Reviews in Anthropology, Vol. 19, pp. 271–280
Reprints available directly from the publisher
Photocopying permitted by license only

Sociobiology and Scientific Reductionism: Redux

Jeffrey H. Schwartz

Kaye, Howard. *The Social Meaning of Modern Biology*. New Haven: Yale University Press, 1986. ix + 184 pp. including references. No price.

Oyama, Susan. *The Ontogeny of Information*. Cambridge: Cambridge University Press, 1985. v + 206 pp. including notes, bibliography, name and subject indices. $34.50 cloth, $12.95 paper.

The two monographs under review are similar in overall intent: an attempt to fracture the veneer of, if not dispell in the entirety of its vicissitudes, sociobiology. And both authors use, albeit with at times markedly different slants and emphases, the same method of argument: to attempt to seriously question if not invalidate the reductionistic background of, and approach to, sociobiological interpretation.

In *The Social Meaning of Modern Biology*, Kaye relies more on an historical review of important and influential individuals to demonstrate the underlying motivations, assumptions, and logical inconsistencies that have nonetheless entered the increasingly accepted dogma in certain quarters that, as Francis Crick concluded, "'the ultimate aim of the modern movement in biology is in fact to explain *all* biology [*biology* in Crick's vocabulary is a synonym for *life*] in terms of physics and chemistry, 'including 'our strange feeling of being conscious'" (p. 54 in Kaye). This level of reductionism may seem radical at best to many, but it does serve to illustrate the degree to which the apparent differences that seem to exist between humans and anchovies, much less humans and mice or monkeys, are perceived as nothing more than different strands of macromolecules with differing molecular weights and valences of bonding. Under such constraints, it would hardly seem appropriate to invest in any sociocultural anthropologist's, sociologist's, or even economist's "analyses" of human behavior. Similarly, since the answers to questions or organismic difference—no matter how defined in terms of behavior, morphology, or something else—lie ultimately at the level of investigation available only to the

JEFFREY H. SCHWARTZ is a Professor of Anthropology at the University of Pittsburgh and is a research associate at both the American Museum and Carnegie Museum of Natural History. Among his research interests are theoretical issues in model and methodology in evolutionary biology. His most recent focus has been on the discrepancies between molecularly and morphologically based phylogenies among the hominoids, which he has addressed in "The evolutionary relationship of man and orang-utans" (Nature, *308: 501–505, 1984), "Hominoid evolution: a review and reassessment"* (Current Anthropology, *25: 655–672, 1984), The Red Ape: orang-utans and human origins (Houghton Mifflin, 1987), and Orang-utan Biology (J. H. Schwartz, ed., Oxford University Press, 1988).*

"hard sciences," even the supposedly scientifically rigorous undertakings of population geneticists, experimental biologists, embryologists, and comparative anatomists, as well as ethologists may only scratch the surface of what makes animals, including humans, distinctive.

As does Oyama, Kaye tries to untangle the armor, or at least the seemingly ironclad logic of scientific reductionism and positivism by addressing the language and nature of the arguments proffered by the more influential in the historical assaults on, and reactions against, social Darwinism and Lamarckism. The approach is an interesting one, largely because it runs the risk of accusations of setting up straw men in the course of arguing that these great figures of science were not all that scientific after all. At best, they were ordinary mortals, having the same difficulties as those before them in keeping either quasi-religious notions or personifications and anthropocentrisms out of theories of evolution and natural selection. Of course, it is to be expected that the earlier evolutionists would have had to overcome far greater obstacles in trying to make clear the distinctions between a world whose occupants were the result of evolution rather than some guiding omnipotent hand (e.g. Eisley 1958; Irvine 1959). Indeed, the line is a fine one, ever the more so because "evolution" to nineteenth century evolutionists was taken as being largely synonymous with "progress" (ibid.; also see Kaye). As such, it is not difficult to appreciate how the biblical "Great Chain of Being" could be transformed easily into an evolutionary "Great Chain of Being" (Schwartz 1987).

