

**The Foundation for Adventist Education
Institute for Christian Teaching
Education Department – General Conference of Seventh-day Adventists**

**LIFE BEFORE DEATH:
CONTEMPLATING LIFE WHEN THE EARTH WAS NEW**

Anthony J. Zuccarelli, Ph.D.
Loma Linda University

**3rd Symposium on the Bible and Adventist Scholarship
Akumal, Riviera Maya, Estado Quintana Roo, Mexico
March 19–25, 2006**

Life before Death Contemplating Life when the Earth was New¹

Anthony J. Zuccarelli, Ph.D.

Associate Vice Chancellor for Research Affairs

Dean, Faculty of Graduate Studies

Professor of Biochemistry and Microbiology

Loma Linda University

I Am Not Resigned

A young man walked along the seashore after a day of heavy surf. The evening sun was golden in a crystalline sky. As he walked he came upon a starfish stranded on the beach. Stooping to examine it, he found that it was still alive. After a moment of thought, he tossed it gently into the ocean. Continuing his stroll, he found another just a few paces down the beach. He threw it into the waves as well. He continued to do this for each starfish in his path. Squinting against the glare of the breakers, a weathered fisherman watched from a seat on some ancient pilings. As this one-act play repeat itself, the fisherman shook his head slowly. When the young man drew near, the fisherman called out. “Lad, tis’ a useless task ye have set yourself. There be thousands of starfish on the sand and this beach goes on for many leagues. Ye canna’ make a difference!” Turning to toss another starfish into the surf, the young man called over his shoulder, “Well, it sure made a difference to that one!”

You may have heard this story before. It is a favorite of motivational speakers seeking to encourage listeners to be persistent in pursuing their goals, even when they seem overwhelming. But it says something more.

What the young man was thinking? Though we smile at his idealism, his intent seems clear. The story simply wouldn’t work if we did not understand what he was trying to do and tacitly approve. Neither did the fisherman object. He simply pointed out that the task was endless. Would he have interrupted the young man if he were simply throwing stones into the sea? Surely he would have rushed to assist if the young man were rescuing a struggling swimmer. You may imagine him helping to save a half-drowned dog or lending his hand to redirect a disoriented dolphin. But the young man was rescuing starfish. Cold, wet, stiff, slow moving, unfeeling, unintelligent, and un-cuddly. What was going on in the young man’s mind when he saw them on the shore?

It is hard to dispel the sense that something is amiss when we are in the proximity of death. Philosophers and poets crystallize that impression in words. In her poignant “Dirge without Music,” Edna St. Vincent Millay was “not resigned to the shutting away of loving hearts in the hard ground.” Dylan Thomas was belligerent as death approached his father. “Do not go gentle into that good night. Rage, rage against the dying of the light.”

Materialists may counter, “That’s just our selfish genes talking.” But what would they say to the observation that our dispute with death extends beyond the loss of friends and relatives. Something in us is not resigned to any death. The feeling is overpowering when a friend or family member is involved, but the impulse to cheat death is still strong when you see a dog desperately scrabbling to pull itself out of your swimming pool. I am probably not alone when I admit to helping a field mouse that is paddling furiously to keep its nose above the surface. I’ve been known to reach for the skimmer to aid a tarantula or even a honeybee.

The explanations of sociobiologists and evolutionary psychologists are unsatisfying both emotionally and logically. What would be more useless and nonadaptive than projecting consciousness onto a bee or spider and expending energy to save it? It has nothing to do with preservation of self or clan. We seem to be built for empathy. Where did that come from? Certainly not from a “warm pond” where creatures terrorized and devoured to survive.

Garden of Life

“And the Lord planted a garden eastward of Eden; and there he put man whom he had formed. And out of the ground made the LORD God to grow every tree that is pleasant to the sight, and good for food; the tree of life also in the midst of the garden, and the tree of knowledge of good and evil. . . And the LORD God took the man, and put him into the garden of Eden to dress it and to keep it.” (Gen 2:8, 9, 15)²

The Scriptures tell us little about life in the Garden; the text is brief and compact. As a result, it is not surprising that many of our mental images of the newly created Earth are, in fact, not in the original document. Even human immortality is implied, not explicit. Further, we get little guidance about the fate of other organisms in the time before the Fall. Restoration texts that describe the New Earth after the abolition of sin offer some help. We assume that they reflect conditions that existed in Eden, but

that assumption is not without risks. To a surprising degree, the mental images that many Adventists have of life before the Fall were painted by Ellen G. White and her sources, with technical assistance from illustrators like Harry Anderson.

Though immortality is a common theme in the theater, movies, fiction and fable, we are not familiar with things that do not die. Indeed, the cycle of birth, reproduction and death is imbedded in our consciousness and enshrined in most scientific and philosophical models of reality. Benjamin Franklin's alter ego Poor Richard expressed this universal awareness in the quip, "In this world nothing is certain except death and taxes."

Immortality is inextricably entwined with our conceptions of Eden and the New Earth. Yet, it seems to contradict both personal experience and science. This dissonance makes the pre-Fall biosphere fundamentally unrealistic and unbelievable. Serious attempts to consider a literal Eden are rare. As a consequence, some sincere Christians may feel that eternal life is something entirely beyond the reach of rational examination. Eden, an integrated, harmonious ecology supporting the existence of undying organisms, is discounted as a pleasing fable, the collective dream of pre-modern minds ignorant of the physical principles of life.

Goals

Does the story of Eden make sense scientifically? We will take several approaches to answering that question. First we will examine a collection of scientific observations that may provide a foundation for reconciling Eden with reality. These include recent biochemical, genetic and physiological studies that bear on how long individual organisms may live. From these observations we may consider the possibility of indefinitely extended life. Then we will describe concepts that may undergird a planetary biosphere in which undying beings might live. They address an equally fundamental question: Is an ecology with undying organisms sustainable? More simply, if humans (and possibly animals) did not die, could the earth survive?" Finally, using what we know or can deduce, we will attempt to approximate the biological and planetary circumstances that existed on the original Earth.

The topic is challenging. It involves issues in every domain of biological and environmental science. Certainly, it is impossible to treat them exhaustively here, even if all the information were available. Firstly, the biochemical, physiological and environmental conditions essential for immortal life are

unknown. On the other hand, there is a growing body of information relating to life extension. It may help us decide if there is a rational foundation for the idea that life can be extended indefinitely or that there is no recourse but to accept eternal life an article of faith. These goals are ambitious. They will require a degree of integration and extrapolation that some may call speculation.

Samples of Immortality

Three men were on their way to the funeral of a friend. One asked the others, “When you’re gone and your family and friends are sitting around your casket mourning your death, what would you like them to say about you?” The first man answered, “I would like to hear them say that I was a compassionate doctor and a wonderful family man.” The second replied, “I would like to hear that I was a good husband and a teacher who made a huge difference in the lives of young people.” They both turned to the questioner and asked, “What about you? What would you like to hear?” He replied, “I would like to hear them say, ‘Look, he’s moving!’”

Everyone would like to be the exception to Ben Franklin’s maxim, but in our sober moments we acknowledge the prevailing pattern. Everything dies! It seems unescapable. Consequently, it is surprising to learn that many forms of life do have the capacity for indefinite survival and reproduction.

Provided with nutrients and protected from hostile environments, microorganisms do not die. A microbial cell splits into two, then into four, and so on. “Old age” ends when the parent cell divides into two rejuvenated daughters. Few individuals in the population senesce or die^{3, 4}; most cells continue the cycle of growth and division indefinitely. When death does occur, it is the result of an external cause: starvation, virus infection, toxic substances, or an inhospitable environment. Microorganisms are functionally immortal.

