SOME EPIDEMIOLOGICAL CONSEQUENCES OF DRASTIC ECOSYSTEM CHANGES ACCOMPANYING EXPLOITATION OF TROPICAL RAIN FOREST

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It is likely that future generations will decide in retrospect that the replacement of long-established natural ecosystems by ones having man as the dominant species was not ultimately beneficial. For sound economic reasons (Schmithusen, 1976), the intrusion by man into moist tropical forest is normally accompanied by clearance and export of trees. However, whether intrusion is required for this reason or to facilitate major engineering projects such as the Panama Canal, the activities justify road-building and general acquisition of more human living space. In consequence, agricultural extension is needed and in this way the interfacial zone where man confronts a hostile and alien environment is increased dramatically. Drastic ecosystem change will perforce have immediate consequences for the indigenous community but these have rarely been assessed. Study is usually justified when the effects are reflected in the wider context. Typically human mortality is the factor which prompts research investment and scientific investigation is most rapid when urban rather than rural populations are involved. Thus, although it has long been known that European visitors to tropical rain forests are subject to brief periods of fever often accompanied by rash, myalgia and general malaise, only in the last twenty years or so were some of the causes identified. In general, viruses were implicated : mycotic and bacterial infections seem not to pose special problems either to the native or to the non-indigenous human rain forest populations. The information currently available suggests that drastic changes in the ecosystem will most probably facilitate human infection by viruses, nematodes and possibly protozoa. The epidemiological consequences have been considered in approximate proportions to the anticipated risks : the potential harm from viruses seems greatest.

The purpose of this paper (which is based on a contribution to a U.N.E.S.C.O./M.A.B./I.U.F.R.O. Tropical Rainforest Ecosystem workshop, Hamburg, May, 1977) is to appraise readers of some of the ecological factors influencing pest/pathogen spread in these regions and to alert them to some epidemiological consequences of exploitation.

Epidemiology is the study of the distribution and determinants of disease or delibitation. On etymological grounds, the term epidemiology (Greek *demos*) should properly be restricted to people. However, the words "epiphytotiology" (Robinson, 1969) and "epizootiology" (Steinhaus and Martignoni, 1970) relating specifically to plant and animal diseases are tongue twisting and have never been widely used. In any event, in the context of this paper, parasites and pathogens of wild animals are inextricably linked with human diseases. Furthermore, it seemed appropriate to include, albeit briefly, plant pests and diseases because if the yields of agricultural crops are affected then in turn losses in food have direct effects on human nutrition.

CONSEQUENCES FOR THE NON-INDIGENOUS COMMUNITY NOTABLY MAN AND HIS CROPS

The study of parasitic worms and protozoal infestations to which man is subject in the tropics has progressed more or less rapidly since 1879 when Manson described filaria developing within mosquitoes. The requirement for costly and sophisticated equipment to identify viruses has however resulted in disproportionate knowledge ; much more is known about viruses present in temperate regions of Western Europe and North America than those important in the developing countries of the tropics. Priority has generally been given to the study of human pathogens and economics dictated that limited resources were allocated in the first instance to diseases causing either high mortality, e.g. yellow fever (Boshell, 1955, Haddow *et al.*, 1947), haemorrhagic dengue (Hammon *et al.*, 1960 ; Halstead *et al.*, 1970) or high morbidity, e.g. filariasis (Cheng, 1964 ; Lavoipierre, 1958 ; Laing, 1960). Although research was initiated in relation to urban populations it soon became apparent that man was not the only source of the pathogens concerned. When attention was directed to the land surrounding the settlements, numerous viruses, malarial protozoans and filariae were identified, often in circumstances where no overt disease was known. In the tropical rainforest, parasites and pests are present at all levels in the vegetation, in the fauna and most significantly in the arthropods. However as in all well established plant or animal communities, diseases are rarely obvious presumably because the most damaging patho-

gens and the most sensitive hosts have been eliminated. When man intrudes, he is exposed to a diverse array of diseases caused by pathogens including roundworms which cause lymphatic obstruction and elephantiasis, protozoa (e.g. malaria) and viruses. Of the viruses present (and they are ubiquitous, infecting : Bacteria, Cyanophyta, Protozoa, Chlorophyta, Arthropoda, Nematoda, Mollusca, Chordata, Eumycophyta, Bryophta, Pteridophyta and Spermatophyta and being transmitted by Nematoda, Arthropoda, Chordata, Eumycophyta and Spermatophyta) the greatest hazard is likely to be presented by a group known as arboviruses (see Andrews and Pereira, 1967 ; Chamberlain, 1968). Arboviruses liable to cause animal disease replicate in and are chiefly transmitted by mosquitoes or ticks. Though filarial worms, malarial plasmodia and arboviruses are very different, all depend for biological continuance upon transmission between at least two types of host : invertebrates which feed on blood and chordates. Thus the causal agents of brugian filariasis (*Brugia malayi*) are known to infest monkeys, domestic and wild cats, civets and pangolins in addition to man (Laing *et al.*, 1960 ; Cheng, 1964) and the list of known vertebrate hosts of other filarial parasites includes most mammals as well as birds, lizards, snakes and frogs. Consequently, even when the human population is treated to minimize spread, there is always the threat of reintroduction from wild reservoirs.

