

THE CHRISTIAN VISION

**MAN AND CREATION**  
Perspectives on Science and Theology

Michael Bauman, Executive Editor  
Lissa Roche, General Editor  
Jon Corombos, Editorial Assistant

Hillsdale College Press  
Hillsdale, Michigan 49242

## Contents

Introduction .....	1
God and Creation: An Historical Look at Encounters Between Christianity and Science <i>Mark A. Kalthoff</i> .....	5
The Evolution of Scientific Creationism <i>Ronald L. Numbers</i> .....	31
Seven Patterns for Relating Science and Theology <i>Richard H. Bube</i> .....	75
Creation Science and Methodological Naturalism <i>J. P. Moreland</i> .....	105
When Faith and Reason Meet <i>Howard J. Van Till</i> .....	141
The Star of Bethlehem: Science of the Ancients <i>Craig Chester</i> .....	165
What Is Darwinism? <i>Phillip E. Johnson</i> .....	177
A Biologist's Approach to Human Nature <i>Richard Alexander</i> .....	191

*Hillsdale College Press*

Books by the Hillsdale College Press include *The Christian Vision* series; the *Champions of Freedom* series; and other works.

The views expressed in this volume are not necessarily the views of Hillsdale College.

The Christian Vision Series  
MAN AND CREATION:  
PERSPECTIVES ON SCIENCE AND THEOLOGY  
© 1993 by the Hillsdale College Press  
Hillsdale, Michigan 49242

Printed in the United States of America

Photo: Andromeda Galaxy, Photri, Inc.

All rights reserved. No part of this publication may be reproduced without the prior written permission of the publisher.

First printing 1993  
Library of Congress Catalog Card Number 93-078627  
ISBN 0-916308-74-X

must creation have occurred if we assume that God had nothing to do with it?" Theistic evolutionists accomplish very little by trying to Christianize the answer to a question that comes straight from the agenda of scientific naturalism. What we need to do instead is to challenge the assumption that the only questions worth asking are the ones that assume that naturalism is true.

Notes

1. Niles Eldredge, *Time Frames* (Heinemann, 1986), 144.
2. *Ibid.*, 93.
3. Stephen Jay Gould, "Is a New and General Theory of Evolution Emerging?" *Paleobiology*, 6 (1980), 119–130, reprinted in Maynard Smith, ed., *Evolution Now: A Century After Darwin* (W. H. Freeman, 1982).
4. See Stephen Jay Gould, "Impeaching a Self-Appointed Judge," *Scientific American*, (July 1992), 118–122. *Scientific American* refused to publish my response to this attack, but the response did appear in the March 1993 issue of *Perspectives on Science and Christian Faith*, the journal of the American Scientific Affiliation.
5. Thomas S. Kuhn, *The Structure of Scientific Revolutions* 2d ed., (Chicago: University of Chicago Press, 1970), 79.

## A Biologist's Approach to Human Nature

*Richard D. Alexander*

---

Richard D. Alexander is the Theodore H. Hubbell Distinguished University Professor of Evolutionary Biology and director of the Museum of Zoology at the University of Michigan. With a Ph.D. in entomology from Ohio State University, he is the author of numerous articles and books, including *Darwinism and Human Affairs* (1979), *Natural Selection and Social Behavior: Recent Research and New Theory* (1981), and *The Biology of Moral Systems* (1987). More recently he has written on the evolution of the human psyche, mechanisms of kin recognition, and the reproductive significance of humor. He has also described almost 400 new species of insects and co-authored a monograph, *The Crickets of Australia* (1983).

