

Review: FRANCIS L. BLACK
YALE UNIVERSITY SCHOOL OF MEDICINE

David Stannard has presented a detailed reevaluation of the generally accepted assumptions about the "precontact" population of Hawaii. He concludes that the population before Cook's arrival was not about 250,000, as commonly assumed, but at least threefold higher. No really new data are available, and the revised estimate is based on extrapolation from very small samples or weak hypotheses. But these, too, were the bases of the earlier assumptions, and the Hawaiian data are not inferior to data used for like calculations of the effects of the post-Columbian epidemics on other newly contacted populations [1]. Stannard has approached the problem from multiple directions, and these several lines of reasoning converge to form a surprisingly strong impetus for revision. The effect is enhanced, because, with agreement of the several arguments, the overall probability is the product, not the sum, of the parts. This effect is reminiscent of the combination of many weak forces at an antigen-antibody interface to form one of nature's strongest bonds.

Stannard uses four principal lines of argument in *Before the Horror:*
(a) Lieutenant James King's original estimate was low because it was

based on a section of coastline that offered less than optimal conditions and made several unwarranted assumptions about other parts of the islands; (b) unrestricted population growth from the time of first settlement to Cook's arrival would give a larger population than previously recognized; (c) the islands had the potential to support a large population, and archaeological evidence of settlement on unfavored areas indicates that much of this potential was indeed utilized; and (d) extrapolation of the number of survivors, at the time of the first census, back to the time of contact indicates a large initial population.

I have no special competence to evaluate points a or c and will leave these to others. My area of expertise is the effects of infectious disease on isolated populations and my firsthand experience is mostly with South American Indian tribes. In the South American rain forest many discrete population groups remained unaffected by direct contact with the cosmopolitan world until the second half of this century, and it has been my privilege to study several of them shortly after contact became regular. These tribes are isolated from one another as securely as the island populations of the Pacific, and like the Pacific populations each can be treated as a demographic entity. Modern-day Hawaiians may be shocked by the comparison, but the climate of the Amazon is not very different from that of Hawaii. The Amazon is warmer, but temperatures over 100°F are rare. In areas of even moderate elevation, nights are cool. Rainfall is no greater than on the windward sides of the islands and comes mostly in the afternoon.

Human Population Growth Potential

Is Stannard justified in concluding that, in the absence of population restraints due to limited resources, a founding population of a hundred persons would be expected to grow to 800,000 by 1778? In reaching this conclusion Stannard uses relatively new evidence that the initial settlement occurred before A.D. 100. Eleanor Nordyke, in a critical commentary appended to Stannard's main essay, contests this date and adheres to that of A.D. 300 given by Kirch [2]. Rouse, in a separate study that integrates data from the whole history of Polynesian migration, gives the date of settlement as A.D. 600 [3]. To attain the 8,000-fold increase postulated by Stannard, the Hawaiian population would have to grow 15 percent per twenty-seven-year generation [4] on Stannard's schedule, or 23 percent according to Rouse. Recently (1980 to 1985) the whole world grew at a rate equivalent to 36 percent per twenty-seven

years [5]. Clearly, the growth Stannard postulates is possible, regardless of which date is correct.

The potential for population growth is actually much greater, as can be illustrated by a South American example. Protasio Frikel studied the Xikrin Indians of Brazil in the early 1960s [6], and we were able to follow their population changes with some regularity from 1970 to 1981. The Xikrin are a northern branch of the same cultural group as the Cayapo, who are described--over-simplistically--by Stannard as having been exterminated by disease through contact with one priest (p. 47). There was warfare between the Xikrin and Neo-Brazilians throughout the first half of the present century. The effect of this was ultimately serious, but in the early part of the period, the acquisition of steel tools may have provided demographic compensation for battle losses by facilitating the clearing of gardens. Poliomyelitis, and possibly hepatitis [7], were introduced to the tribe by contact with contaminated water during this period. However, the worst was to come when, in 1963, the Xikrin moved their village to a place from which they could engage in peaceful trade with the outside world. Half their number died in the next two years. The survivors moved back to the hinterland and delegated their trade to a succession of priests, and then to the Brazilian Indian service, FUNAI. Complete isolation was not restored, but the frequency of epidemics declined and the Indians were able to continue getting machetes and shotguns. During the next seventeen years the population increased threefold, a rate equivalent to 500 percent per generation (Figure 1). Shotguns undoubtedly played a role in making this growth possible, but shells were scarce, and most hunting continued to be with clubs or bow and arrow. A key factor in this growth was the fact that only the young had survived the early sixties. In 1964 only three of the eighty-six survivors were over thirty years of age and only twenty-one were under ten. It was a group of people entering their most productive years. So, too, may have been the initial settlers of Hawaii. A