The crucial question, of course, is whether more complex organisms can also escape death. In the world we know, the mass of cells that makes up the body of an organism (often called the *soma*) experiences a gradual functional decline with time. The soma eventually perishes when an essential function of the corporate body is compromised. Before that happens, in many cases, a few cells from the organism do remain after the death of the soma. The survivors are from the *germline*, tissues that are distinct from the *soma*.

In sexual reproduction germline tissues produce specialized reproductive cells that carry only one copy of the genetic material. (Cells of the soma ordinarily contain two copies of the genes.) Two such reproductive cells, usually from different individuals, fuse to create a new organism. Though sexual reproduction is not required to perpetuate the lives of multicellular organisms, there is evidence that it may reduce the number deleterious mutations that may otherwise accumulate during asexually propagation.⁵ Nevertheless, a few vertebrates, many invertebrates and virtually all plants can reproduce asexually for long periods of time.

Germline tissues give rise to cells that create the next generation. They survive as offspring. Those offspring will, in turn, produce germline cells that will survive in the next generation of progeny. Like microorganisms, germline cells are functionally immortal. Though they must fuse with other germline cells in each reproductive cycle, they persist from parent to offspring in an unbroken chain back to Creation. The life of every person on the planet traces its existence through a pedigree of germline cells to Adam and Eve. In that limited sense, human life has a characteristic of immortality. Poets and philosophers are not far from the mark when they identify our children as the piece of ourselves that lives on.

If we were to consider technological approaches, it may soon be possible to propagate an organism indefinitely using somatic cells. When a nucleus (the structure that contains the genetic material) from a somatic cell is introduced into a special environment (the cytoplasm of a hollowed-out egg cell) the somatic nucleus can regenerate a whole organism genetically identical to the nuclear donor. In principle, this process might be repeated indefinitely to produce an unending series of identical organisms using only somatic cells.

But there is an obvious objection. Sexual reproduction and reproductive cloning (as described above) are not personal immortality. In human beings, individuality, personality and memory are functions of the *soma*. When the corporate body perishes, the “self” (self-awareness and identity) is gone. Children and grandchildren, as much joy as they may bring, do not qualify as eternal life.

On the other hand, the replacement of individual cells in the body does not interfere with the persistence of self. This recalls the ancient legend of Theseus. After slaying the Minotaur in Crete, Theseus returned triumphantly to Athens. His ship was preserved to memorialize his epic quest. Over time, however, as individual planks of the ship decayed, they were replaced, one by one, until all of the original wood was gone. Nevertheless, the Greeks honored the relic as Theseus' boat. In the same way, in the space of a decade, most cells in our bodies will be replaced with new ones. Individuality presumably lies in the relation between the parts, not necessarily on their individual persistence. The pattern encoded in those cellular relationships is still "you." Philosophically, morally, legally, and in every other sense, the continuously remodeled body is the same person, decade after decade.

By extension, since bacteria and germline cells divide and perpetually renew themselves, it is conceivable that somatic cells, if they had the appropriate genetic instructions, could similarly survive or generate replacement cells, indefinitely. Consequently, there are no fundamental philosophical barriers to prevent us from considering the indefinite extension of somatic life. The realization of that goal is a separate matter.

Catalog of Molecular Horrors

Serious studies of ageing and its complement, life extension, are relatively recent. Before 1985 these subjects were avoided as barren and frivolous. The scientific taboo may have been derived, in part, from the underlying assumption that ageing is the natural expression of an intrinsic genetic program. In this conception, organisms are designed to die soon after child-rearing—the equivalent of a biological expiration date. This belief was compatible with the evolutionary perspective that long life jeopardized the next generation by placing parents in competition with their offspring.

Today, the scientific perspective is distinctly different, at least for species with protracted child-rearing or a communal social structure. Evolutionists now believe that long life permits more efficient transfer of acquired adaptive behaviors to other members of the family group. Field observations of African elephants indicate that older individuals, beyond reproductive age, contribute significantly to the survival of their social groups.⁶ The current paradigm is much more compatible with studies of ageing and life extension.

No responsible scientist would claim that human immortality is a goal that will be achieved in the foreseeable future, but there is optimism regarding significant improvements in longevity. To understand that hope, we must first review current ideas about the processes that are believed to cause ageing and death. Let's briefly examine four theories of ageing that have attracted attention in recent years.

Playing with Fire

Most organisms with which we are familiar have an absolute requirement for oxygen; it is the final acceptor of electrons stripped from food molecules. Passing those electrons through a chain of carriers to oxygen releases usable energy. In plant and animal cells the mitochondria are the locations where electron transport drives the synthesis of ATP, the cellular energy currency. But oxygen has its dark side; its avid attraction for electrons endows it with properties that can be destructive. This makes intuitive sense when one appreciates the fact that transferring electrons from donor substances to oxygen is related to combustion, like burning a log in a fireplace. It entails serious risks. The procession of electrons down a carrier chain is not perfect. From 0.5 to 5% of them escape the bucket-brigade prematurely and form reactive oxygen species (ROS). These include the superoxide radical, hydroxyl radical and hydrogen peroxide--toxic substances that react destructively with many cellular components. Since most ROS are generated in the mitochondria, that organelle itself suffers the greatest direct damage. The result is a feedback loop of escaped electrons, ROS generation, mitochondrial damage, more errant electrons, more ROS, and still more damage.⁷

ROS attack all the vital components of cells. We can deduce that oxidative damage is a significant threat to viability from the considerable effort that cells devote to preventing it. An enzyme called superoxide dismutase, for example, is responsible for inactivating superoxide radicals in mitochondria. Defects in superoxide dismutase are correlated, in humans, with progressive, fatal neurological diseases. When the gene for this enzyme is experimentally inactivated in fruit flies, their mean lifetime is reduced by 90%.⁸ On the other hand, when an additional copy of the human superoxide dismutase gene is artificially added to the fly genome⁹, their lifetimes are extended by 40%.¹⁰ Similar effects are observed when catalase, an enzyme that breaks down hydrogen peroxide, is over-expressed.¹¹

Improved linkage between the components of the electron transport system and a full complement of potent antioxidants to neutralize ROS could minimize cellular damage and extend life. We suspect that this is possible because there are wide differences in the “leakiness” of mitochondria in different species. Organisms that have more efficient electron transport (e.g., birds) enjoy longer lives than would be expected from their size and basal metabolism.¹² Parrots, for example, live as long as elephants. Further, electron leaks can be reduced by other means. Mitochondrial efficiency is improved in rats by supplementing their diets with agents that appear to keep the electrons in line.^{13, 14,}
¹⁵ An impressive checklist of life functions (stamina, physical strength, memory, exploratory activity, etc) is simultaneously enhanced.

This Old House

Reactive molecules that leak from mitochondria are significant causes of cellular damage, but biological systems have many other enemies. In addition to being susceptible to attack by ROS, most critical components of living cells are intrinsically unstable. Like the “Deacon’s Amazing One-Hoss Shay,”¹⁶ they fall apart spontaneously without a specific external cause. Further, cellular constituents are continuously exposed to destructive agents from both inside and outside the cell. The repair hypothesis of ageing focuses on the gradual failure of cellular enzymes that would otherwise repair, remediate or remove defective components. It proposes that a robust set of enzymes is needed to repair this damage and promote longevity. For example, the degradation products of the amino acids asparagine and aspartate accumulate in the brains of older people and are correlated with dementia. A single enzyme in humans and mice is responsible for repairing these degraded amino acids. When that enzyme is knocked out in mice, their life span is reduced to 1/20th that of normal. They die of seizures due to cerebral malfunction.¹⁷

Cells have “chaperon” proteins to refold denatured proteins and mechanisms to remove them efficiently before they can affect the cellular economy. The “heat-shock” protein hsp70 is a ubiquitous chaperone in animal cells that restores other proteins to their native structure. Adding extra copies of the hsp70 gene to the genome of *Drosophila* significantly extends their lives. Deleting the normal hsp70 gene makes flies susceptible to neurodegenerative diseases.^{18, 19} Similarly, there are enzymes that repair or remove lipid peroxides to protect cellular membranes. Arrays of DNA and RNA repair

enzymes dedicated to correcting lesions in nucleic acids are fundamental elements of defensive repair systems. Scores of such repair systems have been described in higher organisms.