---

Even though I am an entomologist by training, I have a deep interest in human nature that existed throughout my formal training as a biologist and accounts for my having written extensively on human behavior during the past 20 years. In retrospect I believe that much of my interest in biology and human nature derives from my childhood on a farm in central Illinois. Life on a farm, operated by horses and producing primarily livestock, helped give me an interest in things biological. The woods that almost surrounded my farm, and the river that ran near it, were full of animal and plant life that provided most of my recreation. Not least important, during my childhood the Methodist church was also an important stimulus; it was not only the location of essentially all social activities in my community, but as well the only place where anyone discussed the ultimate nature of human beings. Neither in school nor in my home was this topic taken seriously. Many years later, when I asked my mother why, she said she thought that we were too busy to

discuss such things. I suspect, however, that with more thought she might have agreed that, even though both of my parents were former school teachers, we didn't know enough about the basis for human nature to discuss it. And I also suspect that my household was not different from most others in this regard.

I also remember being frustrated with church, however, beginning at the age of about 12 years, because it seemed to me that one could not easily raise his hand and ask questions or challenge most things being said. I recall considering the fact that, while in the schoolroom I was usually encouraged to question what was said, ironically, basic problems in understanding human nature were not discussed there.

In the announcement of this lecture, someone referred to a line in Robert Frost's poem, *Mending a Wall*, "Good fences make good neighbors." I have used this poem for many years in my course on evolution and human behavior to make a point about analysis that distinguishes science from the humanities. The point comes from my high school English class, in the closest reference to basic human nature that I can recall being made during my elementary and high school days. Frost and his neighbor had an ancient stone wall along the property line between them, with pine trees on one side and an orchard on the other. Each year Frost's neighbor insisted that he and Frost walk the two sides of the wall, replacing the stones that had fallen off during the year. Frost asked his neighbor teasingly why they needed to do this any longer, since there had been no cattle or other animals on either side for a long time and pine cones could not cross the fence and eat apples or vice versa. The neighbor always answered simply, "Good fences make good neighbors." My high school English teacher interpreted this poem as indicating that farmers, somewhat conservative people who do not like to change their ways, thus are likely to cling to an old idea even when it is no longer applicable. I raised my hand to offer a different interpretation. I suggested that when two neighbors walk the property line between them, repairing the fence, they are likely to renew their acquaintanceship and talk about everything that is important to

them. Walking along together on either side and discussing whatever problems might come to their minds was an opportunity to get to know each other again and to re-establish a friendship and common understanding. That was my interpretation of the poem—my metaphor for its message. It is also my metaphor for the relationship between science and the humanities and religion. I think the humanities—and in many respects religion as well—are characterized by the fact that there is no way to decide upon one "correct" meaning of, say, a poem or story or work of art. Even if Frost were here today we couldn't necessarily rely on what he told us is the correct interpretation of his poem. Anyone might change his mind as time goes along, and if someone ever suggested an interpretation that Frost liked a lot better than whatever one he might have placed on it when he wrote it, he might just change to the new meaning and we would never know it. This kind of analysis—trying to decide upon the personal or most significant meaning to you or me of a human intellectual or emotional work—is of course in no way trivial. Anyone can derive great inspiration from such efforts and thereby literally change one or many lives. Everyone, I suspect, shares the feeling that a poem, or any artistic or literary or religious theme, can be a wonderful thing. Part of the beauty is that anyone can make his own interpretation, which may provide a solution for whatever question or problem or decision seems most important or interesting to him or her at the time.

But neither religious nor "humanistic" kinds of interpretations describe well how I have spent my career as a biologist exploring human nature. The scientific approach, which I hope is the one I have engaged, might be said to be a seeking of things undeniable—what we sometimes try to label as "facts"—even, in the end, things undeniable about the background or function of human endeavors such as art, music, drama, literature, humor, and other activities labelled as humanities—even of religion. An undeniable thing, such as that the earth is not flat but rather somewhat spherical, is a piece of knowledge that simply cannot any longer be denied; essentially, anyone who tries to deny it is likely to be ridiculed