The same applies to trypanosomes and malarial parasites, yet the possibility was not seriously considered before 1960. Taking malaria as an example, some *Plasmodium* species are known to infest blood of apes, monkeys, rodents, birds, lizards and frogs (e.g. Baker, 1969). Although other alternate hosts have not been implicated with certainty, it is now known that human malarias infect chimpanzees and simian malarias infect man (Coatney, 1963 ; Warren and Wharton, 1963).

Awareness of the importance of wild sources of infection was largely created as a consequence of detailed virological research sponsored since about 1916 by The Rockefeller Foundation. Following studies in the Neotropics, work was concentrated in West Africa and, with the aim of assessing levels of immunity in the human population and confirming the concept of inapparent infection, activity was extended to the drier borders of the Congo in Uganda and more easterly regions of Africa (Haddow *et al.*, 1947 ; Hughes *et al.*, 1941). The complementary studies in Africa and in the Americas established many of the basic tenets on which epidemiology has subsequently been based and, in particular, revealed the importance of wild animals as reservoirs of viruses affecting man in and near the tropical rainforest (e.g. Smithburn *et al.*, 1949 ; Haddow *et al.*, 1947 ; Lumsden, 1952 ; Lumsden, 1955 ; Smithburn and Haddow, 1949 ; Soper, 1967). The

techniques evolved during these surveys were subsequently applied to other viruses having mosquito vectors such as those with reservoirs in the avifauna as well as rodents and other mammals, e.g. equine encephalomyelitis, Japanese B encephalitis (see Scherer *et al.*, 1959 ; Andrews, 1967 ; Jonkers *et al.*, 1968 ; McIntosh *et al.*, 1964 ; Reeves, 1965). These and other searches lead to the discovery and description of numerous agents which were potentially pathogenic in man but were not associated with overt disease.

After World War II, tick-borne virus infections were investigated with most vigour but although about 70 viruses are now known to have tick vectors (Hoogstraal, 1973a & b) much of the world has not yet been explored and it is likely that a great many more await discovery. The intricacies of complex local biology which interact to influence the epidemiology of tick-borne viruses have only rarely been elucidated and there is a similar paucity of data concerning the ecology of plant viruses. Even in temperate regions of the world where most research is done, the ecology of plant viruses in natural plant communities has until recently been neglected. Consequently, when attempting to extend discussion to include harmful effects of drastic ecosystem change on plants in the tropical rainforest, information has been very scarce ; mostly restricted to one forest crop, *Theobroma cacao* L.

To illustrate some of the interactions involved when man intrudes into the rainforest, we have used three well documented examples referring where appropriate to other pertinent data. Knowledge about yellow fever shows the risks to man the woodcutter, data concerning Kyasanur Forest disease illustrate some of the factors which act when man impinges on the centre of a tick-borne virus and a few of the parameters adversely influencing his agricultural activities are revealed with special reference to the cocoa crop.

YELLOW FEVER

As shown in Fig. 1A, yellow fever in South and Central America is a lethal disease of monkeys and marmosets between which the virus is spread by mosquitoes, notably *Haemagogus spezzinii* Brethes and to a lesser extent *Aedes leucocelaenus* Dyar and Shannon. These insects, like the primates they pester, are for the most part arboreal. Very probably, there are additional cycles of infection possibly involving marsupials or rodents and other vectors. However, when the forest trees are cut down, man is exposed to clouds of mosquitoes from the canopy, is bitten and is liable to infection with yellow fever. About 5 days following infection in man and somewhat less in monkeys, virus is present in the peripheral blood and remains at a high concentration for two or three days whether disease is overt or silent. Thus, virus
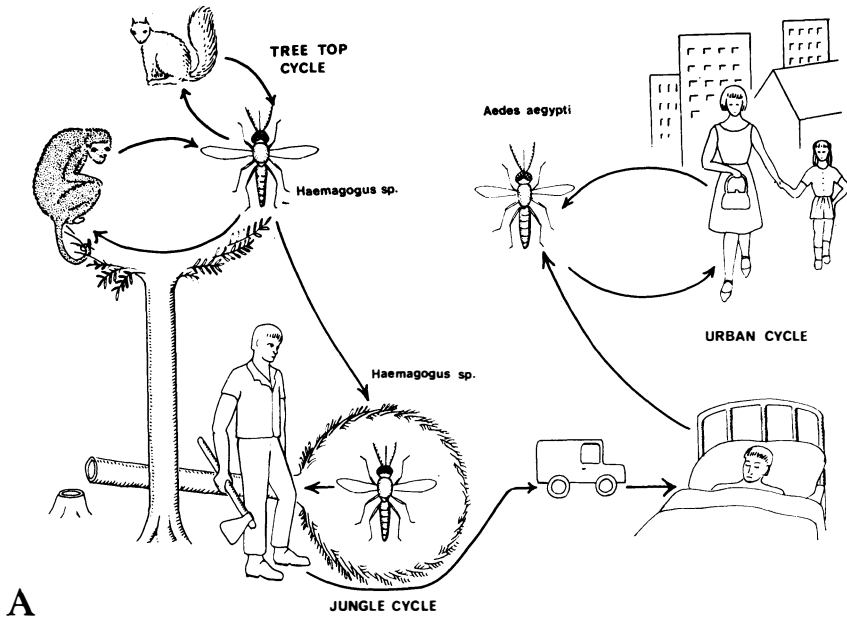
Fig. 1A. — The role of wild animals (primates plus possibly marsupials and rodents) as reservoirs of yellow fever virus in South America. Forest workers infected with virus carried by *Haemagogus* mosquitoes may bring infection to villages where the virus is disseminated by other vectors (notably *Aedes aegypti*).

