Longevity in the Bible and Modern Science

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he beginning chapters of Genesis speak of people who lived for more than 900 years. Adam lived for 930 years,1 Seth for 912,2 Enosh for 9053 and Noah for 950.4 However, as early as Genesis 6:3 we learn of G-d's dissatisfaction with the corrupt behavior of people at the time of Noah and of His plan to decrease human lifespan. G-d declared: "...man's days shall be one hundred and twenty years." Ibn Ezra, one of the major Torah commentators, explained this verse to mean that human lifespan gradually decreased until it reached a maximum of 120 years. Peleg, who lived five generations after Noah, lived 2395 years, whereas Abraham, who lived ten generations after Noah, lived for 175 years.6 Moses, however, lived only 120 years.7 Moreover, Abraham was the first individual mentioned in the Torah who aged. In Genesis 20:2 the Torah informed us that "Sarah conceived and bore a son unto Abraham in his old age, at the appointed time which

What caused such a tremendous

decline of human lifespan and the appearance of signs of aging in Abraham and subsequent generations? To uncover possible answers to these intriguing questions reference is made to some modern scientific literature, which during the last two decades of the 20th century made a tremendous leap in our understanding of human aging. According to recent scientific data, mammalian cells have evolved an intricate set of checks and balances against uncontrolled cell proliferation. One check, termed programmed cell death, or apoptosis, is triggered by mutation of the p53 gene. Furthermore, the rate of DNA mutation correlates with the cells' lifespan. A mouse strain prone to accelerating aging was found to have an "...increased rate of somatic mutations accumulation compared with a corresponding strain that was resistant to accelerated senescence."8 Another check appears to be

G-d has spoken." By the time of the

Exodus from Egypt, extreme longevity

disappeared. Thereafter, the ages of the

people in the subsequent generations

were decreased to modern standards.

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the progressive shortening of the ends of chromosomes, termed telomeres, which are involved in normal cell division and may play a part in cellular aging.9 Kim and Piatyszck showed a correlation between uncontrolled proliferation of cancer cells and the expression of telomerase, an enzyme capable of preventing the shortening of the telomere.10 Their findings suggested that virtually all cancerous cells have activated telomerases to overcome the cellular biological clock. Furthermore, cancer cells are not the only cells for which immortality is dependent on telomerases. The extended life of normal cells is dependent on the activity of these telomerases. In fact, according to an editorial article in Nature Genetics "...normal mitotic cells are 'mortal' in culture and 'senesce' after a finite number of cell divisions. Telomere shorten-

ing has proved to be a potent molecular trigger of cellular senescence." By artificially introducing telomerase into several different cell types, researchers were able to stop the aging of the cells and extend

their lifespan.¹² Additionally, telomerase is critical for progression of stem cells to programmed adult cells. Indeed, mice deficient in telomerase had a diminished capacity to produce new blood and sperm cells.¹¹ Moreover, others reported that undifferentiated embryonic stem cells lacking telomerase are unable to proliferate.¹⁰

Another possible mechanism to account for cell aging and mortality is accumulated genetic damage in mitochondrial DNA. All aerobic cells require molecular oxygen to survive; they use this oxygen in oxidative metabolism to produce ATP in mitochondria. Without the energy provided by mitochondria, multicellular life would not be possible. Unfortunately, in the process of creating cellular energy, mitochondria produce free radicals. Mammalian cells have elaborate mechanisms to avoid free radical accumula-

tion. However, if mammals produce more free radicals than they can control, DNA damage may result. This genetic damage accumulates over time and is passed by mitosis to the next generation of cells. Such accumulation of genetic damage caused by free radicals has been implicated as a potentially major factor contributing to the aging process.13 Genes encoding for proteins that protect the cell from oxidative stress – such as superoxidase dismutases (SOD) and catalase - may therefore promote longevity. For instance, researchers found that the overexpression of SOD in the motor neurons of Drosophila extends its lifespan by 40%, as compared with wild-type controls.8

Another indication of the involvement of mitochondrial DNA in aging can be seen from epidemiological evidence presented by Sir-Masashi Tanaka

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and colleagues in support of the view that the maternal influence on longevity is greater than the paternal.¹⁴ Indeed, mitochondrial DNA is characterized by maternal inheretance.¹⁵ Finnish and Swedish genealogies also showed strong evidence that maternal mitochondria are at least partially responsible for longevity of cells.¹⁶