Nordyke argues that populations grow according to an S-shaped curve and that rates of growth such as those discussed above could not be maintained (p. 110). The S-shaped curve, however, comes from bacteriology, where a time interval equivalent to several log phase generations is needed for full activation of growth, and depletion of nutrients puts the top on the S. There is no scale inherent to the S and the duration of the log phase depends on the ratio between size of initial inoculum and amount of medium. The growth of a human population has no appreciable lag phase and the log phase need end only when the

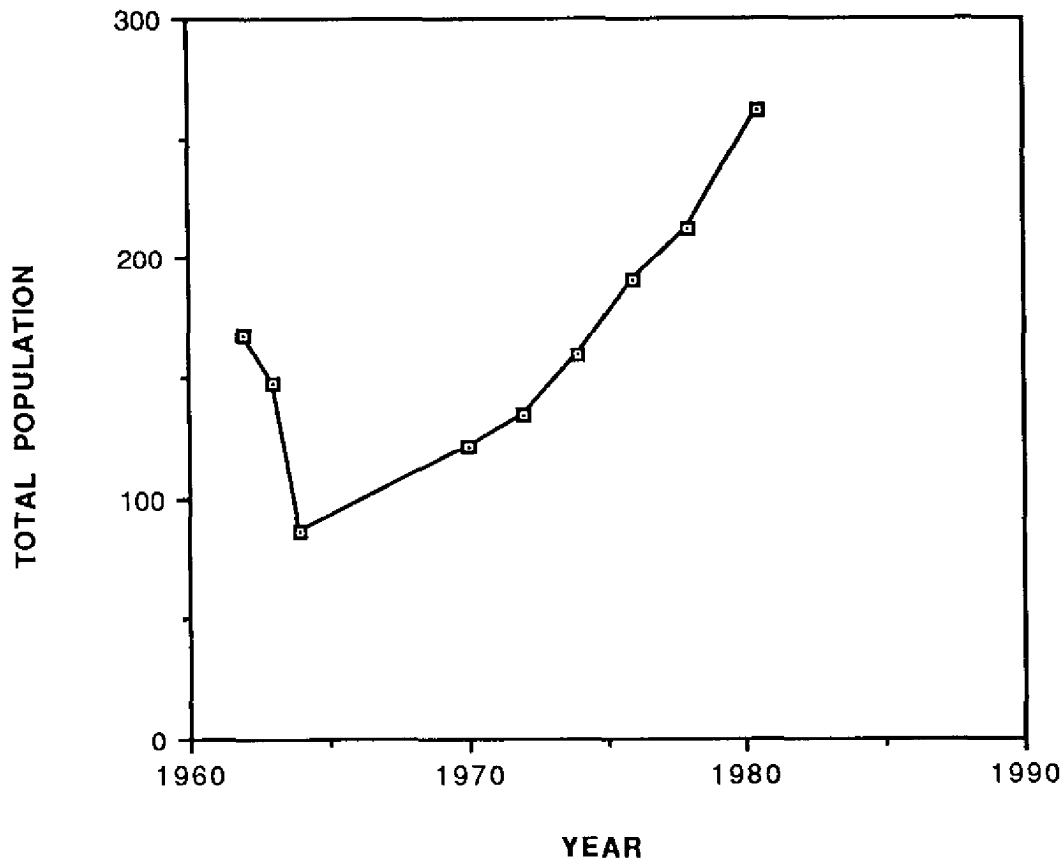


FIGURE 1. **Population changes in the Xikrin tribe of Brazil.**

resources become limiting. The Xikrin growth curve illustrated in Figure 1 failed to maintain a logarithmic rate in the latter periods of observation, and less quantitative data from subsequent years indicate that, while the tribe continues to grow rapidly, the increase does not continue to be logarithmic. The reason for the reduction in the rate of increase becomes apparent when one notes that in the years covered by the chart the proportion of women in their childbearing years decreased from 23.3 percent to 12.6 percent of the population, through dilution and aging. A time scale covering several generations would have permitted averaging out these ages changes. The curve would then show a less rapid initiation, but the logarithmic rate of increase might continue much longer.

Diseases That May Have Depleted the Hawaiian Population

Before examining the potential of disease to cause the population loss postulated by Stannard, it is necessary to determine what diseases may

have been involved. Only the diseases of the first fifty years need concern us, because the rest is documented. The range of possible diseases can be considerably narrowed on general principles.

First, certain infections seem to accompany humans everywhere they go and probably predated Cook's arrival [8]. These are diseases caused by agents that are continuously carried, and continually released, over long periods of time by anybody who has been infected. Selective forces favor strains that do not incapacitate the host, and these agents cause diseases that may be annoying but are seldom dangerous. The most important are probably the intestinal worms: ascaris, hookworm, and the like. Children become infected at an early age and, until they are toilet trained, it is very difficult to keep the worms from spreading in a society without diapers or hard floors. Filariasis may also have been present. Various semisaprophytic bacteria such as *Staphylococcus aureus*, some of the *Streptococci*, and *Escherichia coli* must have been present, but would do little harm to the young and healthy. Tuberculosis, leprosy, and yaws might fall in this class but seem, in fact, not to have been sufficiently persistent to have been carried all the way through the Polynesian migrations. Some viral infections would have been present. The herpes group are prominent here, including EBV, the cause of infectious mononucleosis. EBV, however, rarely