The value of highly responsive and redundant repair mechanisms is graphically illustrated by a remarkable microorganism—sometimes called “the superbug.” Cells of *Deinococcus radiodurans* were first isolated from the cooling water of nuclear power plants, an extremely inhospitable setting where one would not expect to find anything alive. Not only does it survive there, it grows! We now know that *D. radiodurans* lives naturally in highly desiccated habitats. These cells survive 5,000 grays of ionizing radiation without significant mutation. For perspective, that is 1,000 times the lethal dose for humans. As little as 1-2 grays causes acute human radiation sickness.^{20, 21} *D. radiodurans* is nearly immune to the effects of ultraviolet light. Its natural resistance to UV damage corresponds to a sun screen with a protection factor of about 2000. It grows happily in mitomycin C (a DNA cross-linking agent) at many times the concentration that dispatches mammalian cells. Under the direction of a unique regulatory protein, *D. radiodurans* can take a train wreck of chemically or radiation damaged DNA and stitch it back together into a damage-free genome.²² Remarkably, this microorganism contains no unique repair systems; it uses the very same enzyme types found widely in nature. Rather, it is their rapid appearance and high concentrations that account for *D. radiodurans*’ incredible durability.

It does not seem far-fetched to propose that all life forms were so equipped in the Garden. After the Fall, strong selection for reproductive efficiency (number of offspring in a lifetime), rather than long life (number of years in a lifetime), could readily account for loss of repair systems that have their greatest effects late in life and which, consequently, add little to reproductive success.

End Game

In multicellular organisms, the ends segments of the chromosomes, called the telomeres, often play a crucial role in determining the ability of cells to divide and replace themselves. Due to a fundamental chemical limitation of all DNA synthetic enzymes, the telomeres of chromosomes become a little shorter every time the cell replicates its DNA. Telomerase, an enzyme that can restore eroded telomeres, is not active in most human cells. As a result, the ends get progressively shorter with age,

like burning a candle at both ends. When the telomeres become critically short or lose their youthful structure, cells stop dividing and enter a state called “senescence.”²³

We can directly measure telomere shortening as individuals age.²⁴ Telomere loss accounts for the observation that normal human body cells will grow in laboratory culture for only a limited time (usually 50 to 80 doublings), then stop and become senescent. When examined, the telomeres of these cells are critically short. Cells from older people divide fewer times in culture than those from younger individuals. As we age, fewer body cells have the capacity to divide and replace others that die from injury or infection.²⁵ You may have wondered why a wound takes longer to heal now than it did when you were eight years old. Deficits in cell replacement have the greatest impact on tissues with short functional lives, like the epithelial cells lining the digestive tract or those forming the skin, and blood cells that are constantly squeezed through narrow capillaries. Failure to replace lost cells makes the body less resilient and more vulnerable to stresses and insults that would have little effect on a younger organism.

Only cells that continuously restore their telomeres to full length are able to divide indefinitely. These are found naturally in germline tissues and in a few adult stem cell lineages where the enzyme telomerase is active. Artificially adding genes for active telomerase allows normal human cells to continue dividing in culture. Rather than stopping after 80 divisions, such cells have been observed to exceed 300 doublings.²⁶ One wonders what would happen if all our cells had active telomerase.

Noisy Genes

Some conditions that extend the lives of laboratory animals seem unrelated to preventing oxidative damage, repairing cellular components or maintaining telomeres. Rather, they seem to be involved with signaling pathways that affect DNA accessibility. The first evidence for this system came from experiments in which laboratory animals were provided with diets that contained only 60% of the calories that they would have normally consumed. Every organism that has been examined, from yeast to primates, lives much longer under calorie restriction.²⁷ Not only do mammals on calorie restriction live 30 to 50% longer, but they are more active, more resistant to infection, and less susceptible to diseases like cancer, atherosclerosis and diabetes.²⁸

Calorie restriction activates a key control gene, called *sir-2* in yeast.^{29, 30} Similar genes are found in all organisms and their protein products are creatively called *sirtuins*. The Fountain of Youth effect of sirtuins has generated enormous interest and stimulated a lively field of research.

How calorie restriction accomplishes its effects is somewhat mysterious. The problem has become more tractable with the development of DNA arrays that allow the activity of many genes to be measured simultaneously. Sirtuins are deacetylases, enzymes that remove acetyl groups from the proteins that arrange and order DNA in the nucleus. Removal of the acetyl groups makes chromosome structure more compact and affects the activity of genes. The mild stress of calorie restriction stimulates production of sirtuins which, in turn, silence or activate broad sets of additional genes.^{31, 32} The net effect is only superficially understood. Genes related to damage prevention, repair, restoration and many other cellular survival functions tend to be activated. Many other genes are shut off. The observable result is that the animals are healthier, more vigorous, and longer-lived.

As one might expect, examining the effects of calorie restriction in humans requires the adoption of an severely ascetic lifestyle, but a group of volunteers has taken up the challenge. They have reduced their caloric intake 30 to 50% below that recommended for their age and sex for periods ranging from 3 to 15 years. In every case they show marked improvements in health indicators: lower LDLs, higher HDLs, lower blood pressure, lower insulin levels, higher insulin sensitivity, and lower C-reactive protein.³³ If dietary restriction dramatically prolongs life, it may be some time before we get confirming data from this group.

Since calorie restriction is not a life style that many people are likely to adopt, investigators have asked if there are other interventions that might evoke the *sir-2* effect without the rigors of near starvation. This might be considered the search for a pill that would allow you to “eat your cake *without* having it.” The search turned up a family of plant polyphenols that are widely distributed in foods. The best studied and most potent is resveratrol, a compound abundant in grapes and peanuts.³⁴ When resveratrol is added to the diets of test subjects from yeast to primates, it produces the same physiological benefits as dietary restriction—greater vigor, disease resistance and dramatically longer lives. This line of study gives new meaning to the idea of a fruit promoting immortality.

(Unfortunately, resveratrol and related compounds are quite unstable in air, so most commercial preparations are inactive.)

Ageing is a complex process in which all these mechanisms, and others, are likely to play a role.³⁵ We know that cells naturally employ multiple strategies to counter ageing. In spite of these efforts, accelerating rates of structural damage and dysregulation gradually overtake the preventive and restorative programs of cells. The final outcome is death.

We can discard the old evolutionary dogma that living things activate an intrinsic “death program” whose purpose is planned obsolescence, assuring that parents die soon after raising their offspring. There are also good reasons to doubt the more recent evolutionary dictum that mechanisms for prolonging life beyond the needs of reproduction are unlikely to exist because selection is “uninterested” in such matters. Selection, by definition, is concerned with leaving progeny who can, in turn, produce more progeny for the next generation. Traits that reduce the number of offspring because they divert resources away from reproduction are eliminated. Those with no effect whatever on reproduction are “neutral”; they are ignored. That means that their maintenance is not assured, but entirely a matter of chance (often called “genetic drift”). The survival mechanisms we have examined almost certainly siphon metabolic resources away from reproduction. An evolutionary perspective predicts that they should be uncommon. Yet, we find them everywhere, even in organisms that do not rear their young and have no stable social organization. Why?

Simulation

Let’s put some of the pieces together. Consider a planetary ecology with conditions that may resemble those on the newly created Earth. Since the biblical descriptions are sparse, I make no attempt here to justify or support all of them individually. Rather, let’s consider this a simulation and see how closely this construct might replicate the qualitative descriptions of Eden (amplified by the restoration texts) found in the Bible.