What is responsible for the accumulations of free radicals in the mitochondria that perhaps were absent at the time of Adam? Could it be that a change in the diet of Noah played a crucial role in creating inheritable DNA damage? We find in Genesis 8:3 that G-d gave permission to Noah to eat meat. Before the Great Flood all humans were herbivorous, eating seeds, fruits and green vegetation. Meat is a high calorie food containing saturated fatty acids. High fat consumption is implicated in many human maladies such as high

blood pressure, heart disease, and cancer. Animal (saturated) fat in general and red meat in particular are associated with several types of cancers and are strongly linked to malignancies of the colon and rectum. Saturated fats have been implicated in prostate cancer as well. What is missing in meat but is present in fruits and vegetables are antioxidants that neutralize free radicals. Other chemicals present in vegetarian foods may block the cellular signals of steroids, such as estrogen, that induce cell proliferation, especially proliferation of breast tissue.¹⁷

Moreover, diet can exert its effects not only through the type of calories consumed but also through their quantity. Rabbi Pinchas of Koretz once said: "Eating in moderation tends to lengthen life. We find among animals and reptiles that those who eat the least live

> the longest." Researchers now believe that taking in more caloric energy than is expended can be harmful to health. Sixty years ago, scientists at Cornell University discovered that placing rats

on a very low calorie diet prolonged their lives. The lifespan of these rats was increased by 33%, from three to four years. These researchers also found that rats on a low-calorie diet stayed younger longer and suffered from fewer chronic diseases, which were associated with old age. Many years later, similar experiments were performed on primates. Primates that were maintained on a calorie-restricted diet and supplied all important nutrients for animal survival had lower blood pressure, higher sensitivity to glucose, and showed fewer signs of aging.19 Furthermore, recent research indicated that calorie restriction can be useful even if it is not started until middle age. The calorie restriction initiated in mice at early middle age extended their maximum life span by 10 to 20 percent and prevented the development of cancer. Perhaps high calorie consumption accelerates free

radical production by mitochondria, thereby accelerating the destruction of cellular components and decreasing ATP production. A low calorie diet, on the other hand, slowed aging by decreasing the amount of free radicals that enter the electron transport chain.¹⁹

Some scientists think that, in addition to consumption of a low calorie diet, human beings need to postpone their reproductive age in order to live longer. These researchers view senescence as a by-product of a pattern of natural selection. It affects all vertebrates that reproduce sexually. Asexual plants and animals, on the other hand, do not show signs of aging because their offspring, which are genetically identical to their parents, need to be young in order to survive in a highly competitive world. For example, "...asexual sea anemones kept for

decades in aquariums do not show failing health."²⁰ On the other hand, sexually reproducing species age because natural selection declines after the start of adulthood. We can illustrate this point by

looking into the prevalence of two diseases, progeria and Huntington. Progeria is very rare. This disease is caused by a chance mutation in one copy of a gene in an embryo, resulting in rapid aging in childhood. Individuals suffering from progeria are not likely to reproduce and thus do not pass their deleterious progeria genes to the next generation. Although Huntington disease is also caused by a mutation, symptoms of this disease do not appear until midlife, where the afflicted individuals may have already produced offspring. As a result, Huntington disease is more prevalent than progeria. Hamilton found, based on mathematical reasoning, that "...for organisms that do not reproduce by splitting in two, the force of natural selection on survival falls with adult age and then disappears entirely later in life." This prediction has been shown to be true in fruit flies, which

after 10 generations of delayed mating, lived two to three times longer than control fruit flies that mated at their usual age.²⁰

It is curious to note that following the Great Flood, people produced their offspring at an earlier age compared to their counterparts who lived before the deluge. From Genesis 9:28 we see that Noah was able to have children when he was 500 years old. A son of Noah, Shem, also had his children at one hundred years old. However, Shem's subsequent children and grandchildren gave birth much younger. Arpachshad, for instance, "...lived thirty-five years when he begot Shelah."21 Shelah "...lived thirty years when he begot Eber."22 Isaac Abarbanel, one of the major Torah commentators, noted that "...the same difference in the biological clock that resulted in extreme longevity

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may also have delayed adolescence until the sixth decade or later" in the people who lived before the deluge.²³

Nahmanides, however, was of the opinion that Adam's early descendants lived extraordinary long lives primarily because of their biological perfection. According to his opinion, the reduction of the human lifespan was a result of the climatic change that was associated with the flood in the generation of Noah.²³