A major if not the major problem that has persisted historically is how to deal evolutionarily with humans, whether in the sense of trying to unravel their phylogenetic relationship to and among the rest of the animal world, or in trying to understand what it is to be human, i.e. seemingly so different behaviorally and socially from other animals. Darwin (1871) devoted much of the first of the two volumes of *The Descent of Man* to the subject of the evolution of "civilized man" from a savage and barbaric state (as "evidenced" by the primitive peoples of Africa). Indeed, Darwin (p. 141) sought to demonstrate "that man has risen, though by slow and uninterrupted steps, from a lowly condition to the highest standard as yet attained by him in knowledge, morals and religion." And it is the quandary over the attainment of such seemingly specifically human attributes as "morals" and the antithesis of morality, the lack of it, that has yielded a history of debate: Has "evolution" (i.e. natural selection) ceased to impact on human development, or are those "mental facilities" of *Homo sapiens*, including the horrific ones that lead to such atrocities as those inspired under Nazism, inexorably biological (i.e. the result of evolution and natural selection)? Therein lies the more than 100 years of controversy between the adherents of social Darwinism and Lamarckism. And therein also lies the crux of the debates for and against sociobiology, which, even in its less extreme formulations, is characteristically reductionistic and which, therefore, with little modification in at least sentiment, is not too removed from Crick's perceived goal of modern biology (cited above).

This is where many, including Kaye, say "stop we've gone too far." For even those great scientists involved in the formulations and articulations derivative of the Modern Synthesis, such as Julian Huxley, C. H. Waddington, Theodosius Dobzhansky, George Gaylord Simpson, and Peter Medawar, when discussing *Homo sapiens*, constantly affirmed this species' special place in nature, dependent for its continued development upon intellect and culture, and unique among organisms in the pursuit of innate "ideals." In Kaye's review, even the most "hard" of scientists eventually succumb to the appeal of analogy, metaphor, resonance, and personification, and endow evolution and natural selection with qualities of reason, foresight, and intentionality (viz. Jacques Monod (1971)), through his groundbreaking work on deciphering DNA a founding "father" of molecular biology). If the language of scientists can so clearly be seen as venturing into the metaphysical, how, questions Kaye, can anyone claim that scaling the complexity of humanness through the reductionistic viewpoint of the molecule and eventually its atomic constituents is more viable than approaching at least some part of the study of our own species from a more humanistic line of inquiry, even if it is recognized as biologically humanistic?

Kaye ends *The Social Meaning of Modern Biology* on a cautionary note: "More than its Romanticism, it is sociobiology's reduction of human values to the question of biological survival and its reduction of the individual human being to an epiphenomenon of its genes—an utterly 'expendable' 'survival machine' and gene replicator—that may prove so destructive (p. 164) . . . Contemporary biologists have indeed raised profoundly important issues, but in facing the difficulty problems of the day, both scientists and laymen must hold fast, once again, to the delicate distinction between knowledge and speculation, science and myth" (p. 165).

Although, as I mentioned above, there is some overlap of approach if not citation between *The Social Meaning of Modern Biology* and *The Ontogeny of Information*, Oyama focuses her arguments on the issue as she perceives it of "nature versus nurture," and, although she too finds fault with the proclaimed path of enlightenment through scientific reductionism, she does attempt to crack the shell of scientificism through a review of the biology and not just of the biologists. *The Ontogeny of Information*, therefore, is a discourse on two levels: the ontogeny (in this case development and linguistic presentation) of biological information (including thought, theory, and philosophy) as perceived, generated, and disseminated by biologists, and also the life-history ontogeny of an individual (at the levels of genic-through-organismal and phenotypic unfolding, by which I mean "evolution" in the sense of the nineteenth century embryologists and because of which Darwin used the word but once in the *On the Origin of Species* [see, for example, de Beer 1958, and Gould 1977]).