is accessible to other feeding mosquitoes but only briefly and the ambulant human patient typically serves as a link connecting the forest with his urban environment where mosquitoes of other species and genera, particularly *Aedes aegypti* L., may abound in all domestic rubbish containing water.

In Africa, the situation is different (Fig. 1B). The wild primates, not man, act as porters of infection between the sylvan and urban environments. However, mosquitoes are also involved as vectors and monkeys are prime maintainance hosts of the virus in this continent too. Thus, it is well established that, in Africa, Colobus monkeys in particular host the virus in the tree tops where *A. africanus* act as a prime vector. However, both cohabit with more venturesome primates, e.g. red tailed guenon *(Cercopithecus ascanius schmidti)* liable to invade human settlements to feed on banana or other crops (Haddow *et al.,* 1947). Here, the monkeys are exposed to *A. simpsonii* (Theobald) which breeds in leaf axils of plants and is therefore well placed to spread virus to man who will return to his urban surroundings where *A. aegypti* is again the most frequent vector. It may well be that with the
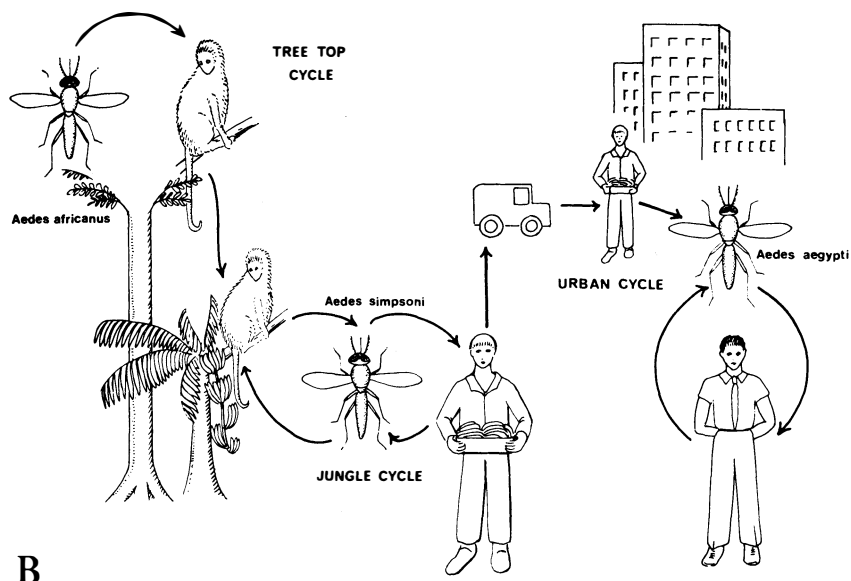
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B

Fig. 1B. — The role of wild animals (chiefly primates) as reservoirs of yellow fever virus in Africa. In the forest, monkeys maintain the virus and *Aedes africanus* is an important vector. This mosquito may infect man directly but the virus is usually introduced to urban man in a different way. Monkeys feeding in periurban plantations are exposed to *Aedes simpsonii* which thereafter facilitate dispersal within the human population. *Aedes aegypti* is in Africa liable to be an important virus vector both in the forest and in villages.

expansion of timber industries in Africa, *A. africanus* (Theobald) will be more frequently able to inoculate man directly.