Furthermore, according to many classical Jewish sources, Adam was not the first human being to be created. The Talmud speaks of 974 generations of humanoid creatures that existed before Adam. These human-like species may have possessed physical and mental capacities similar to those modern men. The main difference between these human-like creatures and Adam was that Adam was created without a set biological clock. It was only after Adam

ate from the Tree of Knowledge that the biological clock was set to terminate his life; however, he still lived extremely long compared to the humans of today. According to Rabbi Aryeh Kaplan "...viewing the longevity mentioned in the Torah as a hereditary trait confined to Adam's descendants also explains how it was gradually reduced." Genesis 6:2 states "... the sons of god saw that the daughters of man were good and they took themselves wives from whomever they chose." According to the Midrash, "sons of god" were children of Adam who possessed the hereditary trait of longevity, and "daughters of man" were primitive humanoid creatures. Interbreeding between these groups resulted in an overall reduction of the lifespan in the descendants of Adam. G-d was not pleased with this interbreeding and with this decline of

> ethical standards of Adam's descendants, and He therefore declared in Genesis 6:3, "...My spirit shall not contend evermore concerning Man since he is but flesh; his days shall be a hundred and twenty

years."²³ In fact, modern scientific literature accounts for the existence of human-like species that "...have been joined by anatomically modern Homo sapiens" and their extinction only a few thousand years ago.²⁴

As we already have seen, the ability to bear offspring until the end of life is the quality of those animals and plants that have unusually long lives. These animals and plants appear to have delayed biological clocks. Moreover, their longevity is associated with a lack of signs of aging. Indeed, Noah was assigned a task of building an ark when he was 480 years old and finished at 600 years of age. Apparently, Noah's strength at 480 years was equivalent to that of a 30-40 year old modern man. Although we do not have, in the year 2000, all of the necessary technology to allow us to extend human life to its maximum potential, we should not despair. We have a prophecy found in

the Book of Isaiah that indicates that the extension of human longevity is indeed a possibility. Isaiah stated: "From then on, there will no more be one tender in years or aged... for as adolescent one shall die at a hundred years old." According to

many Jewish sources, the Messianic Age will not be a time of miracles; rather, it will be an age in which laws of nature will run their course. We may then assume, as stated in the teachings of Maimonides, that extension of human life could be possible solely by technological advances.²³ Hence, do not be discouraged... the 20th century brought us many rapid technological advances, and perhaps if we wait a little longer, we might find immortality.... DH

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NOTES:

- 1. Genesis 5:5.
- 2. Genesis 5:8.
- 3. Genesis 5:11.
- 4. Genesis 9:29.
- 5. Genesis 11:18-19.
- 6. Genesis 25:7.
- 7. Deuteronomy 34:7.
- 8. Anonymous. Comment on: "Be Fruitful and Immortalize." Nature Genetics 19:103-104 (1998).
- 9. Haber, Daniel. "Telomeres, Cancer, and Immortality." New Eng J Med 332: 955-956 (1995).
- 10. Kim, N.W. et al. "Specific Association of Human Telomerase Activity with Immortal Cells and Cancer." <u>Science</u> 226:2011-2015 (1994).
- 11. Lee, H.W. et al. "Essencial Role of Mouse Telomerase in Highly Proliferative Organs." Nature 392: 569-574 (1998).
- 12. Bodnar, A.G. et al. "Extension of Lifespan by Introduction of Telomerase into Normal Human Cells." <u>Science</u> 279: 349-352 (1998).
- 13. Carnes, Bruce. "Human Longevity: Nature vs. Nurture Fact or Fiction." Prospect Biol Med 42:423-437 (1999).
- 14. Tanaka, Masashi et al. "Mitochondrial Genotype Associated with Longevity." Lancet 351:185-186 (1998).
- 15. Russell, Peter. Genetics. 5th ed. Mento Park, California: The Benjamin/Cummings Publishing Company, Inc. 1998.
- Jalavisto, E. "Inheritance of Longevity According to Finnish and Swedish Geneology." <u>Ann Med Int Fenn</u> 40:263-274 (1951).
- 17. Trichopoulos, Dimitrios. "What Causes Cancer?" Sci Amer 275: 80-88 (1996).
- 18. Finkel, Abraham. <u>In My Flesh I See G-d, a Treasury of Rabbinic Insights about Human Anatomy</u>. New Jersey: Jason Aronson Inc., 1995.
- 19. Weindruch, Richard. "Calorie Restriction and Aging." Sci Amer 274:46-52 (1996).
- 20. Rose, Michael. "Can Human Aging be Postponed?" Sci Amer 281:106-111 (1999).
- 21. Genesis 11:12.
- 22. Genesis 11:14.
- 23. Kaplan, Aryeh. <u>Immortality, Resurrection and the Age of Universe: a Kabbalistic View</u>. New Jersey: Ktav Publishing House, 1993.
- 24. Tattersall, Ian. "Once We Were Not Alone." Sci Amer 282:56-62 (2000).
- 25. Isaiah 65:20.