In a more aggressive fashion than Kaye, Oyama highlights various individuals who have contributed to the theory and language of sociobiology and its brand of scientific reductionism. Two such scholars are Richard Dawkins (and his monographs *The Selfish Gene* [1976] and *The Extended Phenotype* [1983]) and E. O. Wilson, largely in reference to the popular *Sociobiology: a New Synthesis* (1975) but also C. J. Lumsden and E. O. Wilson's *Genes, Mind, and Culture* (1981). The importance of Dawkin's work for revealing the potential pitfalls of scientific reductionism should be clear and will be discussed momentarily. E. O. Wilson, on the other hand, is both praised for his courage and daring—in proposing, or at least attempting to centralize in theory and presentation, a novel and controversial interpretation of the evolution of human culture and behavior in the same biological context that one would also deal with the interactions of a colony of ants as individuals to one another and to their outside world—and criticized for what is argued to be the continuing failure of scientific reductionism, whether it be applied to sociobiological or other concepts, i.e. the ultimate reliance on metaphor to substitute for what is still a sorely incomplete understanding not only of development, but of how "the gene"—the level to which all morphology and behavior is reduced—even works.

Suffice it to say that the Dawkinsian notion "the body is the gene's way of getting itself around" is anathema to the antireductionist. To dismiss anything that may be construed as phenotypic, whether it is morphological or behavioral in nature, as superfluous and not the stuff phylogenetic histories are made of would seem to be not only anti-evolutionary, but anti-Darwinian, or just plain anti-Darwin, which is even worse than being anti-American. But, as Oyama points out, it is just not good enough to carry on believing that such items as teeth and claws are genetically determined, and thus inherited, and that things behavioral are not. That, says she, only continues the myth that one can identify with any certainty the actual genetic basis of teeth, claws, or other morphological attributes and that, if one were to make such a determination, one would find that the ontogeny of these hard tissue structures is nothing more than a blind unfolding—intact and unaltered—of the genetic code embedded underneath. The other side of this mythical coin is that the nuances of certain behaviors, and most especially the development of human behavior and culture, exist only in the realm of natural selection and its finer subdivisions. Here, Oyama is reacting in part to Dawkins's assertions that, because everyone understands how genes control morphological differences among organisms (a point with which she disagrees), everyone can also plainly see that if there is a correlation between "gene" and "character," such a correlation would exist in the realm of behavior, as well (a source of further disagreement). She does, however, agree with Dawkins's (1983: 199) contention that "if there is any sense in which the brain is inherited, behaviour may be inherited in exactly the same sense . . . If we object to calling behaviour inherited, as some do on tenable grounds, then we must, to be consistent, object to calling brains inherited too." But Oyama does not agree with Dawkins and others who would subscribe to sociobiological principles. Rather, Oyama uses the sentiment just quoted to pursue the argument that, if there are elements of morphological expression and development that are subject to environmental control, then so there must also be for behavioral attributes, no matter how genetically encoded such features may indeed seem to be. And this is the essence of her perception of the issue "nature versus nurture": there is a fuzzy boundary between the genetically embedded and the environmentally interfered with, and, as such, it may well be the case that, at any given time, the ontogenetic pathway of an individual, or its component features, may be altered by the external, no matter how genetically embedded the initial ontogenetic "messages" may be. This, to me, seems like the other-side-of-the-coin version of sociobiology: sociobiologists assert that everything, on some level, is genetically based and thus inherited; Oyama argues that all things can be ontogenetically interfered with and, thus, the final product may not necessarily be a reflection of pure, "untainted" genetic unfolding. But let me pursue these premises further.

