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In Search of the Fountain of Youth
Preliminary Analysis of Deuterium's Role in DNA Degradation

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Evidence indicates that aging is coupled to a gradual accumulation of errors in DNA that arise due to strand breakage, DNA replication errors, or dysfunctional DNA repair mechanisms. Therefore, it is logical to consider factors that adversely impact DNA and explore the effect of removing them from the cellular environment. Although low-level solar radiation is the most pervasive mutagen known to adversely affect DNA, deuterium oxide is also pervasive and known to adversely affect DNA. Deuterium oxide is present in the Earth's surface waters at concentration of 155 parts per million (ppm). At this low level it is generally thought to have no effect. However, over long periods of time low levels of deuterium could play a role, especially when coupled with radiation and other mutagens that lead to DNA damage. New research indicates that although largely ignored, deuterium oxide may play a key role in the aging process.

Aging Theories Tied to DNA

The theoretical maximum lifespan that an organism can achieve is tied to its DNA and its intra-cellular processes. At the end of chromosomes is a strand of DNA known as a telomere. With each cell division some of the telomere sequence is lost, which in turn limits the number of times a cell can divide. Leonard Hayflick discovered that embryonic fibroblasts (connective tissue cells) could divide a maximum of about 50 times before the telomere is gone. (1) If cells continue to divide after loosing their telemeres, functional DNA is lost and cells soon malfunction. Once the "Hayflick Limit" has been reached chromosome ends begin sticking together and result in cells invoking apoptosis or senescent mechanisms, preventing mutations. This effectively establishes an upper limit on lifespan.

Lifespan and the number of fibroblast doublings are connected as well. Fibroblasts in mice with a 3-year maximum lifespan undergo 15 doublings, chickens with a 12-year lifespan undergo 25 doublings and the Galapagos tortoise with a 175-year lifespan exhibits 130 doublings. (2) These species differ in initial telomere length as well as in the number of telomeres lost in each cell division.

Fig. 1 Harriet, the world's oldest known living resident. Age: 173 years, born around Nov. 15, 1830. Brought back from the Galapagos Islands by Charles Darwin, as confirmed by DNA testing. (3)

If other factors did not come into play before these limits are reached, humans would perhaps live to be far older. One of the prime limiting factors is naturally occurring damage to DNA. In addition to being damaged directly by radiation, DNA is damaged by free radicals produced by radiation, mutagens, and normal metabolic processes.

Accumulation of DNA Errors

There is substantial evidence that aging is tied to a decline in the integrity of DNA. In research performed by Dr. Howard J. Curtis of Brookhaven National Labs, mice were irradiated with sub-lethal doses of radiation to test its long-term effects. This work was done back in the 1960s as part of the Gemini Astronaut Program. Astronauts are subjected to higher levels of radiation due to background space radiation and the lack of shielding in lightweight space capsules. Although astronauts were subjected to elevated radiation levels on Apollo missions, exposure times were relatively short. On a two year manned mission to Mars, radiation exposure becomes a significant problem.