KYASANUR FOREST DISEASE

It took an abnormally high mortality in monkeys to justify investigation of the epidemic of tick-borne virus which came to be known as Kyasanur Forest Disease (KFD) after the region in Mysore, India, where it occurs. In contrast to yellow fever which does not seem indigenous in Asia, but is prevalent over the African and South American continents, KFD occurs in a very circumscribed area a few kilometers across. The associated virus has, however, affinities with a range of viruses associated with marshy scrub from Europe to Asia, where they cause summer encephalitis in foresters and woodcutters (Work, 1958). Following a population explosion among the human inhabitants, 116 % increase is reported in the preceding decade, KFD appeared as an appa-

rently wholly new disease. It was rapidly established that the greatly increased human population had consequential effects on the sylvan environment due to increased cattle and food crop production, coupled with the additional need for structural timber/firewood (Boshell, 1969). Human incursion into the rainforest was also necessary to obtain green manure for betelnut (*Areca catechu* L.) cash crops and this fertilizer/mulch was obtained by pruning forest vegetation. Land cleared in this way was rapidly invaded by *Lantana* spp. (Verbenaceae) ; plants which had been introduced into Ceylon from tropical America for ornamental planting but which 'escaped' and now form dense, sometimes impe-
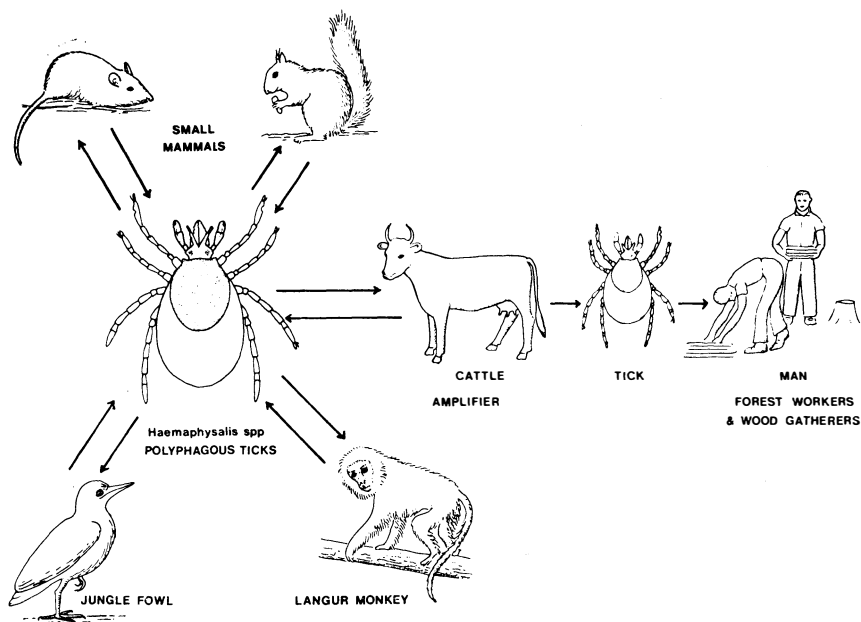

Fig. 2. — The roles of wild and domestic/commensal animals as reservoirs of Kyasanur forest disease virus. Human infection is consequent upon three interacting factors. I) exploitation of forest by man and his animals ; II) invasion of cleared forest by *Lantana* sp. providing cover and food for additional small mammals and birds ; III) greatly increased numbers of polyphagous blood-sucking tick vectors (*Haemophysalis* spp.) exploiting the augmented fauna. Cattle and goats amplify the vector population, acting also as "porters" facilitating vector contact with human hosts.

netrable, thickets harbouring dispossessed refugees from the original forest fauna. Research revealed that the human and indeed the monkey infections were likely to have been mere indicators of an inapparent infection of rodents and jungle fowl (Fig. 2).

The lantana provided cover and food for numerous birds such as *Gallus sonneratii* and small mammals notably mice *Mus booduga*, shrews *Suncus murirus* plus probably to a lesser extent *Rattus* spp. Given the changed conditions, pests prospered and polyphagous blood-sucking Ixodid ticks particularly *Haemophysalis* spp. rapidly exploited the new habitat with its augmented forest floor fauna. Man introduced additional factors into the already complex situation ; foraging cattle and goats provided further sources of blood and amplified an already massive tick infestation (Fig. 2). These animals acted as porters of virus-carrying vectors which were thereby given the opportunity to feed on human blood. It is probable that in this locality the ground fauna acted as the main reservoir of both virus and vector ; arboreal monkeys may have had infection brought to them by langur *(Presbytis entellus)* and other primates e.g. *Macaca radiata* which spend part of their lives on the ground (Sugiyama, 1964).

PLANT PATHOGENS AND PESTS

The importance of the botanical habitat was introduced by reference to the epidemiology of KFD. Although the biology of plant pathogens is generally very poorly understood Bennett, (1952) concluded a survey of the literature then available with the observation that most epidemics of plant viruses have occurred in the newer agricultural regions. This seemed to imply that the viruses moved from wild plants to cultivated ones. In the tropical rainforest most is known about pests and pathogens of cocoa (*Theobroma cacao* L.).

Although *T. cacao* will grow well and yield best in the absence of shade, the crop is usually cultivated either beneath a diverse array of natural forest trees or under monospecific planted trees. When grown under 'natural shade' the agrosilvicultural system remains essentially tropical rainforest — albeit drastically modified by man. Typically, the pre-existing forest is thinned to leave 4 or 5 dominant and about 50 understory trees/ha. The unwanted timber is felled or burned.