I agree with Oyama that metaphor and an appeal to "that which we all know must occur" has pervaded much of the thinking and writing about the gene and its role in ontogeny. It may be true that biochemists have isolated the chromosome and the specific locus on it that is associated with individuals who, for example, develop Duchene's syndrome, and we may learn of the various chemical attributes expressed in the individual who has, for example, Duchene's syndrome, but we do not yet know *how* that gene actually goes about *inducing* the development and expression of Duchene's syndrome. Furthermore, just because it may be possible to identify the chemical deficiencies or at least signals that are associated with various congenital afflictions, and to thereby be able to administer chemicals that might minimize, counter, or control any given affliction, does not get us any closer to the issue at hand here, which is whether or not one can make the assertion that the ontogeny of that affliction is understood. But it is quite common to see in the literature statements to the effect that "the cause of such-and-such is genetic." Probably true. But the assertion—no matter how true it might be—gets us no closer to understanding what the underlying, specific genetic code is and how that genetic message is translated developmentally, and how much of that genetic message has bearing directly on the ontogenetic outcome of the individual. So, even if we can point to a genetic locus on a chromosome and declare that it is associated with some trait of an individual, the information on how that gene produced that trait in question is still lacking. We must, at best, then, resort to unclear notions of development having a genetic basis. But it would seem not necessarily wrong to do so.

Much research on the developmental "messages" encoded in early progenitor cells has taken place within the past twenty years, especially, by such notables as Wolpert, Le Douarin, Noden, Summerbell, Tickle, Kollar, Miller, and Till, to name but a few. The essence of a large part of this work is to determine if and, if so, how, the development of structures (such as teeth or limbs) are controlled. Is the necessary "information" intrinsic to the progenitor cells themselves, from which the "information" is passed on to subsequent generations of cells, and thus the development of a structure and its constituent elements are truly the result of an ontogenetic "unfolding"? Or is the source of developmental "information" external to the duplicating mass of undifferentiated cells, which become induced to take on specific roles or to contribute to specific morphologies?

The latter interpretation of development had been the dominant—actually, the only—one until recently. Beginning decades ago, Butler (e.g. 1939, 1978) adapted a version of this perception of development to explain how mammals could have teeth of differing shapes along their jaws (in contrast to the typically uniform tooth type found along the jaws of reptiles), and how different species of mammals could have teeth of different shapes in the same positions along the jaw. Butler suggested that undifferentiated (in terms of cell function or shape-producing potential) cell masses come to be distributed along the embryonic jaws and that the shape of the tooth that each cell mass would eventually produce is determined by "information" transferred from an external source. In this case, Butler referred to the external source as a "morphogenetic field." There were supposed to be three morphogenetic fields: an anterior one that would produce incisor teeth, a middle field that would produce canine teeth, and posterior field that would produce premolar and molar teeth. Humans and dogs, for example, would have these three fields in operation, producing teeth of expected shapes in their expected positions. Shrews and hedgehogs, which possess a caniniform tooth at the front of the jaw, behind which are a string of premolariform teeth followed by the expected molar teeth, would have had the incisor morphogenetic field swamped by an anteriorly displaced canine morphogenetic field, behind which there would be a greatly expanded premolar and molar producing field. Shrews and hedgehogs would still have incisors, canines, premolars, and molars in their expected positions (incisors in the premaxilla, the canine just posterior to the premaxillary-maxillary suture, followed by premolars and then molars), but the anteriormost teeth, at least, would not look like the teeth whose names—descriptive names—they bore.

