

## News and Views

### A View on the Science: Physical Anthropology at the Millennium

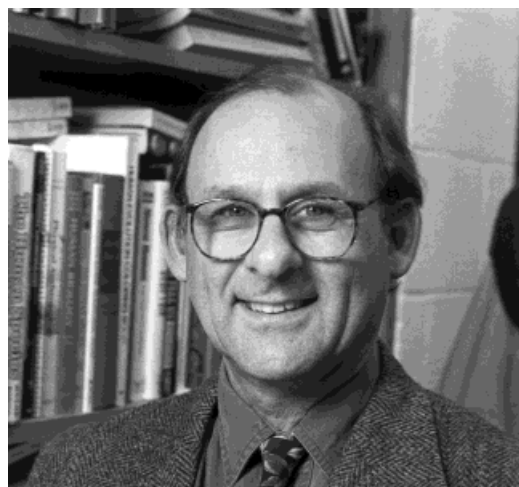
ALAN C. SWEDLUND

*Department of Anthropology, University of Massachusetts, Amherst, MA 01003-4805*

**EDITOR'S NOTE** The year 2000 marks the onset of the 21st century. In this transitional year, prominent physical anthropologists will provide brief reflections on our discipline, including what attracted them to it, and their views on the directions our discipline may pursue as we enter, in January 2001, the third millennium. *Am J Phys Anthropol* 113:1-4, 2000.

© 2000 Wiley-Liss, Inc.

My work has been located primarily at the intersection of physical anthropology,



Alan Swedlund is Professor and past Chair of the Department of Anthropology at the University of Massachusetts, Amherst. He also holds an appointment as Research Associate at the Social and Demographic Research Institute (SADRI) at the University. A recipient of numerous grants and awards, in 1995 he received a Weatherhead Resident Fellowship to the School of American Research, Santa Fe, New Mexico. In 2000 he was awarded a Hewlett Foundation Teaching Fellowship at the University of Massachusetts. Dr. Swedlund's research focuses on demographic approaches to biocultural processes in historical populations.

demography, and historical epidemiology. I have an ongoing interest in long term demographic processes in the prehistoric American Southwest using paleodemographic approaches (e.g., Swedlund, 1994). The majority of my research, however, engages historical population data from North America in general and New England in particular (e.g., Swedlund, 1990, Swedlund and Ball, 1998). I was drawn to demography and genetics as a graduate student in part because the recent availability of what we then called "high speed" computers provided new opportunities for handling relatively large data sets and investigating diachronic processes. Microevolutionary studies of the 1960s and 70s had shown great promise for understanding genetic variation in numerically small, indigenous populations. What I and others found lacking was the greater generational time depth necessary to estimate the effects of long term demographic processes on genetic structure. With colleagues and graduate students, I began an undertaking now known as the Connecticut Valley Project. I have not been able to escape it since, but it continues in a form very different from its origins. In the paragraphs below, I will retrace a few steps to show the intentional and sometimes serendipitous turns my research has taken, I will also point to issues and lessons that come from my areas of research but which may have relevance to a broader physical anthropology in the future.<sup>1</sup>

<sup>1</sup>This essay is motivated by a lot of conversations over the years with a lot of people, none of whom should be held responsible for anything I say. I would like to pay special note to George Armelagos, Michael Blakey, Margaret Connors, Alan Goodman, Ann Herring, Tom Leatherman, Lynnette Leidy, Bruce Levin, Richard Meindl, Debra Martin, Brooke Thomas and Kenneth Weiss. The Connecticut Valley Project acknowledges current support from NSF Grant SBR 9224572.

## THE STRUCTURE OF SUBDIVIDED POPULATIONS

Within the subdiscipline of physical anthropology, studies of historical epidemiology and demography rest largely on the collection, linkage, and computerization of data sets available from eighteenth, nineteenth, and early twentieth century records. These are found principally in North America, Europe, and in other regions with long histories of colonial administrations. Census and vital statistics data (including health and morbidity statistics) permit one to empirically investigate time-series approaches to a number of questions of interest to anthropology. Lacking, of course, are the genetic markers that would be desirable (although DNA sequencing technologies are making this possible now, subject to the availability of human remains). In lieu of genetic markers there are amazing arrays of data, including morphometric and anthropometric statistics from a number of old series on military recruits, students, the insured, and others.

The initial questions that drew many of us to historical data sets had to do with empirical opportunities to test theories of genetic drift, gene flow, and the opportunity for selection. By analyzing fertility, mortality, and marital migration rates among subdivided populations, long standing queries on the magnitude of inbreeding in human populations, for example, could be experimentally, albeit retrospectively, tested. The good news is that during the 1970s and early 80s historical demographers and demographic geneticists actually answered many of those questions. We accumulated a significant body of knowledge regarding the covariance of gene frequencies with geography and demography in numerous places. Many studies pointed to the rapid rates of gene flow among regional populations and relatively low rates of inbreeding. Measures of differential fertility and mortality partitioned by family and lineage—which have now given way to proportionate mortality (PMRs) and fertility ratios, hazards models, and frailty estimates—gave us some insight into the opportunity for selection. In several cases, it was possible to map the historical data onto gene frequency data of living descendants in the regions under study.