In 1936, a severe disease 'swollenshoot and dieback' was recognised in Ghana (Steven, 1936). In 1938, Posnette showed that the disease was transmissible and by implication caused by a virus (Posnette, 1940). Somewhat later Cotterell (1943) demonstrated that 'the virus' was transmitted when mealy bugs (Homoptera : Coccoidea) feed first on infected plants then on healthy. Although branch and chupon symptoms are characteristic, disease is often apparent in foliage. in roots, in the fruit and most dramatically in the yield of dried cocoa. In one series of observations (Crowdy and Posnette, 1947) the disease caused yield to be reduced by 25,

50 and virtually 100 % in the crops succeeding experimental infection : after 3 years, most infected trees were dead. In these circumstances, several factors contribute to the death of cocoa ; the viruses are not always themselves lethal. Thus, when foliage dies, trees are exposed to increased light with consequent diminished humidity. This favours infestation by polyphagous insects particularly capsids (Miridae : Heteroptera) which feed on the plants (see Entwistle, 1972 ; Johnson, 1962) thereby facilitating infection with fungi, notably *Calonectria rigidiuscula* (Berk. + Br.) Sacc. (Crowdy, 1947 ; Usher, 1962).

Two well characterized groups of viruses have been described from cocoa ; cocoa yellow mosaic (Brunt, 1970a) and a complex of isolates more closely associated with the swollen shoot/mottle leaf syndrome (Brunt, 1970b).

TABLE I

Some properties of cocoa viruses

	Cocoa yellow mosaic	Cocoa swollen shoot/ mottle leaf
Known distribution	Sierra Leone	Central and South America, West Africa, Ceylon, Sabah?
Vector	beetle ?	mealybug
Persistence	not known	15 min/2h to two days
Particle	spherical c30nm	bacilliform 130 × 30nm
Experimental hosts	9 plant families	4 plant families
Known natural hosts	*Theobroma cacao* *Cola nitida* Schott. et Endl. *Culcasia scandens* (Willd.) P. Beauv.	*Theobroma cacao* *Cola chlamydantha* K. Schum. *Cola gigantea* Brenan et Keay *Ceiba pentandra* Gaertn. *Sterculia tragacentha* Lindl. *Adansonia digitata* L.

As shown in Table I, the associated virus particles differ in size, shape, vector and experimental host range but have been detected in elements of the natural flora of West Africa. Consequently, in Africa, there are at least two known sources of viruses : cocoa and adjacent native forest trees belonging to the related Tiliales and Malvales (e.g. Todd, 1951 ; Tinsley & Wharton, 1958). Apart perhaps from *Ceiba pentandra* Gaertn. and *Cola* spp. which

may be retained as shade and because of the valuable timber, wild hosts of the viruses probably represent a minor source of inoculum. However, when infected, these native hosts contain virus available to vectors for months or years and in this respect resemble perennial (chordate) hosts of filarial and malarial parasites.

Studies made in Papua/New Guinea show that through causing injudicious and drastic changes in the moist tropical forest, man may also greatly increase risks to his crops from other insects. Indeed there are parallels to be drawn between the factors contributing to the KFD epidemic and those implicated in the local devastation of cocoa caused by plagues of defoliating cocoa army worms (*Tiracola plagiata* Walk.). *T. plagiata* is highly polyphagous (Entwistle, 1972) and frequent in a zone including India, South East Asia and Australia. Though capable of causing damage to many crops, it is normally considered a relatively unimportant pest. However, in 1960, coincident with the planting of 3000 ha of cocoa, extensive areas of rainforest were clear felled and burned. Weed species rapidly became established and were soon heavily infested with *T. plagiata*. *Leucaena glauca* Benth. shade trees were planted to maintain a humid microclimate around young cocoa but unfortunately proved acceptable hosts on which insect populations rapidly exploded. The consequence was devastation (Dun, 1967) probably brought about because felling and burning created an area in which parasites and predators which normally check *T. plagiata* populations were few or absent.

The planting of monocultures generally facilitates the spread and exacerbates economic effects due to pathogens. The scale of possible consequences can be indicated by reference to a fungal disease of rubber spread chiefly via airborne propagules (conidia). Leaf blight caused by *Microcyclus ulei* (P. Henn.) v Arx is an endemic disease on *Hevea* spp. in tropical South America, and is the most important factor limiting the extent of commercial rubber planting (Hilton, 1955 ; Holliday, 1970). At least four species of the genus *Hevea* are susceptible to natural infection but, of these only *H. brasiliensis* (Willd. ex Adr. de Juss) Mull. Arg. is commercially significant. Indeed, the history of commercial natural rubber is bizzare ; virtually all comes from trees selected in South East Asia following a single seed introduction, seemingly by chance originating from an area where only the best yielding species, *H. brasiliensis,* occurred. (Purseglove, 1968).