But the 1970s saw a burst of experimental energy in the realm of developmental biology that added a new dimension to the interpretation of the determination of structural shape and to the understanding of the development of segmentation and merismatic structures, i.e. the underlying causes of positional information and pattern formation (e.g. see the seminal papers by Wolpert, 1969, 1971). Wolpert, among others, was interested in how, for example, the limb bud (the chick embryo was used), with its obvious patterning of bone number and size along an axial "gradient," actually came to acquire these attributes, attributes which in quality and kind obtain equally to other areas of anatomical differentiation (e.g. the lower limb, the vertebral column, teeth). The traditional interpretation was that, as the limb became enlarged and elongate through the cellular proliferation of structurally noncommitted cells, a chemical "message," which coursed along the developing limb from some "source" to the terminal end (the "sink"), "encoded" the cells in various regions to form the structures that would distinguish each region. Through experiments that, by extirpation and transplantation, tested the committedness or noncommittedness to structural development of cells in various parts of the growing limb bud, Wolpert concluded that, in fact, the information necessary for the differentiation of the components of the limb was already contained in the initial cell mass (the stem progenitor cells of the progress zone) of the incipient limb bud and that structural differentiation as the limb bud grew was determined by the cells of the progress zone, which lay just underneath the apical ectodermal ridge (= the "leading edge") of the developing limb. The different "messages", which determined the spatial patterning along the limb, were a function of the age of the cells of the progress zone at any given time during development. That is, the hierarchically nested sets of encoded messages (if I may borrow from current formulations of hierarchy theory in general in evolutionary biology [see review by Greene, 1987]), reach their potential of expression and structural specificity at various times, so that, for instance, at time X, cells in the progress zone would produce structure A, whereas, at time X + 1, cells in the progress zone will produce structure B. Cells encoded, for example, to produce a radius or an ulna will only do so after the humerus has been initiated and, if extirpated, will still do so *and* will continue to proliferate and produce the distal remainder of the limb. In general terms, a relatively small number of (stem progenitor) cells seems to carry all the information necessary to generate a feature as large and as complex as a limb. Thus there would indeed appear to be an embryological "unfolding" or "evolution" of structure.

The ontogenetic independence of anatomical systems has also been suggested by experimental studies on tooth development (Lumsden, 1979). In this case, Lumsden transplanted the presumptive, pre-visible, region of tooth formation of the first lower molar of mice into the anterior ocular chamber of recipient mice, which is an environment devoid of vascular and nervous supply, or any other type of cell or substance that could influence the development of the presumptive first molar cells. Under optimum conditions, Lumsden found that not only did a first molar—recognizable in all morphological detail—develop, but that a second and third molar also formed. Thus, as limb development produces a structure with a regular pattern, so does (at least mouse lower) molar development. Furthermore, it is obvious that the entire molar class (of the mouse, at least) results from the continued cellular proliferation and concomitant "release" of nested sets of ontogenetic "information." Given the seemingly contradictory results of Miller (1971), who concluded that it was the oral epithelium that played the dominant role in tooth forming tissue interaction, and Kollar (1972), who thought it was the underlying ectomesenchyme, Lumsden (p. 101) suggested "that in the 9–10 day mouse embryo there is a region of oral epithelium which specifies beneath it a zone of ectomesenchymal stem cells which subsequently grows to lay out the patter of the molar dentition."

Osborn (1978) drew upon the notion of pattern formation suggested by the work of Wolpert and others on chick limb buds and Lumsden on mouse molar teeth and proposed his alternative to Butler's "morphogenetic field theory of tooth shape." What Osborn dubbed the "clone model" hypothesized that there were initially individual stem cell progenitors for each tooth class (incisor, canine, molar) and that the subsequent number of teeth in a particular tooth class, and the extent to which the teeth in a tooth class exhibit a gradient of shape, was determined by the proliferation of the stem cell mass and the potential for the production of teeth that each stem cell mass possessed initially. Tooth classes were delineated by the morphological discontinuities between them. Osborn's "clone model" assumed that all mammals would develop some representation of each tooth class and thus he did not deal with the contradictions in morphology between, for example, the incisiform incisors that occur at the front of the jaw of humans and the caniniform incisor at the front of the jaw of a shrew. To try to accommodate this apparent inconsistency, I (e.g. 1982) proposed that tooth shape reflected its class membership and that a mammal may not necessarily develop teeth representative of each tooth class. Thus, in the case of the shrew, only stem cells that would produce a single tooth in the canine class (at the front of the jaw) and those that would generate an extensive molar class (comprising six premolariform and two molariform teeth) would invade the presumptive jaws. Humans, on the other hand, as indicated morphologically as well as by the embryological studies of Ooë (e.g. 1957; see Schwartz 1982, for discussion), would be interpreted as having the stem cell progenitors of each tooth class. Teeth would be found along the jaws not because incisors, canines, and the rest always occupy the same positions in the jaws of all mammals, but as a result of the deposition or activation of some number of original stem progenitors and their subsequent potential to produce teeth.