1. Planet Earth had a stable climate with minimal seasonal variation and mild weather at all latitudes. This might be achieved by arranging the planetary rotational axis orthogonal to the ecliptic (i.e., perpendicular to the plane of its path around the Sun). We might also include atmospheric patterns and bodies of water that would further moderate temperature extremes.

(Other geophysical features like the presence or absence of mountain ranges, global oceans, tectonic activity or rain would contribute to the simulation, but they cannot be discussed adequately here.)

2. Organisms ate food (reduced organic or inorganic substances) to sustain themselves, as they do now. Living things excreted incompletely oxidized organic molecules as waste products. Though the process may have been more efficient than we now find it one can hardly dismiss the fact that waste production is universal and bears all the marks of being part of the original design. Some organisms could, as plants do now, completely oxidize fatty acids or simple sugars to CO₂ and H₂O without any waste.
3. All observable animal life consumed the deciduous or replaceable portions of plants – fruit, seeds and vegetative elements that were replaced readily. Fruit-eating and browsing did not cause the death of individual plants.

“And God said, Behold, I have given you every herb bearing seed, which is upon the face of all the earth, and every tree, in the which is the fruit of a tree yielding seed; to you it shall be for meat. And to every beast of the earth, and to every fowl of the air, and to every thing that creepeth upon the earth, wherein there is life, I have given every green herb for meat: and it was so.” (Genesis 1:29, 30)

Food chains were short with green plants (or their photosynthetic microbial equivalents) at the base.

4. There was no predation. Only plant sources and their microbial equivalents served as food. There was no parasitism or infection, though symbiotic and mutualistic associations may have existed. Disease, a condition in which one organism appropriates resources from another with little or no contribution to the relationship, did not occur.

There were no disease-causing organisms or parasites to cause infection. Consider the fact that there are an estimated 10 million species of animals and plants on the planet (of which about 1.7

million have been described). Compare that with estimates of microbial species (single-celled organisms without nuclei) that range upwards from one hundred million.^{36, 37} Of this enormous number, even today, no more than a few hundred species cause disease.

The concept of a planet without microbial pathogens is more plausible now than it was 10 years ago, before we had the complete DNA sequences of microbial genomes. Since 1995 the DNA of several hundred microorganisms have been decoded. (A more precise estimate is pointless since the sequences of new microbes can be effectively completed in half a day.) Remarkable generalizations can be drawn from the avalanche of new genetic sequences.

The genomes of human pathogens are incredibly similar to their nearest non-pathogenic relatives.^{39, 40, 41} Pathogens typically have a few dozen virulence genes among the several thousand that comprise their genomes. These distinctive virulence genes are essential for infection; their products allow microbes to attach to host cells, gain access to protected environments, produce toxins and evade host defenses. The genetic differences between pathogenic and related non-pathogenic species are limited to these small sets of virulence genes.

A second surprise is that virulence genes, in spite of their diverse physiological properties, are not distributed throughout the genomes of pathogens, but clustered tightly in compact groups often called “pathogenicity islands.”⁴² For example, a single gene cluster in *Mycobacterium tuberculosis* contains 13 open reading frames (i.e., potential genes). Mutations in seven of them reduce virulence. The entire cluster seems to be involved in the biosynthesis of envelope components found primarily in pathogenic mycobacterial species.^{43, 44}

In the evolutionary scenario, cells without nuclei (bacteria and archaea) came into existence nearly four billion years ago, followed by the first unicellular eukaryotes (cells with nuclei), a half billion years later. Multicellular eukaryotes are considered latecomers, appearing about one billion years ago. In this scenario, microorganisms were present during the entire evolutionary history of multicellular organisms. One billion years seems sufficient to expect that some bacteria would become “professional” parasites with virulence mechanisms intimately integrated

into their genomes. But that is not what we observe.⁴⁵ At the DNA level, virulence looks like a recent innovation.

Additional evidence suggests that pathogens are newly converted non-pathogens. Not only are disease-related genes in compact clusters, but the features of virulence genes and pathogenicity islands suggest that they have been imported from some other source. Their nucleotide sequences (GC/AT ratios, codon usage, dinucleotide frequencies, GC skew)⁴⁶ are distinct from the bulk of the bacterial genome in which they reside. In every other context such features are considered evidence of a recent DNA acquisition. The boundaries of pathogenicity islands are also distinctive. They often indicate the mechanism by which the segments were acquired and sometimes suggest their source. It is remarkably rare to discover virulence-related genes that appear to be native to their host genomes. The take-home lesson is that pathogenicity looks like a recent modification. It will be interesting to see if this pattern extends to the genomes of non-microbial parasites.

5. Macroscopic life forms did not die. Living things that were readily visible to the eye did not experience organismic death.

In this discussion, organismic death is defined as the death of a whole animal or plant. The loss of deciduous or replaceable components of an organism (for example, individual body cells; superficial elements like skin or hair or fur; leaves, fruit or seeds from plants) does not end the existence of the organism and, therefore, does not constitute organismic death.

In our world, predation, infection and death are pervasive sources of pain, fear and conflict among both human and nonhuman species. In contrast, Isaiah's vision of the New Earth reflects the harmony of a world without predation or death.

“The wolf also shall dwell with the lamb, and the leopard shall lie down with the kid; and the calf and the young lion and the fatling together; and a little child shall lead them. And the cow and the bear shall feed; their young ones shall lie down together: and the lion shall eat straw like the ox. And the sucking child shall play on the hole of

the asp, and the weaned child shall put his hand on the cockatrice' den. They shall not hurt nor destroy in all my holy mountain: for the earth shall be full of the knowledge of the LORD, as the waters cover the sea.” (Isaiah 11:6-9, see also Isaiah 65:25)

6. The divide between life forms that experienced death and those that were functionally immortal is debatable. I am indebted to presentations and publications by Leonard Brand⁴⁷ and Barry Taylor⁴⁸ on this subject. We agree that individual cells were never immortal and that programmed cell death is an essential feature of embryological development and adult life. Without it we would be incapable of replacing epithelial cells lost by the passage of food through our digestive tracts nor would we grow hair. However, our views diverge somewhat regarding what animals and plants experienced death.

We get some insight into which creatures were considered to possess life from the language of the scriptures:

“All in whose nostrils was the breath of life . . .” (Genesis 7:22) and *“For it [blood] is the life of all flesh; the blood of it is for the life thereof . . . for the life of all flesh is the blood . . .”* (Leviticus 17:14)

Bible writers considered even the smallest creature that breathed or bled “alive.” When Jesus represented the extent of God’s concern for humanity he referred to common birds (Matthew 10:29, 31; Luke 12:6, 7). If the death of a sparrow did not pluck a string of compassion in the hearts of his listeners, those illustrations would have been meaningless. It would have made no sense at all, for instance, if Jesus had said that God was concerned about the pebbles that cracked under their sandals.

The status of invertebrates is less clear. Larger invertebrates (hemichordates, cephalopods, echinoderms, tunicates, arthropods, helminths, annelids, etc.) respond vigorously to environmental stimuli. It is difficult to know if they perceive pain, but their physical reactions mimic the pain responses of higher organisms.

Consult your own sense of compassion. Recall the starfish. I have difficulty imagining that Adam and Eve were less affected by the demise of small creatures than we are. Would the Garden be a paradise if the butterfly visiting your garden today lay in tattered ruins tomorrow? What did Isaiah mean when he wrote *“They shall not hurt nor destroy in all my holy mountain . . .”* (Isaiah 11:9)

We are pained to some degree when we experience the death of any living creature. Where does that feeling come from? Neuropsychologists tell us that our minds recreate the feeling and emotions we perceive in others. When test subjects are shown pictures of people expressing strong emotions, functional MRI shows activation of the same cerebral regions in the viewers’ brains as are activated in the people experiencing the primary emotion.^{49, 50, 51} When we see a person in distress, we don’t imagine what they feel; we experience it ourselves. These “mirror” responses are even stimulated by emotions we impute to human surrogates, like our pets.⁵² Few people are unaffected by the yelps of a dog that has tangled with a car. Further, these responses appear to be “hardwired.” They occur in infants. When my daughter was very young, if I rolled out my lower lip, screwed up my mouth, and lowered my eyebrows, she started to cry. She appeared feel the emotion I was portraying. “I feel your pain” is more than a bumper sticker.