In the first half of the twentieth century, numerous attempts were made to cultivate rubber in South America using material imported from and selected in South East Asia. The consequences were disastrous because the reintroduced plants were all extremely sensitive and susceptible to the fungus which defoliated then killed the trees. Commonly, the plantations were established

following clearance and were therefore surrounded by natural forest in which scattered trees infected by *M. ulei* occurred. In these circumstances it took about 5 years for significant amounts of infection to spread from the wild trees. Thus, in Surinam during a seven year period, the fungus killed more than 10,000 trees and in Costa Rica only 10 % of an initial stand of 36,000 trees remained after the epidemic spread of the pathogen.

Less drastic, but nonetheless ill-conceived modifications wrought by man on the moist forest are also liable to upset the equilibrium which Garrett (1956 ; 1970) suggested operates between fungal root parasites and natural forest vegetation.

When forest trees are only partially cleared, the decaying timber and stumps are rapidly colonized by a wide range of fungi of which species pathogenic to introduced plantation crops form only a fraction. However, it is well known that the process augments sources of *Ganoderma* spp. (Turner, 1965) and *Armillaria mellea* Vahl. ex. Fr. (Wardlaw, 1950) which kill trees irrespective of genus. These and other root infecting fungi occasionally devastate oil palm (*Elaeis guineensis* Jaq.) and in these conditions *Fomes lignosus* (Klotzsch) Bres. also kills rubber *(Hevea brasiliensis)* on a similar large scale (Depoerk, 1946 ; Fox, 1969). Strangely, root infecting fungi are relatively minor pathogens in cocoa. However, Warton (1962) suggested that in Ghana, cocoa usually escaped epidemic spread of *A. mellea* because general felling was rarely practised, seed being planted under natural forest conditions. The gradual and selective thinning of understory forest trees and necessary clearing of undergrowth which was done when the cocoa was established presumably created few food bases from which the fungal pathogens could spread rapidly.

CONSEQUENCES FOR THE INDIGENOUS COMMUNITY

Until recently, the effects of massive ecosystem change on climate as well as the survival, breeding and species composition of both plant and animal populations were but rarely considered. However, it is known that the removal of cover, nesting sites and probably food locally affects the distribution and abundance of vertebrates and in particular birds (McClure, 1968), rodents and bats (Harrison, 1968). Man-made changes in microclimate and normal host distributions are also more or less rapidly reflected in insect species composition and habits. In particular, clearance of vegetation removes some of the habitats where blood-sucking animals, notably mosquitoes, ticks and leeches can breed and contact their normal vertebrate hosts. When this happens, the biting behaviour is likely to change so that man or his domestic/ commensal animals become the main blood source. This seems to

have occurred when malaria and its "normal" mosquito vector *Anopheles darlingi* Root were virtually eradicated from regions of Guyana to be replaced by *A. aquasalis* Curry which took over as the principal agent of *Plasmodium* transmission (Gigioli, 1963). In this instance immigrant workers were the most likely source of protozoa for the new epidemic and, it is pertinent to note, that there are numerous reports (e.g. Ackerknecht, 1965) showing that man is a significant carrier of diseases which, following his intrusion, wreak havoc with the indigenous population.

The impact of man is rapidly harmful to ephemeral insects such as mosquitoes but benefits gained are usually of short duration. Despite the brief life span of individuals in any one mosquito species, the numbers of microhabitats available and the diversity of mosquitoes adapted to each specialized environment ensures that there is a continuity of supply. Thus, although forest clearance may temporarily remove the species having larvae which infest ground water pools filled with rotting vegetation and others (notably *Haemagogus, Culex* and a few *Aedes* species) typically breeding in tree holes (Service, 1965) plus the few anophelines (e.g. *Kerteszia bellator* Dyar and Knab) breeding exclusively in epiphytic bromeliads (Pittendrigh, 1948) ; numerous new habitats are provided and rapidly colonized (Boyd, 1941). The most important vector of malaria in Africa, *A. gambiae* Giles breeds almost exclusively in earthlined man-made pools (from foot prints to bulldozer tracks) exposed to the sun. Similarly, the most important yellow fever vector *A. aegypti* rapidly exploits domestic rubbish containing water, tins, discarded tyres, etc.

Having further regard to human populations, it is known that a new group of infected/infested persons can have harmful effects on an immunologically unprotected community. These consequences may be disproportionate to the numbers of carriers initially involved if pathogen spread is readily accomplished in the new environment. Persistent infections such as malaria (e.g. Worsley, 1969) and infestations such as filariasis (Wilcocks and Manson-Bahr, 1972 ; Davis, 1976) are much more likely to be introduced into new areas by human carriers than are, for example, arboviruses associated with viraemia of typically short duration. However, isolated human communities may for a variety of reasons be unusually susceptible and sensitive to introduced pathogens generally and infections which are normally inconsequential in Europeans may be lethal in these circumstances. Thus, measles, which frequently causes mild disease in the developed world where food is plentiful, is much more severe and often fatal in malnourished children (Puffers and Serrano, 1973). Furthermore, influenza viruses, which, in developed countries where antibiotics and other forms of chemotherapy are available, are widely regarded as causing comparatively minor infections have severe

effects in populations without previous exposure (e.g. Brown *et al.,* 1966).