or thought to be something of a crackpot because of the evidence supporting it, which is also contrary to all suggested alternative ideas. This state of affairs can prevail even though we all realize that virtually any fact can be overturned if new evidence becomes available that does a better job of contradicting it than the current evidence does supporting it. Even though I remain fascinated by all forms of literary, religious, and other forms of human endeavor, as far as my formal career of attempting to understand human nature is concerned, scientific analysis supplanted whatever else had impressed or interested me. The point of my presentation today is to argue that scientific analysis of human nature can continue on all fronts—including evolutionary themes—without necessarily becoming adversarial to religious approaches or any aspect of the humanities. I do not believe that there is any necessary incompatibility between scientific and other kinds of analyses of humans or any other natural phenomenon, even though I believe that avoiding such adversarial relations requires thoughtfulness and concessions on both sides. I am referring to the relationship between science—here evolutionary biology—and religion and the humanities because I believe it is the theme of this symposium, and because I have long been interested in understanding the similarities and differences between science and the humanities and religion as ways of thinking about human nature and the products of human nature.

If, for example, we were to take the attitude that every seemingly unsolvable problem in human nature happened because of special creation by a supernatural being, and that's all there is to it, then we might be caused to give up on further analysis and understanding, especially whenever we encountered anything really puzzling or difficult. We might believe that we could not or should not continue. Yet scientists are most likely to answer crucial questions by focusing deliberately on the seemingly insoluble as the best possible challenge and the likely most important or general problem.

Despite whatever we may know or think now about ourselves, no one can doubt that the world is full of human

misery. People often do not know why they do the things they do, or why others behave as they do. Governments know they don't know enough about how to govern people. No one understands well enough how people interact collectively. Countless unfortunate things are happening continually to humans all over the world which could surely be changed for the better by additional knowledge about humans that might be gained from scientific study. I believe that the way we approach such solutions is by continuing unrestricted analysis of ourselves and our history on every front.

Here I want to take a single example and illustrate briefly an analytical approach from a biological viewpoint. I hope I can show that such an approach can lead to findings and conclusions that not only are extremely important, but that were not intuitively clear beforehand.

I choose the human mortality curve (Figure 1) as my example of a human trait to analyze. When people look at a mortality curve they may often think it is something one cannot do much about, but I will suggest that there is much we can alter about it, and that how to think about it and change it in desirable ways becomes much more apparent when it is analyzed in detail in terms of our extended history of evolution by natural selection.

At first it may not seem likely that the mortality curve is the same for all humans everywhere. But, at least in a general way, it is. Admittedly, there will be little bumps that change from one situation to another, as when a war causes young men to die at a higher rate. Prior to medical technology the curve rose at a higher rate than it does today. Child mortality obviously varies in different circumstances. But, generally speaking, the mortality curve is a trait of humans as certainly as are five fingers, two eyes, menopause, concealed ovulation, a large complex brain, or a certain developmental pattern. It's a part of the life pattern of humans according to which we all must live. The curve in Figure 1 is a plot of age-specific mortality across the human lifetime. The horizontal axis shows changes in age, the vertical axis deaths per 1000 per year. Males and females are plotted differently