7. There was no fear, terror or dread. For sentient humans, the anticipation of death, disease or attack is the primary source of dread and fear. Woody Allen’s quip, “I’m not afraid of death. It’s just that I don’t want to be there when it happens” expresses an emotion that is common in the Bible:

“My heart is sore pained within me: and the terrors of death are fallen upon me. Fearfulness and trembling are come upon me, and horror hath overwhelmed me.” (Psalms 55:4, 5) *“The sorrows of death compassed me, and the pains of hell gat hold upon me: I found trouble and sorrow.”* (Psalms 116:3) *“And thy life shall hang in doubt before thee; and thou shalt fear day and night, and thou shalt have none assurance of thy life: In the morning thou shalt say, Would God it were even! and at even thou shalt say, Would God it were morning! for the fear of thine heart wherewith*

thou shalt fear, and for the sight of thine eyes which thou shalt see.” (Deuteronomy 28:66-67).

The absence of severe pain does not imply that the first humans were incapable of that sensation, but that few circumstances generated that sensation. Essential sensory mechanisms designed to forestall serious injury were intact. There were occasions for discomfort and they would have moderated the range of action. Adam would have felt pain when he stubbed his toe. He would have felt discomfort when separated from his partner. However, there was nothing in his world that would have stimulated fear, dread, panic, anguish, distress, grief, sorrow or anxiety. People and animals could pursue their lives and interests with an exuberant sense of self-determination and independence. They were fearless, joyful, playful and inquisitive. It was an ecology of freedom.

“And God shall wipe away all tears from their eyes; and there shall be no more death, neither sorrow, nor crying, neither shall there be any more pain: for the former things are passed away.” (Revelation 21:4)

Some contend that physical and psychological pain are potent motivators, that human creativity is piqued by discomfort. A well-known proverb might well be paraphrased “pain is the mother of invention.” The absence of pain could stifle investigation and curiosity. (This recalls the *Eloi* in H.G. Wells’ “The Time Machine.” They were a race that lacked curiosity, science, philosophy and intellect because they received, without effort or discomfort, everything they needed.) On the other hand, many early scientists, philosophers and creative geniuses were independently wealthy “gentlemen scholars,” buffered, to a degree, from the hardships and stresses of the common folk. Yet they were paragons of inventiveness and creativity. Unparalleled advances in science during the last two centuries were often achieved by men and women who were physically, socially and economically “comfortable.”

8. Childbirth was a benign experience. For Bible writers, however, birth was a symbol for pain, both physical and psychological.

“Like as a woman with child, that draweth near the time of her delivery, is in pain, and crieth out in her pangs; so have we been in thy sight, O LORD. We have been with child, we have been in pain . . .” (Isaiah 26:17, 18; see also Isaiah 13:8; 21:3, Psalms 48:6, Micah 4:10, 11, Revelation 12:2).

The clear implication from the curses pronounced at the Fall is that birth was not associated with pain before sin.

The physical basis for pain during childbirth is the passage of a human fetus with a large skull and broad shoulders through a narrow birth canal powered by strong contractions of uterine muscles. Several means for avoiding the distress of birth are conceivable. One is that adult humans were considerably larger and their size allowed a less traumatic delivery. Another intriguing idea is auto-anesthesia. Voluntary control of endogenous opioids and related pain modulators could allow birth to occur much as it does today, but without the sensation of extreme pain. Some techniques of natural childbirth approach this goal.

9. Microscopic forms of life, though functionally immortal, experienced death. The Bible gives no indication how far down the tree of life immortality may have extended, but our own sensitivities provide a clue. You may have seen the television commercial that was broadcast during the cold season recently. A man with a shaved head, dressed in a saffron robe came upon a small turtle flipped on its back. He gently righted it. He cupped his hand under a little fish flopping in the grass and returned it to the pond. It’s only a commercial, but we resonate with the holy man’s responses to the distress of small animals, his respect for life. But how “small” does that empathy go? As the commercial continued, the monk anticipated a sneeze and covered his mouth with a tissue. His eyes widened in horror as he read the statement on the tissue box – “Kills 99.9% of all bacteria and viruses.” He was responsible for the demise of an uncountable number of invisible organisms! We smile. Why? Surely, the theme has been extended to the point of absurdity. People care about the death of animals, even little ones. No one feels a twinge at the death of a billion microbes.

At the lower end of the biological scale, microscopic life--bacteria, phytoplankton, zooplankton, both unicellular and multicellular--are organisms that do not meet any biblical definition of "life." Furthermore, they are invisible. Their death is unobserved. They are incapable of pain. They perish incidentally or are consumed by macroscopic animals. The phytoplankton (photosynthetic forms in the photic layer of the oceans) constitute the marine equivalent of "the grass of the fields," intended for food.

10. The reproduction of macroscopic life forms was tightly regulated by environmental contexts. Life in Eden was exquisitely sensitive to population density and environmental signals indicating depletion of the resources needed for optimal well-being. The earliest signs of crowding, range degradation or nutritional limitation would have triggered intrinsic neuroendocrine pathways to suppress reproduction. By this means, animal populations would plateau at the point of optimal and sustainable environmental utilization.

Even in the gene-centric environment of modern biology, natural scientists know of many non-genetic means for regulating the physical and behavioral traits of animals. These "epigenetic" mechanisms would work well to control reproduction. They require that the organism detect subtle environmental signals and respond with specific physiological changes that result in suppression of reproduction.

A few examples may be helpful. When juvenile *Daphnia* ("water fleas" or "sea monkeys") develop in pond water inhabited by predator fish, they alter their morphology to grow a protective spike that makes them less susceptible to predation. We know that something secreted by the fish induces this change because water in which fish were grown triggers the same response as the fish themselves.⁵³ In this case, the signal is a chemical produced by the *Daphnia*, but is activated by something from the predator. The prey responds by becoming less susceptible to predation. In the unfallen world a similar signal-response pair could have been a subtle change in the environment and a suppression of reproduction.

We know that feedback mechanisms like these still affect reproduction. Sex determination in some turtles, lizards and fish is context dependent; the relative numbers of existing males and

females can trigger sex reversal in the young. In some cases environmental conditions, not chromosomes, determine the sex of organisms. The resulting sex change modulates the rate of subsequent reproduction. In one case, conservation biologists working to save an endangered turtle species collected some live animals for a captive breeding program. Unfortunately, the temperature of the breeding facility produced only male offspring. Since the sex of the hatchlings was difficult to determine, the conservationists unknowingly released thousands of tiny turtles that had almost no chance of finding representatives of the opposite sex.

In insects, nutritional factors often produce different physical types in individuals that have exactly the same genes. Queen and worker ants start out with the same genomes; it's the food they receive during early growth that makes the difference in their adult forms. Such responses are *epigenetic*—the genomes of the castes are not different. Rather, chemical modifications of protein molecules associated with the DNA change the expression of underlying genes.^{54, 55}

A further example is the nematode *Caenorhabditis elegans*, a soil-dwelling worm about 1 mm long with a lifespan of about three weeks. When exposed to overcrowding, a condition it detects by the accumulation of a chemical pheromone it secretes, *C. elegans* diverts its normal development into a long-living, non-reproductive form called the *dauer* larvae.⁵⁶ Projecting such epigenetic responses to the reproductive processes of terrestrial mammals and humans is not far-fetched. Indeed, the adaptive branch of the mammalian immune system generates context-specific physiological responses that could readily serve as models for reproductive control.