Following this decade or so of considerable success in understanding microevolutionary processes, those more invested in population and molecular genetics tended to move on to questions of ancestry and genetic epidemiology, while those of us who were more demographically and ecologically oriented tended to focus on the history of human health from other physical anthropology perspectives.

### Investigations of mortality

Interest in, and accumulation of, the historical mortality data collected in the 1970s and early 80s precipitated a long term interest in the correlates of mortality. I was particularly focused on infant and childhood mortality, since they represented a significant proportion of all deaths in nineteenth-century America (as well as elsewhere). It was about this time in my career when I embarked—with the help of several graduate students, and NSF and NIH funding—on the systematic linking of vital statistics and census data into family, household, and community units for analysis. Working closely with then Ph.D. candidate Richard Meindl (Kent State), we undertook a series of tests of cohort and family specific mortality utilizing the linked data.

We began our mortality project with an analysis of paired cohorts of newborns some of which had experienced epidemic disease while others had not (e.g., Meindl and Swedlund, 1977). It was interesting to note that the “stressed” cohorts—those assaulted by epidemic disease early in their life course—might in the end experience overall greater average life expectancy than “unstressed” cohorts, despite a high level of infant mortality. This suggested to us a “pruning” effect of early childhood mortality that may have been the result of selection against those genetically or constitutionally less fit to the exposure of the specific disease-causing pathogen.

The linked records also gave us the ability to measure infant and childhood mortality among families. We identified the major structural correlates of mortality to be wealth class, parity, maternal age, and other variables. Nevertheless, we discovered that deaths were not randomly distributed across all families in the Connecticut Valley. We

found residual groups of families that showed higher than expected deaths which prompted us to ask whether these deaths might be explained familiarly (“genetically”) or environmentally. Alas, the numbers were by then too small to reject either hypothesis.

I describe this example because I think it is a common one in many research programs in physical anthropology. It illustrates the problem of inadequate statistical samples that can occur even in large data bases. Consequently, even if the results point in favor of “family” or lineage effects, the genetic interpretation is still circumstantial and untested. However, the analyses suggested a number of cultural and environmental correlates strongly associated with childhood deaths. This prompts one to ask then, “Does one choose to study the small residual with little hope of resolving anything, or does one decide to take a broader biocultural perspective and address the total variability that remains available to epidemiological modeling?” That is, if one can develop satisfactory models to account for variation in infant and childhood mortality using maternal age, wealth class, parity, and family size (among others) which can be measured historically (e.g., Meindl, 1980). Is this not preferable to modeling an imputed genetic cause which one cannot measure?

Answering this question affirmatively provided an important turning point in my career. At this juncture, I became more interested in the broader questions of human mortality history and in the evolutionary history of pathogens than in the genetics of their human hosts. Other projects emerged or came to my attention. Particularly noteworthy in this regard was the work of Larry Sawchuck (Toronto) and Ann Herring (now, McMaster) on the historical demography/epidemiology of the colonial city of Gibraltar (e.g., Sawchuck et al., 1985).

### HUMAN BIOLOGY WITHOUT THE GENOME

As with several other topics in physical anthropology, those of us interested in the history of human health are challenged in the age of the genome. We are not likely to have strong inferences on specific human genotypic correlates to acute infectious disease, but I

would submit that this is not a very significant problem and may even reflect a rarity of such polymorphisms. Indeed, if measured by magnitude, the important events of the last few hundred years center largely on epidemics and transitions in infectious diseases for which the human genome appears to offer little in the way of explanatory power. (Perhaps the genomes of our pathogens may offer considerably more.) More to the point, observed mortality increases and declines can be accounted for largely by political-economic and environmental-historical factors affecting exposure and generalized immune competence without recourse to genetic variability in the host population(s).

Yet, as physical anthropologists, we are almost bound by professional oath to consider evolutionary factors in these disease histories. If so, physical anthropologists interested in historical epidemiology—and for that matter, genetic epidemiology—are obliged to become better cultural ecologists in order to collect the kinds of data and to deploy the more sophisticated models necessary to disentangle cultural-environmental-genetic factors and their interactions. We must also borrow more freely from molecular biology in order to identify the specific loci and mechanisms, both heritable and nonheritable, by which our species responds to a particular microparasite. However, it is not sufficient to find family or lineage effects, an hypothesized mechanism, and infer genetic causality. Obviously, the skills required suggest the need for improved collaboration among variously trained specialists.