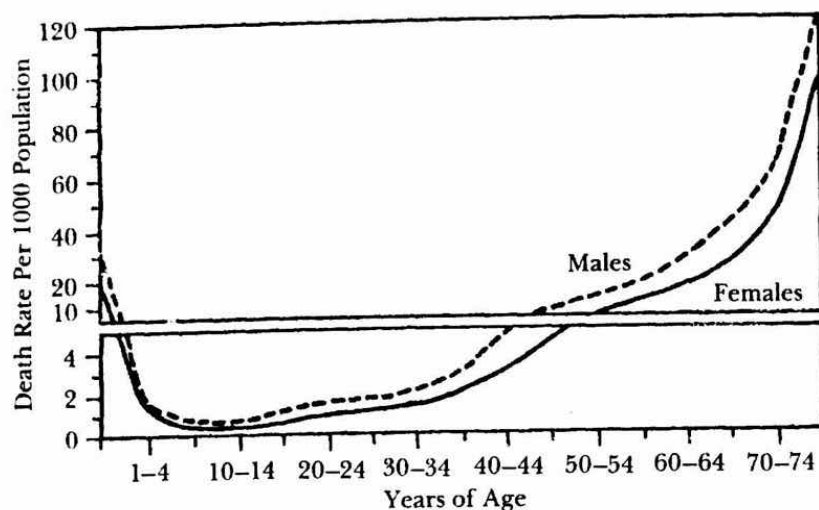


Figure 1. Lifetime mortality curves for human males (solid line) and females (dashed line) in modern America in 1950 (from Alexander, 1987). The general shape of the curve remains the same across the world, and the differences between the sexes also remain about the same. The implication is that human lifetimes follow predictable programs, evolved as a result of natural selection (data from U. S. Department of Health, Education, and Welfare, National Office of Vital Statistics, Special Report 37, 1953).

because their mortality rates are different in the same way all over the world.

How can one approach an analysis of this curve? First, we can imagine it as divided into sections that can be examined somewhat separately. One section could involve the increase in mortality during early and middle adult lifetimes. This increase is generally described as owing to senescence, defined as a gradual increase in susceptibility to environmental insults such as diseases and accidents. Why does this increase in susceptibility take place? One might think we simply wear out. This hypothesis, however, makes little sense when we realize that unlike nonliving things, living organisms are not composed of static materials but are constantly changing the molecules that compose them; how could we "wear out" if this is the case? Moreover, each of us begins as

a single cell and eventually develop into an organism containing billions of cells, yet, as the mortality curve shows, later in life we cannot even maintain this body; this change in ability to maintain ourselves is what we must explain. Finally, in different species, the individual organisms wear out at greatly different ages—most species in a few hours, weeks, or months, others such as elephants, parrots, turtles, and humans in a hundred years or more, and redwood and bristlecone pine trees only after a few thousand years. Why should such enormous differences among species occur if organisms simply wear out? Some people have noted that only a finite number of cell doublings occur in culture tissues and thought that this limit is what decides lifetimes. But, again, why is the number of cell doublings different in different organisms? Still others have noted that toxic materials accumulate in our bodies and suppose that this is the reason for deterioration. But we still must answer why should they accumulate in a few hours in some organisms and across several thousand years in others. The method I am using here to eliminate certain hypotheses that have been erected to explain senescence is called the comparative method. By examining the array of different kinds of organisms and comparing them to humans, we can falsify many hypotheses invoked as general explanations for something that happens in a single species.

Biologists such as Peter Medawar and George Williams took an approach to this problem of advancing senescence that was quite different from those of their predecessors. They noted that mortality inevitably occurs as a result of accidents, predation, and disease. They also realized that this accumulating mortality gradually reduces the reproductive significance of events happening later and later in life because many organisms are dying, leaving their genetic materials no chance to reproduce themselves, and the residual reproductive possibilities for the organism are being reduced. In other words, any genetic element which contributes the same positive effect toward the maintenance of the organism in which it finds itself throughout the life of the organism cannot create as large a benefit later as it does

earlier. The later effects of the genetic unit cannot affect its own spread as much, via benefits to the reproduction of the organism, because most individuals will have died, diluting its later effects, and those still alive at advanced ages will have less of their reproduction remaining. The consequence is that, if conflict ever occurs between early and late effects, the early effect will tend to win. There are two relevant circumstances: first, genes may have multiple effects, some earlier than others. Because development is a unitary phenomenon it is probable that all genes have multiple effects. As one biologist put it, because of the unity of the individual organism, all genes affect the action of all other genes. Genes spread by contributing to the reproduction of the whole organism. If different effects of the same gene are adversarial (some beneficial, some deleterious), early effects tend to be worth more than later effects: indeed, a gene may be saved (may reproduce itself) because of its beneficial early effects even if there are also inevitably accompanying late deleterious effects. Or if a gene only gives a beneficial effect for a short period during the lifetime and is neutral at other times, such time-specific effects will be more beneficial if they occur early. Whenever the beneficial effect is not occurring, the result is in effect deleterious. In a third case, if a gene has precisely the same effect throughout life, but the individual's lifetime changes so that the effect changes from being beneficial to being deleterious, exactly the same consequences will result (for example, if I had a gene that gave me an inclination to play touch football during the noon hour it would long ago have ceased to have any beneficial effects and become increasingly deleterious!).