11. Plant life had a higher reproductive “set-point.” Plant growth was also responsive to environmental cues, but at optimal densities they continued to generate sufficient fruit to supply the local animal population and to replace vegetative elements consumed by animals.
12. No creature in Eden was intrinsically immortal. Plants and animals did not die of old age, predation or infection, but accidental death was a possibility. A benign environment, however, would have made accidental death unlikely. Jagged mountains, precipitous cliffs, swamps or tar pits probably did not exist. The climate was mild and stable, without destructive storms or droughts. Flash floods, white water, lightning and wildfires may not have existed.

God continuously sustains the operations of the physical world that are necessary for life. All creatures borrow the elusive and mysterious property of life from God. Whether consuming fruit from the Tree provided an otherwise unavailable nutrient, was a means by which God evaluated human willingness to recognize his sovereignty, or worked by some other mechanism, is immaterial. Creaturely life was always derivative and conditional.

Earlier discussions about the extent to which living things may avoid death in the New Earth have been helpful. My focus here, however, is slightly different. I prefer to consider the original Garden for several reasons. First, it is not always clear which restoration texts apply to the New Earth. (Are the young lions of Psalms 104:21 looking for prey in the New Earth or the fallen one?) It is sometimes difficult to judge the literalness of the images. (Will the redeemed regularly contemplate the rotting corpses of the wicked dead as Isaiah 66:24 suggests?) And there are indications that the New Earth may not be a simple replica of Eden (Revelation 21 seems much more urban than Eden). Finally there is the phrase “. . . *neither have entered into the heart of man.* . . . ” (I Corinthians 2:9) to discourage speculation about the future.

On the other hand, we are encouraged to examine the past and learn what it can teach us. It existed. Archeologists (biblical and otherwise), paleontologists and geologists spend countless man-years deciphering what the earth and its inhabitants were like in the past. For these reasons this essay has focused on the original Earth, rather than the restored Earth.

Could the Earth Survive if Animals Lived Forever?

How did the ecology of Eden work? If some organisms never died, one must consider their impact on the environment. Immortal organisms would consume plant materials and generate waste products. To close the circle, those waste products must be processed to release nutrients needed by plants to regenerate the elements that were consumed.

Our human perspective misleads us when we think about the biological world. We often overrate the significance of big things and underestimate the contributions of the small. In actual fact, large animals and plants do not run the world. The basic maintenance of the planet is accomplished by organisms we

can't see. Earth is a microbial world. Large animals and plants are only slightly more than decorations. Microorganisms have a dominant role in both the primary production and consumption of organic matter. More than half of all the biological carbon on the planet is in prokaryotic cells (i.e., bacteria and archaea). In aggregate, microbes weigh more than all other living things combined.

On the synthetic side of the balance, half of all carbon fixation (conversion of CO₂ into organic compounds) is accomplished by marine photosynthetic bacteria and microscopic algae.^{57, 58} The remainder is catalyzed by green plants. Microbes are also the principal players in the cycling of other nutrient elements. About 3/4 of all the nitrogen and phosphorus in living things is in microorganisms. Essentially all nitrogen fixation (conversion of atmospheric N₂ into nitrites and nitrates) is accomplished by microorganisms that are sometimes, but not always, associated with green plants. The plants in our gardens, fields and forests could not exist without the nitrogen fixed by bacteria.⁵⁹

Whatever the extent of pre-Fall oceans, a sustainable ecosystem would require that the original aquatic environments make a similar contribution to the global economy. Pelagic photoplankton (cyanobacteria that fix nitrogen and carbon, photosynthetic diatoms, golden algae and green algae) were the foundation of the pre-fall marine food chain. They were food for zooplankton (protozoa and microscopic crustaceans). Shallow waters would have also supported photosynthetic marine plants and green algae. Water animals consumed aquatic and marine plants, photoplankton and zooplankton. There was no predation above the level of microscopic organisms.

On the degradation side of the equation, microorganisms again do the heavy lifting. Animals that consume fruit, seeds and tubers—plant parts that contain significant quantities of readily digestible carbohydrates, fats, and proteins—use their own digestive enzymes to reduce those nutrients to sugars, fatty acids and amino acids. Those building blocks are reassembled into new animal tissue, while some is oxidized to generate the energy needed to power the synthetic effort.

But animals cannot digest one of the most common polysaccharides on the planet—cellulose. For fruit-seed-and-tuber eaters, like humans, cellulose is indigestible “fiber“ (baby boomers may recognize it as “roughage”) that is eliminated as waste. Grazers and ruminants, on the other hand, consume plant parts that are quite low in proteins, lipids and simple carbohydrates. The bulk of their diet is cellulose.

Surprisingly, even grazers lack the enzymes needed to digest cellulose to its simple-sugar building blocks. They would starve were it not for the commensal microorganisms in their digestive tracts. Microbes digest cellulose in the rumens and crops of grazers as an aid to their hosts. Free living microbes digest cellulose in the droppings of non-ruminants, returning simpler compounds to the soil. Termites and other insects living on “high-fiber” diets, do so by virtue of resident microbes. The “carbon cycle” is powered by microorganisms. It would spin just as smoothly without the macro-organisms that typically occupy our attention, or with plants and animals that lived forever.

All the elemental biological cycles of the planet would operate in a situation where macroscopic organisms lived indefinitely. Green plants, symbiotic and free-living nitrogen fixing-bacteria, and photosynthetic microbes would be at the base of planetary nutritional pyramids. Animals would use them for food. Living indefinitely, their bodies would be part of a “living carbon” reservoir. Animal wastes and deciduous plant parts would be degraded back to simpler compounds by microorganisms. Intrinsic reproductive controls in animals and plants would limit the size of the living carbon reservoir.

Filling the Earth

The Genesis account suggests that there were relatively few representatives of large terrestrial species at the end of Creation week and that they were localized near the Garden (see Genesis 1:26). In contrast, the text indicates that plants covered the earth and aquatic environments supported large populations of plants and aquatic animals. A large portion of the land area may have been devoid of animal residents. The plan was for terrestrial animals and humankind to reproduce and disperse from the Garden into green, but otherwise unoccupied land area. The variety of new habitats and niches would have been ideal for rapid diversification. Built-in mechanisms for genetic change (programmed DNA rearrangements, mobile extra-chromosomal elements, virus-like agents, transposable elements, etc.) would have provided most or all of the genetic variation needed for this process. Accidental changes like mutations probably contributed little.

It is not my intent here to describe how life has changed since the Fall. However, mechanisms that were designed to facilitate rapid adaptation of organisms dispersing from their original locus were surely important. Viruses may have played an essential role. Only recently have we begun to appreciate the powerful influence they exercise over the biosphere. There are thought to be some 10^{31} virus particles on

the planet, an astounding figure that invites astronomical comparisons. (One investigator calculated that their aggregate genetic material, laid end-to-end, would span *250 million light years*. Another suggested that they contain more genetic diversity than all other organisms combined.)⁶⁰ Their staggering diversity, ubiquity and genetic agility suggest that these sub-cellular entities may have been a potent force in the original ecosystem.

Since the original populations of some land mammals may have been very small (like the human couple), there would have been an acute need to expand their gene pools. Some viruses are not cytopathogenic and have no debilitating effects on their hosts. Viruses of this type may have been designed to carry supplementary genetic alleles for specific organisms. By this means viruses may have contributed to the effective size of biological populations. The ability of viruses to carry genes that are important only to their hosts was supported recently by the striking observation that certain viruses carry individual elements of the photosynthetic pathway found in the marine bacteria they infect.^{61, 62} As another example, the giant mimivirus carries spare parts for its host's protein synthesis apparatus and its DNA repair system,⁶³ functions that do not operate in the viruses themselves.