Related to these problems has been our relative lack of attention to coevolutionary issues of host and pathogen and the evolution of virulence. Most anthropologists interested in disease-mediated selection have been strongly influenced by classic papers such as that of Haldane (1949) in which he argued that the long history of association with humans should afford many opportunities for pathogens to be agents of natural selection. There are caveats to this argument that deal with the ecologies of exposure, varying generation times of host and pathogen, and host resistance, as well as the methodological difficulties in empirically demonstrating a selection effect (e.g., Barrett, 1990; Svanborg-Eden and Levin, 1990; Levin, 1999).

### Historical epidemiology in the new millennium

The place of historical demography and historical epidemiology seems secure within the domain of physical anthropology. Understanding the interactions between biology and culture is fundamental to an appreciation of human variation and diversity. I concur with our colleagues who maintain that to be good physical anthropologists we not only need to be good biologists, but also attentive to the theories and findings of cultural anthropology. Humans are not genes and they are not rats (most of them, anyway). We know a lot about how socioeconomic and other culturally mediated factors contribute to increased exposure risks and the weakening of stressed immune systems.

In understanding the ecology of human infectious disease, I believe we need to pay increased attention to the *coevolution* of pathogens and hosts as well as to cultural and political-economic processes. Furthermore, where disease-mediated selection is concerned in the evolutionary contest between microparasites and humans, I tend to bet on the bugs.

Against a background of cultural and environmental information, we will be better equipped to estimate and infer genetic processes. In fact, when we encounter historical contexts in which the cultural and environmental variables are not sufficient, we may be in an improved position to consider evolved virulence in the microparasite responsible for the disease episode in question or novel genetic variants in the hosts. We sometimes forget, or under-emphasize, how important the cultural-environmental history of Central Africa was in interpreting the relationships between falciparum malaria and sickle cell (one of our favorite and most often told stories). The genetic side of the story is incomplete without knowing the part played by the cultural-environmental context.

I predict that molecular biology is likely to provide more on the evolution of attenuation and virulence of microparasites than it will on disease-mediated selection in humans. As we have seen with recent episodes of acute infectious disease, such as the AIDS epidemic, knowledge of cultural vectors and cultural-

environmental conditions are indispensable to understanding the circumstances that gave rise to the epidemic, and without this knowledge the molecular biology is simply insufficient (e.g., see Ewald, 1994; Armelagos, 1998). From where I stand, the exciting future of physical anthropology is in the reintegration of biology and culture in ways that incorporate complex models of culture and ecological history with a human biology that is judicious and rigorous in its claims for genetic causality and disease-mediated selection.

### LITERATURE CITED

- Armelagos G. 1998. The viral superhighway. *The Sciences* January/February 38:24–29.
- Barrett JA. 1990. The detection of selective differences in populations. In: Swedlund A, Armelagos G, editors. *Disease in populations in transition: anthropological and epidemiological perspectives*. New York: Bergin and Garvey. p 47–51.
- Ewald P. 1994. *Evolution of infectious disease*. Oxford: Oxford University Press.
- Haldane JBS. 1949. Disease and evolution. *Supplement to La Ricerca Scientifica* 19:68–76.
- Levin BR. 1996. The evolution and maintenance of virulence in microparasites. *Emerg Infect Dis* 2:1–11.
- Levin BR. 1999. Infectious disease as an agent of natural selection: observations, opinions, and unsolicited advice. Paper presented to the 25th annual meeting of the American Society for Histocompatibility and Immunogenetics. New Orleans, October, 1999.
- Meindl R. 1980. Family formation and health in nineteenth century Franklin County, Massachusetts. In: Dyke B, Morrill W, editors. *Genealogical demography*. New York: Academic Press. p 235–250.
- Meindl R, Swedlund A. 1977. Secular trends in mortality in the Connecticut River Valley, 1700–1850. *Hum Biol* 49:389–414.
- Sawchuk LA, Herring DA, Waks LR. 1985. Evidence of Jewish advantage: a study of infant mortality in Gibraltar, 1870–1959. *Amer Anthropol* 87:616–625.
- Svanborg-Eden K, Levin BR. 1990. Infectious disease and natural selection in human populations: a critical examination. In: Swedlund A, Armelagos G, editors. *Disease in populations in transition: anthropological and epidemiological perspectives*. New York: Bergin and Garvey. p 31–46.
- Swedlund A. 1990. Infant mortality in Massachusetts and the United States in the nineteenth century. In: Swedlund A, Armelagos G, editors. *Disease in populations in transition: anthropological and epidemiological perspectives*. New York: Bergin and Garvey. p 161–182.
- Swedlund A. 1994. Issues in demography and health. In: Gumerman G, Gell-Mann M, editors. *Understanding complexity in the prehistoric Southwest*. Reading, MA: Addison-Wesley. p 39–58.
- Swedlund A, Ball H. 1998. Nature, nurture, and the determinants of infant mortality: A case study from Massachusetts, 1830–1920. In: Goodman A, Leatherman T, editors. *Building a new biocultural synthesis: political economic perspectives on human biology*. Ann Arbor: University of Michigan Press. p 191–228.