The overall result of the process I have described as resulting from these three "kinds" of genetic units is that over long periods of natural selection there will be an accumulation of beneficial effects early in life and an accumulation of incidentally accompanying deleterious effects (or lack of beneficial effects) later in life. This is the basis for the "pleiotropic" theory of senescence, published 36 years ago (Williams, 1957); it was named for the phenomenon of pleiotropy, or multiple effects of genes. That senescence remains

of great importance is indicated not only by peoples' concern with the finiteness of individual existence, but also by the fact that long series of papers and books have been published on senescence, many within the past few years. One reason for continued attention is the implication, seen repeatedly in newspapers and other popular publications, that there is a medical possibility of increasing the human lifetime dramatically, perhaps even doubling or tripling it. The pleiotropic theory, however, does not support this prediction. Nor does the effect of 100 years of medical technology (Fries, 1980), which appears mainly to have reduced the likelihood of premature deaths, but not increased the longest lives, or what demographers call the maximum average life length. These are not trivial facts, considering the amount of money used in gerontological research under the presumption that massive increases can be effected in the human lifetime.

But does the pleiotropic theory continue to be supportable with closer scrutiny? Let us see. One of its predictions obviously is that over long periods of natural selection a generally higher mortality will lead to higher rates of senescence, the reason being that the higher the mortality rate the greater the reduction of the reproductive significance of gene effects that occur late in life. Therefore, species with higher rates of mortality should have shorter average maximum lifetimes, and they do. Even within species the prediction holds. Men die accidentally and as a result of within-species competition more often than women: they have a higher mortality rate. As predicted from the pleiotropic theory, men also have a higher rate of senescence, and this is the fundamental reason why there are much larger numbers of widows than widowers everywhere in the world and more women than men in homes for elderly people. It is a further prediction that this gender gap will not disappear just because women undertake the same kinds of risky lifetimes that men have led across human history. Because of the residual genetic difference that must have appeared between the two sexes with respect to senescence rates, the gap will only narrow. Another prediction is that the more a population deviates from monogamy, over a sufficiently long pe-

riod, the greater will be the difference in senescence rates between the two sexes because polygyny tends to cause the two sexes to lead different kinds of lives, in which, under increasingly extreme polygyny, males tend to take higher risks than do females, thus to die at a higher rate from accidents and within-species competition and combat.

One more dramatic example tests the pleiotropic theory by comparative study of mortality rates. In most of the so-called social (or eusocial) insects there tends to be a single reproducing queen living with tens, hundreds, thousands, or millions of her own offspring; in the Hymenoptera (wasps, bees, and ants) the colony—no matter what its size—is a one-parent nuclear family. The workers and the queen have the same kinds of genes: their differences are determined only by the kind of food they receive during development—an aspect of the environment. Nevertheless, the workers have short lives because their particular phenotype has throughout history undertaken the tasks in the colony that more often lead to mortality—searches for food and defense of the queen and the nest. The queen, in contrast, remains inside the nest and is protected there by the workers. In honeybees, for example, the difference in life length ranges from a few weeks of life for a worker to a few years for a queen. Because the queen's phenotype (body, soma) has, across history, tended to outlive the worker kind of phenotype as a result of being kept safe from accidents and predation, that kind of phenotype has also evolved to undergo a relatively slow rate of senescence. Regardless of how well one treats a queen or a worker, the queen lives many times as long as a worker. Thus, the eusocial insects very strongly support the pleiotropic theory of senescence, and in a rather remarkable way.