In the light of knowledge concerning yellow fever, it is tempting to infer that the virus with its most important urban vector *Aedes aegypti* was introduced to the Neotropics where it decimates primate populations, from Africa where it does not. Indeed, *A. aegypti* is now world wide because man has carried this insect with him (e.g. Gillett, 1971).

EPILOGUE

In animals, virus infections typically lead to long lasting immunity following transient illness ; alternatively, death results. Thus, viruses carried by ephemeral insects such as mosquitoes, if not transmitted through the insect's eggs, must usually be maintained by moving continuously through a susceptible population. It is likely that only the three or four greatest forests, notably those of Amazonia, provide sufficient continuity in space to support an endless search for non-immune hosts and in time to allow regeneration of the essential individuals which had not previously encountered any particular virus. Whereas mosquito-borne infections can only remain in situ if the vertebrate hosts reproduce at an adequate rate or if there is a continuous supply of new hosts provided in another way, tick-transmitted viruses do not need this continuity. The life-span of tick vectors is a year or more. Indeed, it may be greater than that of some of its hosts. Furthermore, blood feeds are not frequent. Additionally, the known tickborne viruses are often transmitted through their vector's eggs to infect progeny ticks and may also spread within populations of chordate maintenance hosts by tissue contact, in blood, in urine and in milk (e.g. Hoogstraal, 1973, a and b). Consequently, tickborne infections are typically persistent but confined to circumscribed, sometimes very restited ecological niches. Whereas in chordates, arboviruses are available in blood for subsequent transmission during only a few days, protozoal and malarial infestations are typically chronic causing in some instances long periods of distress and debilitation but rarely death. In this respect, they resemble virus infections in plants. Each has a long lived source of future infection which will spread if the appropriate carriers are available.

More than 150 infections with parasites transmissible in nature between human and other vertebrate animals are known to be zoonoses and animal reservoirs are certainly widespread in long established and complex communities such as only the extensive rainforests now provide. "In this age of jet planes and soon, of supersonic transport, the only way to prevent the old plagues, and

some new ones, from spreading from continent to continent and from country to country is to help the poorest nations of the world to reach such a level of economic and technical development that it will be possible for them to combat the evil at its source" (Dorolle, 1968). If the infections are carried from the tropical forests, the effects on immunologically unprotected persons are likely to be severe. Unknowingly, man has often facilitated the dissemination of vectors which have become established in temperate regions because suitable breeding conditions have been provided by irrigation or in other ways. Many hitherto unrecognised mosquito-borne arboviruses having natural reservoirs within avian populations could well become established in urban communities and be maintained there in new mosquito-vertebrate (not necessarily avian) associations (e.g. Surtees, 1971). The danger from tick-borne viruses seems at least as great because these viruses have efficient auxiliary means of dissemination augmenting the effects of bloodsucking vectors. In this respect, commensal rodents are likely to provide the most potent domestic reservoirs.

Although emphasis has been placed on the dangers from arboviruses, this is largely because what little is known of epidemiology in the tropics concerns this group. It is, however, tempting to speculate on the possible origins of viruses following hybridization of "new" with "old" parents. This phenomenon has been clearly implicated in the evolution of influenza viruses (see Stuart-Harris and Schild, 1976). There are puzzling, perhaps frightening, features concerning the occurrence of monkey pox in persons living in remote tropical forests where monkey is hunted for food (e.g. Henderson, 1976). This disease is caused by a virus related to but distinct from small pox, and the possibility that the two viruses could interact to produce a deviant against which the widely used vaccination procedures would be ineffective is at least thought provoking. This is only one of several enigmata. Another is Lassa (AR) disease (Frame, *et al.,* 1970) which has caused death and debilitating fever in Nigeria and Sierra Leone albeit involving only a few dozen people. The vectors and the maintenance reservoirs are unknown but it seems likely that the disease was merely a spill-over from epidemics involving wild animals as prime hosts. Marburg virus similarly raises unanswered questions although in this instance the immediate source of infection causing acute febrile illness and death in Europe was vervet monkeys (*Cercopithecus aethiops*) imported from Uganda for laboratory use (see Smith, 1971). The disease is usually fatal in monkeys and so it would seem unlikely that monkeys constitute maintenance hosts. Monkey infection with Marburg virus might well have occurred in transit when numerous monkey species were housed near other vertebrates. In any event, it is noteworthy that many natural virus infections of monkeys have been detected (e.g. Hull, 1968)

particularly in stocks imported for experimental use. Some, such as the herpes virus B from rhesus monkeys (*Macacca mulatta*) are typically lethal for man and it is pertinent to note that thousands of monkeys are used each month for a range of laboratory tests. These collections of experimental animals are likely to deplete significantly the wild stocks and to increase dramatically the possibilities for dissemination of parasites and pathogens which the natural populations have been selected to tolerate.