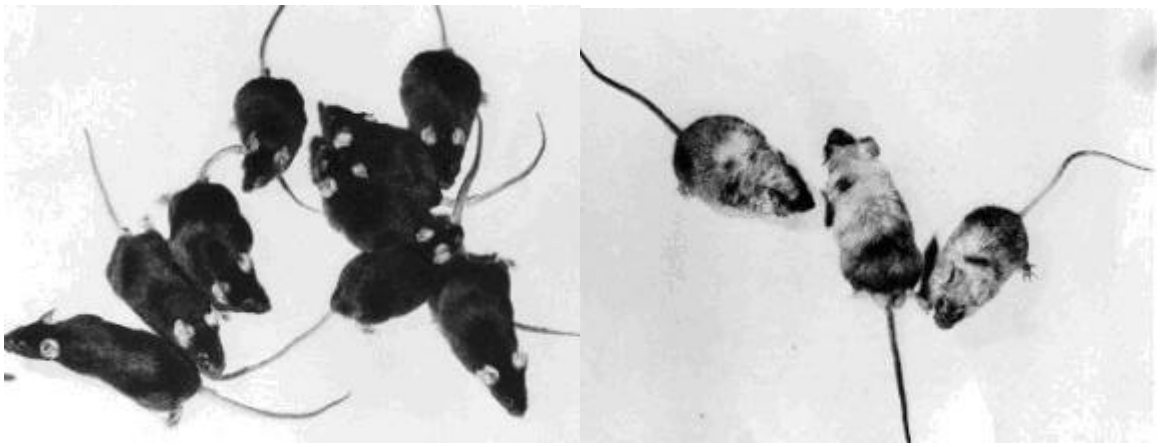

Fig. 2 Effect of Radiation on Aging. **"These mice pictured above are all 14 months old. As young adults, nine mice were given sub-lethal doses of radiation and nine others were left as untreated controls. The control mice (left) are still sleek and vigorous at 14 months, while six of the irradiated mice have died and the remaining three show signs of extreme aging (right). [Research photographs of Dr. Howard J. Curtis.]" (4)**

The Earth's atmosphere provides shielding from a large percentage of space radiation, but cosmic rays and energetic protons from the sun still penetrate the atmosphere down to the Earth's surface. This radiation in conjunction with natural terrestrial radioactive sources produce ionizing radiation that adversely affects DNA. In humans almost all DNA damage is repaired by effective repair mechanisms. However, in some cases DNA repair mechanisms are dysfunction and result in diseases that mimic the effects of aging.

Examples of Dysfunctional DNA Repair. Other clues that aging is DNA based may be found in rare inherited diseases that occur when genes that maintain the integrity of the genome are mutated. For instance when genes responsible for DNA repair are corrupted the result may be premature aging causing effects such as: wrinkled skin, gray hair, and shorten lifespan. In Werner's syndrome hair turns gray after age 20 and by the late 40s the patient shows signs of advanced aging such as cataracts, osteoporosis, and atherosclerosis. Werner's syndrome is caused by mutations in WRN, which encodes a helicase essential for maintenance of telomeres and DNA repair. Cockayne syndrome is caused by mutations in genes involved in transcription-coupled DNA repair. Although patients do not show signs of advanced aging, they do suffer greatly reduced lifespans.

Error-Free DNA Repair. It is possible that aging may not be caused by DNA mutations in general, but by mutations in genes required for error-free repair and replication of all DNA. In 1974 R. W. Hart and R. B. Setlow, published their paper: "Correlation between Deoxyribonucleic Acid Excision-Repair and Life-Span in a Number of Mammalian Species" (5) in which they measured the ability of fibroblasts to perform unscheduled DNA synthesis after UV irradiation. Fibroblasts' ability to perform unscheduled DNA synthesis is a measure of excision-repair.

Correlation between lifespan and the relative effectiveness of DNA repair in cells of certain mammals. In each case, cells growing in tissue culture were irradiated with ultraviolet light and then the efficiency with which they repaired their DNA was determined. (From the work of R. W. Hart and R. B. Setlow, 1974.)

Species	Average lifespan, yr	Effectiveness of DNA repair as measured by the amount of unscheduled synthesis (grains/nucleus)
Human	70	50
Elephant	60	47
Cow	30	43
Hamster	4	26
Rat	3	13
Mouse	2	9
Shrew	1	8

Fig 3. **"Aging represents the inevitable consequence of a failure of DNA repair" (4)**

Hart and Setlow found that "both the rate and extent of unscheduled DNA synthesis after UV irradiation of fibroblasts increases with the life-span of the of the species."(5) This model assumes that UV radiation effectively mimics normal wear and tear in cellular DNA. Hart and Setlow cautioned that there is "more to aging than just the failure of an excision-repair system for dimmers." (5) Nevertheless, these tests show that error free DNA repair is essential for species with long life spans.