causes disease when infection occurs early in life, and early infection is always the pattern in less developed societies. Occasionally chickenpox virus, another member of the Herpesvirus group, has been left behind in the formation of Amazonian tribes [9], but, because it erupts in older people as shingles, it may have been carried to Hawaii with the original settlers. Hepatitis B was probably present. It may lead to liver cancer when infection occurs very early in life but, for reasons that are not clear, in Polynesia [10] and Amazonia [11] it usually infects in later childhood when it causes neither cancer nor serious liver disease. Members of the wart virus family, including the venereal strains that cause cervical cancer, may have been present; if so, these particular strains would have exacted a significant toll. In general, the diseases brought to Hawaii by the original settlers were mild or had their main effect late in life. None would have occurred in epidemic waves.

Another class of diseases that must have occurred in Hawaii before Cook's arrival are the zoonoses. These diseases of animals that may spill over into humans, however, were probably rare because of the limited Hawaiian fauna. Many insect-transmitted diseases would not have been present because of the absence of appropriate mosquito vectors and of small mammalian hosts. Certain bacterial diseases--plague, tularemia,

and brucellosis --would also have been excluded for lack of the primary host. Trichinosis and tapeworm could have been brought in with the settlers' pigs, but it seems that porcine tapeworm, at least, was often left behind in serial migrations [12]. Possibly the most important diseases in this class would have been the parasitic infections acquired from fish, capillariasis and anasakidiasis, but these are seldom common enough to be a major problem. It is easy to believe that the Hawaiian population was in excellent health when contact was made, as stated by several early sources whom Stannard cites (p. 60).

Apart from the preexisting diseases, we can eliminate from consideration a large group of diseases that spread quickly through small human populations and would not have persisted in Cook's crew on the long sea voyages from England to Polynesia and from Tahiti to Hawaii. It is not possible that Cook brought influenza with him from England, as Stannard suggests (p. 70). The influenza cycle lasts at most one week in human and, in the cramped quarters of an eighteenth-century ship, there would not have been more than three cycles. It took Cook seventy-four weeks to complete the trip from England to Hawaii. Some of these diseases may already have been seeded in Tahiti and reintroduced to the crew at that point, but it still took six weeks for the voyage from there to Hawaii and this is too long for influenza or the parainfluenza viruses to have been maintained. Most of the viruses that cause common colds would not have persisted six weeks. Measles, rubella, and mumps could have been carried that far, but the crew, having grown up in England, would have been immune to these viruses.