Arthropods have been on earth for some 50 million years longer than mammals, have acquired a great many parasites during this period and have come to tolerate most. By contrast primates reproduce very slowly. Consequently they are likely to take hundreds of years to come to terms with any agent to which they are newly exposed. In the process, many deaths would probably occur.

Agricultural enterprises in tropical rainforests are equally liable to be affected by indigenous pest/pathogen problems. From these local foci of typically inapparent infection, man has carried a great range of pathogens and pests to infest other regions of the world. The potato (*Solanum tuberosum* L.) probably best illustrates man's influence. Thus, all but one or two of the 30 known potato viruses, arguably the most damaging fungus (*Phytophtora infestans* de Bary) and the chief nematode pest of temperate regions (*Heterodera* spp.) were disseminated with planting stock from the Andean regions of South America to all countries in which *S. tuberosum* is now grown for food (e.g. Large, 1940 ; Salaman, 1949 ; Jones and Jones, 1969).

However, having regard to a more typical crop of the moist tropics, banana (*Musa* spp.) provides, with the fungus *Fusarium oxysporum* s. sp. *cubense,* Smith, Snyder and Hansen, an appropriate example. Man unknowingly moved *Fusarium* in planting stock to wherever Gros Michel bananas were grown commercially (see Stover, 1969). Susceptible bananas uniformly died and propagules of the causal agent remain in soil for many years effectively preventing replanting with this crop. Significantly, the fungus is prevalent but symptomless in a range of wild grasses and in herbs of the tropical rainforest (Waite and Dunlop, 1953).

Data concerning the origins of viruses of *Theobroma cacao* are not unequivocal. Tinsley (1971) suggests that the evidence to support the idea that the viruses of cocoa may perhaps have been brought from the New World to West Africa with the planting stock was just as plausible as the viruses being of West African origin. His speculation was based on the knowledge that in Africa no native members of the genus *Theobroma* are known and that virus infected wild hosts in West Africa seem discontinuously distributed and are not consistently associated with the occurrence of disease in the crop. Dale (1962) suggested that infection of

rainforest plants originated in other hosts, e.g. *Adansonia digitata* L., having different normal geographic distributions within Africa.

Large scale interference with the environment is probably essential if the food needs of the world's escalating population are to be satisfied. However, the greatest care should be exercised when man exploits the tropical rainforests. Clearance of an area of moist tropical forest may bring rapid benefit for a few people for a short time. Assessment of long term cost-effectiveness is probably impossible but due care should be taken in the light of knowledge available to date. The harmful consequences could be confined to the area in question but may be disastrous for the world at large.

RESUME

Les auteurs donnent quelques exemples des conséquences épidémiologiques inattendues de la modification drastique de l'écosystème forestier tropical humide par les activités humaines.

L'homme est exposé aux effets débilitants, voire mortels, d'agents infectieux auxquels certains animaux sauvages servent de réservoirs et chez lesquels ils n'entraînent que des affections bénignes, voire inapparentes. Les relations complexes existant entre parasites et arbovirus d'une part, réservoirs et vecteurs d'autre part, sont illustrées par les cas bien connus de la fièvre jaune et de la maladie de Kyasanur.

Les végétaux cultivés peuvent aussi être infectés par des virus ou des ravageurs divers, normalement inoffensifs pour les populations de plantes sauvages. Des exemples en sont donnés dans le cas des plantations de Cacaoyers attaquées par des virus, des champignons et des insectes.

Les nouveaux complexes pathogènes ainsi créés peuvent avoir des conséquences désastreuses tant pour l'homme que pour ses cultures vivrières ou industrielles.

SUMMARY

The epidemiological consequences of tropical rainforest exploitation are discussed in terms of the effects on both exotic and native elements of the community. Incursion exposes man to debilitating or fatal infections/infestations having reservoirs in the wild fauna. As examples, the ecological relations of blood parasites (*Brugia malayi* and *Plasmodium* spp) and arboviruses having mosquito (yellow fever) or tick (Kyasanur forest disease) vectors are discussed.

Agricultural crops may be similarly affected by pests/pathogens naturally present in the flora ; by viruses (e.g. cocoa swollen shoot/mottle leaf), insects (e.g. *Tiracola plagiata*) and fungi (e.g. *Microcyclus ulei, Ganoderma, Fomes* and *Armillaria* spp). The harmful consequences for indigenous humans are illustrated with reference to the effects of measles and influenza on immunologically and/or chemotherapeutically unprotected persons.

Some of the risks to the world's population as a whole, have been considered in the light of fragmentary information concerning animal viruses (e.g. monkey pox, lassa fever, herpes virus B) and plant pathogens/pests which have spread from local foci of typically inapparent infection to cause devastation elsewhere.

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