At this point I think it is acceptable to conclude that there are certainly tantalizing if not more tangible reasons for acknowledging that (at least in some, if not many, if not all cases) a final, fully expressed, and anatomically complex structure, such as a limb, or system, such as a string of teeth, is not necessarily the composite result of selection acting on each individual element that makes up the total package. Since it is becoming clearer by the year that we can trace stem cells of various systems back to neural crest cells, and it is well known that neural crest cells themselves become distinct early on in embryogenesis, and these cells in turn derive ultimately from the original zygote, there is an apparent ontogenetic hierarchy of levels of cellular information coding for different levels of specificity and differentiation. We need only make the leap to linking everything to the original genetic "stuff" of the zygote to get the full sense of predetermination. But can we then conclude, to paraphrase Dawkins, that if there is any sense in which teeth are inherited, then so, in the same sense, may behavior? That the ontogeny of behavioral and, in the case of humans, cultural attributes or, more precisely, complexes, follows a course similar to the zygote's differentiating into specific cells, such as neural crest cells, which, in turn proliferate and differentiate into yet other cells with more specific roles and functions, and so on, until a limb or set of teeth form?

Oyama may accept Dawkins's statement about similarity in the inheritance of anatomy and behavior, but she does so in the context of arguing that even the non-behavioral morphology of an organism is not tied to "the gene," that there are a series of potential "environments" through which the developing feature under scrutiny will pass and under whose influence it could be subject. For instance, molecules are in the environment of other molecules, genes other genes, cells other cells, hormones other hormones, and organs other organs, much less the more commonly thought of case in which the organism is in the environment of other organisms, etc. The argument, therefore, is that one cannot be certain when "the genetic code" is being played out unaltered and when it has veered off to the beat of a slightly different drummer. Epigenetic phenomena (e.g., Gould 1977; Løvtrup 1974, 1981a, 1981b)—at all levels—represent, therefore, pathways of producing not only novelty in the profound phylogenetic sense (e.g., in the sense of the advent of new species), but of maintaining what we perceive of as "normal." Thus it is even more unclear and undeterminable exactly what one is referring to when one speaks of the "genetic basis" of this, that, or any other feature, behavioral or otherwise.

As a student of methods of and problems involved in phylogenetic reconstruction I suggest that some, if not all, of the issues of debate and disagreement are not dissimilar from arguments that abound about the nature and delineation of homology. Recognizing the importance of inheritance is a large part of phylogenetic investigation. For, after all, are not homologous features those similarities that are shared by organisms by virtue of their having had an ancestor from which they inherited the feature or features they hold in common? "Proving" that similarity does indeed represent homology is another matter altogether, but it does seem that a logical and instructive approach is to test one theory of homology with others with the end result being the generation of competing, alternative theories of homology, of which the most robust (= most highly corroborated) would emerge as the most likely (e.g. see Eldredge and Cracraft 1980, for review and references). The taxonomic inclusiveness which the corroboration of homology circumscribes in turn delineates monophyly and, thus, relatedness. If behavioral and non-behavioral attributes have indeed been inherited by the organisms under consideration, and inheritance would apply to ancestor-descendent relationships just as much as to those between parents and offspring, it could be argued that a series of tests of apparently similar features would yield a number of potential theories of relatedness, of which the preferred would elucidate homology and, thus, serve as a guide to the likelihood of inheritance. Even if there is a largely epigenetic basis for the production of any or all of the hypothesized homologous features, by definition these features must be inherited. It must, of course, be borne in mind that the chosing of one of the available theories of homology demands the recognition of all other similarities among other combinations of taxa as non-homologous features whose existence thereby falls more easily victim to arguments of environmental rather than genetic influence.

Although I found each book easily accessible and, especially in instances where I would disagree with the intent or conclusion of the author, that the arguments of each author were usually well thought out, referenced, and presented, it should be obvious that I found Oyama's work the more challenging and stimulating. There is still alot to be hashed out in the arena of sociobiology and its application and applicability to the interpretation of human behavior and culture. In many instances, when the dust of the current debates has settled, we will probably discover that we are not appreciably closer to a single explanation or all-encompassing theory, that some of the arguments have been on the wrong level, and that some of the questions were not even the right ones to begin with.