A fourth category of disease can be eliminated because we know that epidemics affecting all age groups occurred after the first census. Measles (1848) [13], and smallpox (1853) [14], two of the most dangerous diseases, are in this category. It would have been possible for smallpox to have been carried in on Cook's ship in the form of infected scabs, but the likelihood of scabs initiating infection is low. Apparently the introduction of these two diseases awaited the initiation of a shipping connection with California.

Finally, there are the diseases that were excluded during the precontact period by an inappropriate environment or lack of intermediate hosts. There can have been little environmental change during the era of very occasional contact and these diseases would have remained excluded during the period that concerns us here. I would eliminate the typhuses and plague from consideration on this basis. Cholera causes large epidemics only where it is spread by large bodies of fresh water, and is likely, at most, to have become sporadic. Cholera was not easily carried over long distances prior to emergence of the eltor strain in the

1960s and it is improbable that it was introduced by the initial contacts.

This still leaves a great variety of diseases that might have been brought to Hawaii for the first time by early European explorers. Most prominent, as Stannard indicates, are tuberculosis and the venereal diseases gonorrhea and syphilis. These diseases, and also venereal *Chlamydia*, would not have caused drastic immediate depopulation but rather loss of fecundity and slow death. Other newly introduced diseases would have had more pronounced, but not necessarily more serious, consequences. Diphtheria is often carried for long periods by immune persons and might easily have been imported. New pathogenic enterobacteria, causes of diarrhea and dysentery, were probably introduced early. Typhoid seems the most probable chief factor in the 'ōku'u [15]. Many types of pneumococci were probably new to the islands, as were also hemolytic streptococci, including the causes of scarlet fever and of streptococcal sore throat and rheumatic fever. The *Meningococci* and *Hemophilis influenza b*, important causes of meningitis, were probably new and could have survived the voyage. Most types of enteroviruses (there are more than a hundred) are unlikely to have been carried through the serial Polynesian migrations and many, including the polioviruses and hepatitis A, would have been brought by early Caucasian visitors.

Reasons for High Mortality Rates

If the first European explorers brought only a limited, albeit long, list of diseases with them to Hawaii, one may well point out that the Europeans of that time lived and thrived in the presence of a nearly unlimited list of diseases. How could the restricted set of diseases cause the postulated havoc among the Hawaiians? Stannard amply demonstrates that the Hawaiian population losses were not unusual in newly contacted populations. Robert Schmitt, a statistician, cites instances in which other newly contacted populations were less affected in a critique printed in part 2 of *Before the Horror*, but this does not change the generality of high mortality rates in isolated populations after contact with cosmopolitan cultures. Although Stannard's backward extrapolation postulates that the numbers that died prior to 1828 were greater than after that date, he does not claim that the *rate* was higher in the earlier period. His smoothed curve suggests that half the remaining population was lost in each of the first three twenty-five-year periods. Although several important diseases played no role in the first two periods, others were probably better tolerated in the third.

Why then did these people die? The implied assumption is that New World people were genetically inferior. Yet the Polynesians had been separated from the main body of the human species on the Southeast Asian mainland for only 120 generations [3]. With the complex interdependence of human genetic traits, little evolution can proceed that fast. In spite of extensive studies, no widespread deficiency of the immune system has been found either in Polynesian survivors or in populations of New Guinea or the Amazon. If the original populations were genetically heterogeneous with respect to resistance to infectious disease, as Dobyns [1] seems to imply, we might not see defects in the Hawaiian survivors, but we should have found them in the other populations. The others remained sufficiently isolated when studied that adverse traits could not have been eliminated by post-contact selection. Antibody titers to a wide variety of viral and bacterial infections are at at least normal levels in the Amazonians, and their white cell functions were in no way unusual for populations heavily infected with helminthic parasites. We did find that Amerinds who had had no prior experience with measles virus reacted to attenuated vaccine virus with more fever and slightly higher titers than measles-experienced populations [16], but an alternative explanation for that is offered below.