In still another test of the pleiotropic theory, we can ask why some organisms die suddenly without much evidence of senescence, quite unlike our own gradual increase in mortality across the adult lifetime. The answer again supports the pleiotropic theory. The organisms that die suddenly without evidence of gradual senescence are those, like salmon and soybeans, that reproduce only once in their life-

times. Ecologists call them "semelparous" (one-time breeders) and ourselves "iteroparous" (iterative or many-time breeders). For a semelparous organism the last reproductive act is the same as the first one, hence is always identifiable. Once an organism has reproduced for the last time, natural selection can no longer protect it from any source of mortality, meaning that if it dies, for whatever reason, because no reproductive possibilities remain there can be no further selection, hence no tendency to remove the source of death. On the other hand, if no act of reproduction is reliably the last one, as in iteroparous organisms like ourselves, then some selection, however weak, will remain against mortality. Long-term selection thus causes semelparous organisms to tend to die suddenly just following reproduction, and iteroparous organisms to become gradually more susceptible to mortality, thus to senesce gradually. Again, the pleiotropic theory is strongly supported by comparative analysis of different kinds of organisms, and so we are increasingly led to believe that it forms the basis for understanding our own mortality curve and what can be done about it.

Why is medical technology unlikely to increase human lifetimes dramatically? What does the pleiotropic theory of senescence predict will happen at the end of life? The chances of reproducing again at this stage of life are almost nothing. Throughout history selection very late in life would be expected to operate as follows: One or another source of mortality will tend to be most important, and when this is the case selection automatically will work more intensely against the most important source until its effects are reduced so that another source of mortality exceeds it in its reproductive effects. Again, selection will work most intensely to reduce the effects of the new source of mortality, but only until still another source exceeds it. This process will continue, its long-term overall effects being to leave an increasingly great number of sources of mortality lurking just "below the surface" to affect the aging organism. To reduce the effects of any one—or even several or many—of these multiple sources of death cannot have a dramatic effect on life lengths. Medical scientists have recently begun to real-



ize the trivial effects on population structure of cures for individual problems that occur very late in life, even before they began to understand the pleiotropic theory of senescence. With this theory they know the reason.

If the pleiotropic theory of senescence is the general explanation for mortality curves, medical technology can primarily remove premature causes of death, thereby making the curve more angular (Fries, 1980), but is unlikely to increase substantially the average maximum lifetime. This I believe is an astonishing and non-intuitive result with enormous significance for the support of research.

There actually is a way to extend the average maximum human lifetime that is consistent with the pleiotropic theory of senescence, but not many people are likely to engage in it. It is simply to have one's reproductive organs removed—to be castrated. We know this not only from domestic animals but from the castrati—humans castrated within recorded history, either accidentally, or on purpose to serve in harems or because of the effects on their singing voices. Just abstaining from sex and other reproductive activities won't delay senescence (though it might reduce the likelihood of certain kinds of accidental and competitive sources of mortality in the individual's lifetime) because our unaltered bodies are still programmed by evolution to senesce at particular rates. Selection has thus caused us to begin the process of senescence not at the first age of actual reproduction but at the usual first age of reproduction, whether or not we actually reproduce then. But, as several investigators have suggested from work on rats and other organisms, staying too lean to reproduce is expected to retard senescence because physiologically it is a little like castration; it inhibits activity of the reproductive organs, and in some regards changes the phenotype temporarily in a direction similar to that of castration. Of course, not everyone can imagine enjoying such a condition even more than, say, castration, and it is also true that emaciation can lead to death from other causes such as susceptibility to diseases.

A little reflection shows that the pleiotropic theory of senescence explains not only the gradually increasing rate

of mortality among adults as they age, and the very high rate near the end of life, but as well the extremely high vigor and strength of young adults. Beneficial effects of genes are concentrated among juveniles and young adults; these are the times of greatest ability to resist most sources of mortality and illnesses that lead to mortality. Genes that give their greatest benefits to juveniles and young adults will be more likely to reproduce themselves because they are influencing the greatest number of individuals with the greatest amount of their reproduction still remaining. Such genes are more likely to remain in a population.