REFERENCES

Butler, P. M.
1939 Studies on the Mammalian Dentition. Differentiation of the Postcanine dentition. Proceedings of the Zoological Society of London 109: 1–36.

Butler, P. M.
1978 The Ontogeny of Mammalian Heterodonty. Journale Biologique Buccale 6: 217–227.

Darwin, C.
1871 The Descent of Man. Vol. 1. London: John Murray.

Dawkins, R.
1976 The Selfish Gene. Oxford: Oxford University Press.
1983 The Extended Phenotype. Oxford: Oxford University Press.

de Beer, G.
1958 Embryos and Ancestors. Oxford: Oxford University Press.

Eisley, L.
1958 Darwin's Century. New York: Doubleday & Co.

Eldredge, N., and Cracraft, J.
1980 Phylogenetic Patterns and the Evolutionary Process. New York: Columbia University Press.

Gould, S. J.
1977 Ontogeny and Phylogeny. Cambridge, Massachusetts: Harvard University Press.

Greene, M.
1987 Hierarchies in Biology. American Scientist 75:478–489.

Irvine, W.
1959 Apes, Angels, and Victorians. Cleveland: Meridian Books.

Kollar, E. J.
1972 Histogenetic Aspects of Dermal-Epidermal Interactions. *In* Developmental Aspects of Oral Biology. H. C. Slavkin and L. A. Bavetta, eds. pp. 125–149. New York: Academic Press.

Løvtrup, S.
1974 Epigenetics. A Treatment of Theoretical Biology. London: J. Wiley.
1981a Introduction to Evolutionary Epigenetics. *In* Evolution Today, Proceedings of the Second International Congress of Systematic and Evolutionary Biology. G. G. G. Scudder and J. L. Reveal, eds. pp. 139–144. Pittsburgh: Hunt Institute for Botanical Documentation, Carnegie Mellon University.

1981b The Epigenetic Utilization of the Genomic Message. *In* Evolution Today, Proceedings of the Second International Congress of Systematic and Evolutionary Biology. G. G. G. Scudder and J. L. Reveal, eds. Pp. 145–161. Pittsburgh: Hunt Institute for Botanical Documentation, Carnegie Mellon University.

Lumsden, A. G. S.
1979 Pattern Formation in the Molar Dentition of the Mouse. Journale Biologique Buccale 6: 77–103.

Lumsden, S. J., and E. O. Wilson
1981 Genes, Mind, and Culture. Cambridge, Massachusetts: Harvard University Press.

Miller, W. A.
1971 Early Dental Development in Mice. *In* Dental Morphology and Evolution. A. A. Dahlberg, ed. pp. 31–44. Chicago: University of Chicago Press.

Monod, J.
1971 Chance and Necessity. New York: Knopf.

Ooë, T.
1957 On the Early Development of the Human Dental Lamina. Okajimas Folia Anatomica Japonica 30:197–211.

Osborn, J. W.
1978 Morphogenetic Gradients: Fields versus Clones. *In* Development, Function, and Evolution of Teeth. P. M. Butler and K. A. Joysey, eds. pp. 171–201. New York: Academic Press.

Schwartz, J. H.
1982 Morphological Approach to Heterodonty and Homology. In Teeth: Form, Function and Evolution. B. Kurten, ed. pp. 123–144. New York: Columbia University Press.
1987 The Red Ape: Orang-utans and Human Origins. Boston: Houghton Mifflin.

Wilson, E. O.
1975 Sociobiology: The New Synthesis. Cambridge, MA: Harvard University (Belknap) Press.

Wolpert, L.
1969 Positional Information and the Spatial Pattern of Cellular Differentiation. Journal of Theoretical Biology 25:1–47.
1971 Positional Information and Pattern Formation. Current Topics in Developmental Biology 6: 183–224.