Four mechanisms can be suggested as explanations of the Hawaiian mortality, which must have been high no matter which value is accepted for the precontact population. All may be important. First, although it is unlikely that unusual human genes caused the debacle, it is possible that the pathogens were unusually virulent. A bacterium can go through 120 generations in two days and, with their simpler metabolism, microorganisms are freer to change. The production of large numbers of progeny is obviously an advantage to a microorganism, but this is likely to kill the host and reduce chances of spread. In virgin soil new hosts are plentiful and evolution tends to increase pathogenicity.

Second, the combined impact of multiple infections introduced by one group of visitors is greater than that of the same diseases singly. When a parainfluenza infection coincided with administration of measles vaccine in the Tirio Indians, the fevers were much more threatening than when the vaccine was experienced alone [17]. Parainfluenza normally causes only a common cold in adults. Several viral and parasitic diseases have a direct suppressive effect on the immune system, giving a combined action that is not just additive, but synergistic.

Third, when a new infection enters a population, everyone is susceptible, nearly everybody gets sick at once, and the social structure breaks down. Nobody remains well enough to carry on the simple service tasks such as hauling water and stoking fires. A sense of doom pervades, and

people simply curl up in their sleeping places and wait to die. Neel et al. observed a virgin soil epidemic of measles among the Yanamama Indians in 1968 and have written emphatically on this point [18]. Even basic nursing care can make a big difference in the survival rate, as evidenced by the change in mortality in Brazil's Xingu Park when help arrived during their first experience with measles [19].

The last factor, the effect of inbreeding, is less widely recognized. If a hundred settlers came to Hawaii from Tahiti, they were probably a highly interrelated group and carried much fewer than the two hundred potential germ lines. Similar genetic bottlenecks must have occurred in the migration from Samoa to the Marquesas and the serial migrations that led the Polynesians to Samoa [3]. The net effect is that the whole population was derived from very few progenitors. This is most evident in the HLA system. Taking just the Class I genes, there are three different loci, each with twenty to thirty alleles in cosmopolitan populations. Every person has two genes at each locus, so the possible number of different genetic constitutions is about twenty-five to the sixth power, or twenty-four million. A linked set of half of each parent's repertoire, a haplotype, is passed to every child. In cosmopolitan societies, the chance that a child will get identical haplotypes from both parents is extremely low. In Polynesia [20], and in the South American tribes [21], only four or five different alleles have been retained at each locus and these tend to occur repeatedly in just a few sets. The result is that in these societies children often get only half the usual number of HLA antigens [22]. Those who have the reduced complement survive less well. It is not certain that the disadvantage is mediated by reduced resistance to infectious diseases, but the HLA antigens are an essential part of the immune defense. The effect of inbreeding due to population bottlenecks is not confined to the HLA system and homozygosity at other genetic sites also reduces resistance. Even when precontact Hawaiian marriage partners were not closely related in an immediate way, they were related through so many lines of descent that their genetic similarity approached that of siblings. The measles vaccine virus given to Amerinds may have multiplied more freely because of similarly reduced resistance; production of larger amounts of virus would have caused the higher fever. The Amerind, and original Polynesian, genes are not inferior; there are just too few different genes.

Summary

I stated at the outset that the coincidence of Stannard's several lines of inquiry gives strength to his conclusion, Only two of these lines have

been evaluated here, but both stand up well. Whether or not Stannard's estimate of the population of Hawaii deserves to be accepted as a preferred hypothesis depends on whether his other lines of analysis also withstand independent evaluation. The general reevaluation of the effects of contact between Europeans and New World populations led by Dobyns is based on evidence that is no better than this, and it too depends on internal consistency for creditability. If the other components of Stannard's work are confirmed, his study will be one of the strongest elements in the broader revision. The horror is that, if Stannard is right, half a million people died as a result of our culture's incursion without our even being aware.