Apparently, higher rates of mortality among juveniles than among young adults result either primarily or solely from higher vulnerability. Very young juveniles, for example, tend to be vulnerable just because they are so small, and sometimes because they lack protective structures or behaviors. Lacking suitable parental protection, such juveniles are more susceptible to predation. Compared to experienced adults, juveniles also undergo more novel tests by the environment, and sometimes more severe tests; as a result of being exposed for the first time to this or that disease, those lacking immunity or the ability to develop it tend to die out.

Why doesn't senescence begin in juveniles, or before the usual first age of reproduction? The reason is that, even though deaths are occurring, because none of the individuals in a population are reproducing, and because they are nearing the age of reproduction and as well growing and developing, their likelihoods of reproducing are going up, not down as in adults past the usual first age of reproduction. This set of circumstances causes mortality to be precisely offset by increases in reproductive values of the remaining individuals. A good way to think this through is to consider the offspring of a single parent, given that the parent must provide the protection and calories necessary to bring the offspring to reproductive success. If half of the offspring typically die on the way to adulthood and reproduction, then any individual past the period of mortality will be worth twice as much as one before it. Any individual is worth the most, reproductively, at just the usual first age of reproduction, as can easily

be seen by considering at what age one should purchase breeding stock of, say, cattle or other farm animals, if the price is the same regardless of age. The best age would be at the time of first reproduction because none would be lost to mortality before reproducing and no feed or other expenses would be required to cause them to reproduce. Now it can be seen that the reproductive value of an entire population of growing, developing juveniles will be the number of individuals multiplied by the reproductive likelihood of each. If there are half as many individuals, they will be worth twice as much, in terms of the proportionate representation of their genes in the next generation. Unlike juveniles, the reproductive probabilities of adults are diminishing continually, and that is why they senesce and juveniles do not.

Now I am going to discuss briefly one more human trait that has to do with the lifetime and the pattern of mortality across the human lifetime. The trait is menopause, and it is an extremely important trait that affects everyone of both sexes, women directly, but children and men indirectly. Menopause refers to that time in a woman's life, usually between 45 and 50 years of age, when she ceases to prepare ova, and the remainder of her body, for the act of further baby production. In layman's terms she becomes postreproductive. But we will have to examine that term "postreproductive" very carefully. Perhaps the first idea to explain menopause was that women have simply senesced so far by middle age that they cannot continue to produce offspring successfully, so they stop. But this idea leaves many unanswered questions. Why should menopause be virtually restricted to human females, and why should it be such a definite event in a woman's life if it is merely a part of senescence? Why should women have senesced so far as to cease reproduction halfway through the average maximum lifetime of humans, which is somewhere around 85 years? For a long time it was also thought that human ova become so likely to be mutated deleteriously when the mother is middle-aged that the population is damaged by the addition of damaged babies. But that hypothesis will not explain why individual mothers cease reproducing, for those that continued, de-

spite the occasional problem, would surely outreproduce those who abstained. A third hypothesis derived from the realization that humans have added about 50 years to their average maximum lifetimes during the long period of their evolutionary history (we surmise this by comparing the lifetimes of the primates most similar to us, and by evidence from the archaeological record of humans). This added period of life is essentially all postmenopausal for women, so some people argued that selection actually lengthened men's lives and only incidentally dragged women's lifetimes along with it, the correlate being that men continue to make sperm (hence, to be at least potentially directly reproductive) all their lives. The falsifier for this proposition appears to be that women typically outlive men. But the question remained how natural selection could add almost 50 years to the human female's lifetime if it was all postreproductive.