Rapid diversification might be expected as organisms migrated to favorable settings and niches. There they would express their maximum reproductive potential until that niche was optimally populated. Animals that found themselves in occupied territory would have suppressed reproduction until they migrated to open zones. Diversity would likely have increased as organisms radiated from the Garden. Diversification would have been powered by programmed genetic changes rather than by mutation and selection.

During dispersal, bio-organic synthesis would not have been balanced by degradation. As the number of land animals (and possibly plants) increased with reproduction and migration from the Garden, there would have been a net increase in fixed organic carbon withdrawn from inorganic carbon reservoirs (atmospheric and dissolved CO₂ and carbonate minerals). When the earth reached its carrying capacity, animal reproduction was designed to gradually decrease. Plant reproduction and regrowth would continue only to the extent required to balance consumption by animals. Animal waste and deciduous plant structures were recycled by microbes. At the level of elemental cycles, the pre-fall world would have worked much like the one we are used to.

As an archeologist assembles ancient pottery shards, the form and purpose of the vessel are gradually revealed. At some point there may come the realization that the artefact was not a common utensil, but an elegant ceremonial urn of great beauty and significance. In the same manner, deeper insights are emerging as we sift through the pieces of life. The fragments in our hands may not have their original color or luster, but observations from many different sources suggest that immortality, as represented in the Scriptures, is a clear possibility. There is no unfathomable chasm between life as we know it and life in the Garden. The fundamental principle of life may be debated, but it seems to be designed to last. Basic considerations indicate that immortal organisms can be integrated into a sustainable biosphere without violating accepted principles of ecology. A belief in the reality of Eden is compatible with science. The story of the world before sin makes perfectly good sense.

*The fair earth, as it came from the Creator's hand, bore no blight of decay or shadow of the curse.”*⁶⁴

Endnotes

1. This topic was first presented at the Symposium III on the Bible and Adventist Scholarship, March 24, 2006, Cancun, Mexico.
2. Bible texts are from the King James Version.
3. Stevens, C. 2005. Even bacteria get old. *Curr Biol* 25:R308-R310.
4. Nystrom, T. 2003. Conditional senescence in bacteria: death of the immortals. *Mol Microbiol* 48:17-23.
5. Paland, S. and Lynch, M. 2006. Transitions to asexuality result in excess amino acid substitutions. *Science* 311:990-992.
6. McComb, K., Moss, C., Durant, S.M., Baker, L. and, Sayialel, S. 2001. Matriarchs as repositories of social knowledge in African elephants. *Science* 292:491-494.
7. Balaban, R, Nemoto, S. and, Finkel, T. 2005. Mitochondria, Oxidants, and Aging. *Cell* 120:483-495.
8. Parkes, T.L., Kirby, K., Phillips, J.P. and Hilliker, A.J. 1998b. Transgenic analysis of the cSOD-null phenotypic syndrome in *Drosophila*. *Genome* 41:642-651.
9. The word “genome” denotes all of the genetic material of an organism, including its chromosomes, accessory genetic elements and the DNA in its cellular organelles.
10. Parkes, T.L., Elia, A.J., Dickinson, D., Hilliker, A.J., Phillips, J.P. and Boulianne, G.L. 1998a. Extension of *Drosophila* lifespan by over expression of human SOD1 in motor neurons. *Nat Genet* 19:171-174
11. Schriener, S.E., Linford, N.J., Martin, G.M., Treuting, P., Ogburn, C.E., Edmond, M., Coskun, P.E., Ladiges, W., Wolf, N., Van Remmen, H. and Rabinovitch, S. 2005. Extension of murine life span by overexpression of catalase targeted to mitochondria. *Science* 308:1909-1911.

12. Harper, M.-E., Bevilacqua, L., Hagopian, K., Weindruch, R. and Ramsey, J.J. 2004. Ageing, oxidative stress, and mitochondrial uncoupling. *Acta Physiol Scand* 182:321-33.
13. Liu, J., Killilea, D. and Ames, B.N. 2002b. Age-associated mitochondrial oxidative decay: improvement of carnitine acetyltransferase substrate binding affinity and activity in brain by feeding old rats acetyl-L-carnitine and/or R- α -lipoic acid. *Proc Natl Acad Sci USA* 99:1876-1881.
14. Hagen, T.M., Liu, J., Lykkesfeldt, J., Wehr, C.M., Ingersoll, R.T., Vinarsky, V., Bartholomew, J.C. and Ames, B.N. 2002. Feeding acetyl-L-carnitine and lipoic acid to old rats significantly improves metabolic function while decreasing oxidative stress. *Proc Nat Acad Sci USA* 99:1870-1875.
15. Liu, J., Head, E., Gharib, A.M., Yuan, W., Ingersoll, R.T., Hagen, T.M., Cotman, C.W. and Ames, B.N. 2002a. Memory loss in old rats is associated with brain mitochondrial decay and RNA/DNA oxidation: partial reversal by feeding acetyl-L-carnitine and/or R--lipoic acid. *Proc Nat Acad Sci USA* 99:2356-2361
16. Oliver Wendell Holmes. *The Deacon's Masterpiece or the Wonderful One-Hoss Shay, A Logical Story*
17. Kim, E., Lowenson, J.D., MacLaren, D.C. and Clarke, S. 1997. Deficiency of a protein-repair enzyme results in accumulation of altered proteins, retardation of growth, and fatal seizures. *Proc Nat Acad Sci USA* 94:6132-6137.
18. Tatar, K. 1999. Transgenes in the analysis of life span and fitness. *Am Nat* 154:S67-S81.
19. Gong, W.J. and Golic, G. 2006. Loss of Hs70 in *Drosophila* is pleiotropic, with effects on thermotolerance, recovery from heat shock and neurodegeneration. *Genetics* 172:275-286.
20. ICRP Publication 60, *1990 Recommendation of the International Commission on Radiological Protection*, Pergamon Press, New York, 1991.
21. Pochin, E. 1983. *Nuclear Radiation: Risks and Benefits*, Clarendon Press, Oxford.
22. Ashlee, M., Earl, M., Mohundro, M., Mian, I.S. and Battista, R.J. 2002. The IrrE protein of *Deinococcus radiodurans* R1 is a novel regulator of recA expression. *J Bacteriol* 184:6216-6224.
23. Stewart, S.A., Ben-Porath, I., Carey, V.J., O'Connor, B.F., Hahn, W.C. and Weinberg, R.A. 2003. Erosion of the telomeric single-stranded overhang at replicative senescence. *Nature Genetics* 33:4892-496.
24. Hastie, N.D., Dempster, M., Dunlop, M.G., Thompson, A.M., Green, D.K. and Allshire, R.C. 1990. Telomere reduction in human colorectal carcinoma and with ageing. *Nature* 346:866-868.
25. Herbig, U., Ferreira, M., Condel, L., Carey, D. and Sedivy, J.M. 2006. Cellular senescence in aging primates. *Science* 311:1257
26. Bodnar, A. G., Oullette, M., Froklic, M., Holt, S. E, Chiu, C-P., Morin, G. B., Harley, C. B., Shay, J. W., Lichtsteiner, S., and Wright, W. E. 1998. Extension of life-span by introduction of telomerase into normal human cells. *Science* 279:449–352.
27. Masoro, E.J. 2000. Caloric restriction and aging: an update. *Exp Gerontol* 35:299-305.
28. Garsin, D.A., Villanueva, J.M., Begun, J., Kim, D.H., Sifri, C.D., Claderwood, S.B., Ruvkun, G. and Ausubel, F.M. 2003. Long-lived *C. elegans daf-2* mutants are resistant to bacterial pathogens. *Science* 300:1921.
29. Tissenbaum, H. A. and Guarente, L. 2001, Increased dosage of a sir-2 gene extends lifespan in *Caenorhabditis elegans*. *Nature* 410:227-230.
30. Lin S.-J., Defossez P.-A. and Guarente, L. 2000. Requirement of NAD and SIR2 for life-span extension by calorie restriction in *Saccharomyces cerevisiae*. *Science* 289:2126-2128.
31. Gems, D. and McElwee, J.J. 2003. Ageing: microarraying mortality. *Nature* 424:259 - 261.