REFERENCES

1. Dobyns, HF, Estimating Aboriginal American Population. An appraisal of techniques with a new hemispheric estimate, *Current Anthropology* 7:395-416, 1966.
2. Kirch PV, *Feathered Gods and Fishhooks*, Honolulu, University of Hawaii Press, 1985.
3. Rouse I, *Migrations in Prehistory*, New Haven, Yale University Press, 1986.
4. Black FL, Pinheiro FP, Oliva O, et al., Birth and survival patterns in numerically unstable proto-agricultural societies in the Brazilian Amazon, *Medical Anthropology* 2: 95-127, 1978.
5. United Nations, *Population Annual, 1986*, Geneva.
6. Frikel P, Notas sobre a situação atual dos índios Xikrin do rio Caetete, *Revista do Museu Paulista N.S.* 14: 145-158, 1963.
7. Jacobson DL and Black FL, Hepatitis A antibody in an isolated Amerindian tribe fifty years after exposure, *Journal of Medical Virology* 19:19-22, 1986.
8. Black FL, Modern isolated pre-agricultural societies as a source of information on pre-historic epidemic patterns, in *Changing Disease Patterns and Human Behavior*, edited by NF Stanley and RA Joske, London, Academic, 1980.
9. van Mazijk J, personal communication, 1981. The Trio of Suriname experienced a virgin soil epidemic of chickenpox with three deaths in 1975.
10. Gust ID, Lehmann NI and Dimitrakakis M, A seroepidemiologic study of infection with HAV and HBV in five Pacific islands, *American Journal of Epidemiology* 110:237-342, 1979.
11. Black FL, Pandey JP and Capper RA, Hepatitis B epidemiology and its relation to immunogenetic traits in South American Indians, *American Journal of Epidemiology* 123: 336-343, 1986.
12. Gadjusek, DC, Introduction of *Taenia solium* into West New Guinea with a note on an epidemic of burns from cysticercus epilepsy in the Ekari people of the Wissel Lakes area, *Papua New Guinea Medical Journal* 21:329-342, 1978.

13. Gulick LH, On the climate, diseases and materia medica of the Sandwich Islands, *New York Journal of Medicine* 14:169-211, 1855.
14. Fenner F, Henderson DA, Arita I, et al., *Smallpox and Its Eradication*, Geneva, World Health Organization, 1988.
15. Schmitt RC, The oku'u--Hawaii's greatest epidemic, *Hawaii Medical Journal* 29: 359-364, 1970.
16. Black FL, Pinheiro FP, Hierholzer WJ and Lee RV, Epidemiology of infectious disease; the example of measles, in *Health and Disease in Tribal Societies*, Ciba Foundation Symposium No. 49, pp 115-135, 1977.
17. Black FL, Woodall JP and Pinheiro FP, Measles vaccine reactions in a virgin population, *American Journal of Epidemiology* 89:168-179, 1969.
18. Neel JV, Centerwall WR, Chagnon NA and Casey HL, Notes on the effect of measles and measles vaccine in a virgin-soil population of South American Indians, *American Journal of Epidemiology* 91:418-429, 1970.
19. Nutels N, Medical problems in newly contacted Indian groups, in *Biomedical Challenges Presented by the American Indian*, Pan American Health Organization Scientific Publication No. 165, pp 68-76, 1968.
20. Searjeantson SW, Ryan DP and Thompson AR, The colonization of the Pacific: the story according to human leukocyte antigens, *American Journal of Human Genetics* 34: 904-918, 1982.
21. Black FL, Interrelationships between Amerindian tribes of lower Amazonia as manifest by HLA haplotype disequilibria, *American Journal of Human Genetics* 36:1318-1331, 1984.
22. Black FL and Salzano FM, Evidence for heterosis in the HLA system, *American Journal of Human Genetics* 33:894-899, 1981. Hedrick PW and Thomson G, Evidence for balancing selection at HLA, *Genetics* 104:449-486, 1983.