B. A. K. Khalid. A socioeconomic, social behavior, and dietary pattern among Malaysian Aborigines and rural native Malays. *Med. J. Malaysia* 46: 221-229, 1991. (On Kuala Pangsoon Temuan, in Selangor.)

212. Osman, A., et al. Protein energy malnutrition, thyroid hormones and goiter among Malaysian Aborigines and Malays. *Asian Pac. J. Clin. Nutr.* 1: 13-20, 1992. (On Kuala Pangsoon Temuan, in Selangor.)

213. Osman, A., et al. Prevalence of NIDDM and impaired glucose tolerance in aborigines and Malays in Malaysia and their relationship to sociodemographic, health, and nutritional factors. *Diabetes Care* 16 (1): 68-75, 1993a. (Among Bukit Lanjan Temuan in Selangor and Lanai and Betau Semai in Pahang, only 1 of 321 testees had diabetes.)

214. Osman, A., et al. Serum thyroid stimulating hormone (TSH) in malnutrition: preliminary results. *Singapore Med. J.* 34: 225-228, 1993b. (On 26 Temuan children in the Kuala Pangsoon area of Selangor.)

215. Osman, A., et al. Promoting community participation in determining prevalence of malnutrition, goitre, and diabetes mellitus: Malaysia's experience. *J. Perubatan* UKM (Malaysia) 15 (2): 105-115, 1993c. (Studied Semai at Betau and Lanai, Pahang, and Temuan at Bukit Lanjan, Selangor; overall, 72% of the 2 to 6 yr. old Orang Asli were stunted, indicating chronic malnutrition; for 675 Orang Asli over the age of 2 yrs., 37.8% were goiterous.)

216. Osman, A., et al. The effect of cassava leaf uptake on thyroid hormone and urinary iodine. *E. African Med. J.* 70: 314-315, 1993d. (Experimentally, Orang Asli showed a decrease in thyroid function after 12 days of a diet rich in cassava leaves.)

217. Osman, A., et al. Iodine content in drinking water not an important determinent of endemic goiter. *Asian Pac. J. Clin. Nut.* 2: 115-118, 1993e. (On Lanai and Betau, Pahang, Semai and Bukit Lanjan, Selangor, Temuan.)

218. Osman, A., et al. (published as O. Ali et al.) Thyroid function and pubertal development in malnutrition. *Ann. Acad. Med. Singapore* 23 (6): 852-855, 1994. (207 Orang Asli were studied, location not identified; poor nutrition was found to affect thyroid function, including goiter formation, as well as growth hormone levels in children.)

219. Osman, A., Khalida Muda, and B. A. K. Khalid. Iodine content in urine samples among Malays and aborigines. *Acta Med. Okayama* 48 (6): 289-292, 1994. (On Post Lanai and Post Buntu, Pahang.)

220. Osman, A., et al. The prevalence of goiter in remote inland versus coastal areas. *Med. J. Malaysia* 50 (3): 256-262, 1995a. (Mahmeri on Pulau Carey and Kensiu, or perhaps Kinta, at Kg. Lubuk Legong, Baling Dist., Kedah, were studied; 6% of Mahmeri and 30% of Lubuk Legong people had goiters, despite the fact that the iodone level in drinking water in the Kedah area was "surprisingly high.")

221. Osman, A., et al. Endemic goiter and hypothroidism in Orang Asli and Malays in Peninsular Malaysia. *Med. J. of the Islamic Republic of Iran* 9 (1): 19-25, 1995b. (On Lanai and Betau, Pahang, Semai and Bukit Lanjan, Selangor, Temuan.)

222. Osman, A., and Zaleha Md. Isa. Nutritional status of women and children in Malaysian rural populations. *Asian Pac. J. Clin. Nutr.* 4 (3): 319-324, 1995. [Studied Semai at Betau and Lanai, N=343; 80% of 2-6 yr. olds and 35% of women over 17 yrs. of age were malnourished. For Betau vs. Lanai children: 80% vs. 60% had protozoan infections; 30% vs. 8% had roundworms, 30% vs. 16% had threadworms, and 9% vs. 0% had hookworm. Goiter generally increased with age, from 23%

for 2-12 yrs. of age to 50% for older ages, with a 2 to 1 bias toward goiter in females. Cassava and millet were mentioned as local goitrogens.]

223. Osman, A., et al. Levels of thyroxine, TSH, thyroid volume, and mental performance among Orang Asli in selected settlements in Malaysia. *East African Med. J.* 73 (4): 259-263, 1996. (Mental performance was statistically the same in all locations.)

224. Osman, A., et al. Blood glucose and glycosylated hemoglobin in Malays and aborigines in Malaysia. *Med. J. Malaysia* 51 (2): 179-187, 1996. (On Lanai and Betau Semai and Bukit Lanjan Temuan.)

225. Petrakis, N., et al. Evidence for a genetic cline in earwax types in the Middle East and Southeast Asia. *Amer. J. Phy. Anthropol.* 35: 141-144, 1971. (Reports 7% of Orang Asli had dry ear wax; see also Baer et al., 1976.)

225a. Pillay, M. R., H. Frank, and J. Ponnampalam. Malaria antibody titres as measured by the indirect fluorescent antibody test in relation to parasitemia and treatment. *Southeast Asian J. Trop. Med. Pub. Hlth.* 12 (1): 111-113, 1981. (Studied 39 Orang Asli malaria patients from "deep jungle.")

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Susan Pernia

Temiar women waiting for visitors to purchase their handicrafts, Gua Musang, Kelantan.
The ills of 'civilization' – poverty, drug addiction, alcoholism, suicide, hypertension and so on – are likely to increase in Orang Asli enclaves over time. Beyond medical strategies, cultural factors are the most important guarantee for the future health and survival of the Orang Asli. If culture is a kind of blueprint for survival, then the dissolution of a culture may well be a blueprint for disaster. And such dissolution may already be happening.

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The Orang Asli comprise 18 ethnolinguistic groups on the Malaysian peninsula. The cultures and biological settings of the Orang Asli are unique and fascinating.

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A Biomedical and Genetic Analysis of the Orang Asli of Malaysia

Thus the past and present circumstances of the Orang Asli have long interested scholars and scientists.

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