32. Murphy, C.T., McCarroll, C.A., Bargmann, C.I., Fraser, A., Kamath, R.S., Ahringer, J., Li, H. and Kenyon, C. 2003. Genes that act downstream of DAF-16 to influence the lifespan of *Caenorhabditis elegans*. *Nature* 424:277-284.
33. Fontana, L., Meyer, T.E., Klein, S. and Holloszy, J.O. 2004 Long-term calorie restriction is highly effective in reducing the risk for atherosclerosis in humans. *Proc Nat Acad Sci USA* 101:6659-6663.
34. Howitz, K.T., Bitterman, K.J., Cohen, H.Y., Lamming, D.W., Lavu, S., Wood, J.G., Zipkin, R.E., Chung, P., Kisielewski, A., Zhang, L.-L., Scherer, B. and Sinclair, D.A. 2003. Small molecule activators of sirtuins extend *Saccharomyces cerevisiae* lifespan. *Nature* 425:191-196.
35. Kirkwood, T.B.L., 2003. Genes that shape the course of ageing, *Trends in Endocrinology and Metabolism* 14:345-347.
36. Ward, B.B. 2002. How many species of prokaryotes are there? *Proc Nat Acad Sci USA* 99:10234-10236.
37. Curtis, T.P., Sloan, W.T. and Scannell, J.W. 2002. Estimating prokaryotic diversity and its limits. *Proc Nat Acad Sci USA* 99:10494-10499.
38. Lawrence, J.G. 1999. Gene transfer, speciation, and the evolution of bacterial genomes. *Curr Opin Microbiol* 2:519-523.
39. Read, T.D. et al., 2003. The genome sequence of *Bacillus anthracis* Ames and comparison to closely related bacteria. *Nature* 423:81-86.
40. Ivanova, N. et al. 2003. Genome sequence of *Bacillus cereus* and comparative analysis with *Bacillus anthracis*. *Nature* 423:87-91.
41. Parkhill, J. and Berry, C. 2003 Genomics: relative pathogenic values. *Nature* 423:23-25.
42. Wilson, B.A. and Salyers, A.A. 2003. Is the evolution of bacterial pathogens an out-of-body experience? *Trends in Microbiology* 11:347-350.
43. Camacho, L.R., Ensergueix, D., Perez, E., Giequel, B. and Guilhot, C. 1999. Identification of a virulence gene cluster of *Mycobacterium tuberculosis* by signature-tagged transposon mutagenesis. *Mol. Microbiol.* 34:257-267.
44. Cox, J.S., Chen, B., McNeil, M. and Jacobs, W.R. Jr. 1999 Complex lipid determines tissue-specific replication of *Mycobacterium tuberculosis* in mice. *Nature* 402:79-83.
45. Perna, N.T. et al. 2001. Genomic sequence of enterohaemorrhagic *Escherichia coli* O157:H7. *Nature* 409:529-533.
46. The two strands of cellular DNA have complementary features in that adenine (A) nucleotides in one are always paired with thymine (T) nucleotides in the other. Similarly, guanines (G) in one strand are matched with cytosines (C) in the other. The cell uses the order of nucleotides in a segment of one DNA strand to order the sequence of amino acids in a protein. Three successive nucleotides (a triplet) on one strand are used to specify a single amino acid in the chain that constitutes a protein. Correspondence between nucleotide triplets and specific amino acids was determined in the 1960s and is now enshrined in a table called the “genetic code.” An important feature of the code is that there are 64 possible triplets of nucleotides but only 20 amino acids commonly found in proteins. Three of the triplets were found to act punctuation marks, leaving 61 to code for the 20 amino acids. Clearly, there could not be a one-to-one correspondence between triplets and amino acids. Indeed, some amino acids are specified by two, three, four or even six different nucleotide triplets. Consequently, it is possible to imagine two markedly different DNA sequences that would make precisely the same protein. Several of features of a DNA region are independent of the protein it encodes. GC/AT indicates the fraction of nucleotide pairs that are G-C (rather than A-T). Codon usage indicates which of the various synonymous nucleotide triplets are used to specify amino acids. Dinucleotide frequency

indicates how often the 16 different sequences of two nucleotides occur in a strand. GC skew indicates if there is a tendency for there to be more G nucleotides than C nucleotides in one strand. All these features tend to have a consistent value or pattern in DNA that typical of a single organism. Inconsistencies are interpreted as acquired sequences that came from some other organism.

47. Brand, L. 2003. What are the limits of death in Paradise? *J. Adventist Theol. Soc.* 14:74-85.
48. Taylor, B. 2003. Will there be death in Paradise? Faith and Science Conference, August 19; Mind and Spirit in Dialogue, University Church of SDA, January 27, 2006.
49. Völlm, B.A., Taylor, A.N.W., Richardson, P., Corcoran, R., Stirling, J., McKie, S., Deakin, J.F.W. and Elliot, R. 2006. Neuronal correlates of theory of mind and empathy: a functional magnetic resonance imaging study in a nonverbal task. *NeuroImage* 29:90-98.
50. Carr, L., Iacoboni, M., Mazziotta, J.C. and Lenzi, G.L., 2003. Neural mechanisms of empathy in humans: a relay from neural systems for initiations to limbic areas. *Proc Nat Acad Sci U.S.A.* 100:5497-5502.
51. Rizzolatti, G. 2005. The mirror neuron system and its function in humans. *Anat Embryol* 210:419-421.
52. Kaufman, K.R. and Kaufman, N.D. 2006. And then the dog died. *Death Stud.* 30:61-67.
53. Stabell, O.B., Ogbeto, F. and Primicerio, R. 2003. Inducible defences in *Daphnia* depend on latent alarm signals from conspecific prey activated in predators. *Chem Senses.* 28:141-53.
54. Van Speybroeck, L., Van de Vijver, G. and De Waele, D., eds., in *Epigenesis to Epigenetics: the Genome in Context.* Ann New York Acad Sci, vol. 988, 2002.
55. Judson, C. 2002. *Dr. Tatiana's Sex Advice to All Creation: The Definitive Guide to the Evolutionary Biology of Sex.*, Metropolitan Books
56. Riddle, D.L. and Albert, P.S. 1997. Genetics and environmental regulation of dauer larva development. In "C. elegans II", eds. Riddles, D.L., Blumenthal, T., Mayer, B.J. and Press, J.R. Cold Spring Harbor Laboratory Press, Plainview, New York, pp. 739-768.
57. Fuhrman, J. 2003. Genome sequences from the sea. *Nature* 424:1001-1002.
58. Arrigo, K.R. 2005. Marine microorganisms and global nutrient cycles. *Nature* 437:349-355.
59. Whitman, W.B., Coleman, D.C. and Wiebe, W.J. 1998. Prokaryotes: the unseen majority. *Proc Nat Acad Sci USA* 95:6578-6583.
60. Hamilton, G. 2006. The gene weavers. *Nature* 441:683-685.
61. Mann N.H., Cook A., Millard A., Bailey S. and Clokie M. 2003. Bacterial photosynthesis genes in a virus. *Nature* 424:741.
62. Suttle, C.A. 2005. Viruses in the sea. *Nature* 437:356-351.
63. Raoult, D., Audic, S., Robert, C., Abergel, C., Renesto, P., Ogata, H., La Scola, B., Suzan, M. and Claverie, J.-M. 2004. The 1.2-megabase genome sequence of Mimivirus. *Science* 306:1344-1350.
64. White, E.G., 1894. Redemption. *Adventist Review*, February 24, 1894.