If we attempt to use comparative method to understand menopause, as with several human attributes (such as concealment of ovulation, our uniquely large and complex brains, or our unique sociality), we find nothing among nonhuman species that strictly compares with human menopause. Perhaps some whales, elephants, and maybe a few other species such as horses, have something near the ends of their lives that could be a rudimentary menopause. But nothing like menopause in humans seems to exist in any other species. What else is distinctive about humans that might be relevant? Humans are one of the most extensively and intensively parental organisms. Unlike practically all other species they tend their offspring until they, the parents, themselves die. Indeed, through wills and bequests we humans arrange to provide for our offspring even long after our deaths. The hypothesis was generated, again by George Williams in his 1957 paper on the pleiotropic theory of senescence, that menopause evolved because a time came in a woman's life when it became more reproductive for her to tend the descendants she had already produced than to produce additional ones. This hypothesis has been expanded to include the possibility that women actually undergo changes at the time of menopause that are considerably more pro-

found and involves turning them into much more politically inclined individuals than before, so that in fact they are overseeing the fates of not only their own offspring and grandchildren, but at least sometimes the entire clan of relatives that are reproductively important to them (Alexander, 1990). This hypothesis may be able to explain why elephants and horses and whales give some evidence of rudimentary menopause. They, too, are highly parental, and they live in female-dominated groups of close relatives. As William D. Hamilton showed in 1964, a gene can contribute to its own spread and persistence not only by causing its bearer to produce and assist offspring but also by causing it to assist non-descendant relatives such as nephews, nieces, and cousins. This is true because, just like descendant relatives, non-descendant relatives carry the genes of an individual in proportions that are correlated with social circumstances that can be used to set up particular patterns of social interaction. In other words, genes can increase their spread and persistence through causing their bearers to aid known non-descendant relatives as surely as they can by causing parental or grandparental care. And the more relatives one can help at once, the more likely any genetic unit contributing to such help can spread itself and become consolidated in the species as a whole.

I have discussed only one example of human attributes that can be analyzed by biologists, and some of the questions and possibilities that derive from it. There are many such examples. Moreover, in the space available here, I have had to deliver a greatly condensed version of a very important subject. More detailed discussions can be found in Williams, (1957) and Alexander (1987), and in recent issues of *Science* and *Nature* (e.g., see Letters, *Science*, June 11, 1993).

Senescence is not an easy example for my purposes here because it is a non-intuitive phenomenon, and it is not a direct result of selection, but rather something that happens *in spite of* selection. As my arguments indicate, selection works against senescence and susceptibility to mortality, but cannot entirely prevent it. I chose this difficult topic because it is of great importance in the everyday lives and thoughts of indi-

vidual humans and to medical practice and the distribution of research money; it is likely always to remain closely connected to issues of very great importance to humans. I felt that it illustrates well the nature of the scientific approach to basic human attributes or human nature.

Returning to the introduction, I would like to draw a fairly simple conclusion. Analyses of human nature, whether evolutionary in their approach or not, need not conflict with anyone's ideas about the nature of the universe, including the human aspects of it, and there seem to be no good reasons for foregoing such analyses. This conclusion presupposes that people will generate and cling to views of religion and other nonscientific topics in fashions and forms that admit to the usefulness of scientific analyses of even the most hallowed subjects; this is what I meant earlier by "concessions on both sides." I think it is a poor practice to adopt views that deny the validity of analytical approaches to anything in the universe, living or non-living. It is equally poor practice, however, to dismiss or disparage the ideas of people who for whatever reasons choose not to engage in this kind of analysis. There is ample reason in our world for scientific studies that seek to identify the undeniable on every hand, and also for searches for personal meaning via religion and the humanities that need not involve science or any general versions of undeniability at all. Good fences really do make good neighbors.

#### References

- Alexander, R. D. 1987. *The Biology of Moral Systems*. Hawthorne, New York: Aldine de Gruyter.
- Alexander, R. D. 1990. How did humans evolve? Reflections on the uniquely unique species. *University of Michigan Museum of Zoology Special Publication* 1: 1-38.
- Fries, J. F. 1980. Aging, natural death, and the compression of mortality. *New England Journal of Medicine*. 303:130-135.
- Hamilton, W. D. 1964. The genetical evolution of social behavior I,II. *Journal of Theoretical Biology* 7:1-52.
- Williams, G. C. 1957. Pleiotropy, natural selection, and the evolution of senescence. *Evolution* 11:398-411.