Hitting the Aging Wall. It is general accepted that aging is not a linear process. The rate of aging increases with time with the result that humans age more rapidly at the end of a typical lifespan. The body can be viewed as an extremely complex feed back control system, employing multiple feed back control loops. When one feed back loop is compromised it can adversely affect others with the result that the entire system begins to perform in a non-optimal fashion. When this effect is coupled with a decline in the overall health of cells, the result can be a critical failure, i.e. death. In 1963, Orgel published the seminal paper on aging: The Maintenance of the Accuracy of Protein Synthesis and its Relevance to Aging. (6) Orgel proposed that protein synthesis will have some initial error rate **P**o and that the rate of error increase will be proportional to some constant alpha.

$$\frac{dp}{dt} = \alpha p \qquad [1]$$

The solution to this differential equation is:

$$p = p_o e^{\alpha t} \qquad [2]$$

This equation states that the rate of error accumulation in protein synthesis grows exponentially over time. Orgel conservatively proposed this model as one explanation for the progressive deterioration of cells and not as a model for aging of the entire organism. However, other aspects of the aging process also exhibit exponential growth, such as cancer rate as a function of age.

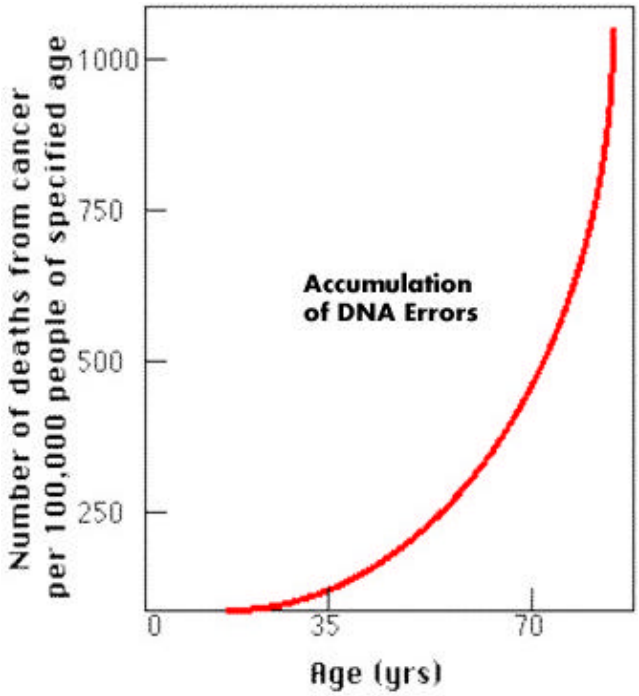

Fig. 4 Possible Link Between DNA Errors and Cancer Rates. **"Cells taken from old people (and people with premature aging syndromes) show marked reductions in the transcription of many genes, especially genes involved in DNA replication, DNA repair, and in checkpoints that ensure accurate mitosis of the cell. Many of these changes also cause cancer so it is no accident that the incidence of cancer rises with advancing age." (**4**)**

Cancer and a general decline in the integrity of DNA are undoubtedly related. An accumulation of DNA errors likely increases exponentially over time. This indicates that towards the end of life, one "hits a wall" where the integrity of DNA has been so compromised, that no method short of reordering all DNA would significantly extend life. At this juncture the quality of life is likely to be so severely diminished that there is no point in extending life further. Therefore a premium exists on maintaining the integrity of DNA starting early in life, so that the overall *length and quality* of life is optimized.

Ionizing Radiation Threshold for DNA Degradation

Literature addressing the biological effects of low-level radiation shows that gradual changes to DNA induced by radiation and other mutagens are below the threshold of current detection methods. It is established that acute radiation exposure will lead to premature aging, but the debate continues as to whether or not normal levels of radiation exposure lead to mutations. Ionizing radiation produces free radicals that attack DNA, but so do normal metabolic processes. Some groups even claim that low-level radiation triggers an "immune response", which protects DNA from further damage. Options on the subject seem to be influenced by political and financial considerations. Normal levels of ionizing radiation have little adverse effect, but it only takes a small number of mis-repaired strand breaks for DNA damage to accumulate over decades of exposure, and result in aging effects.

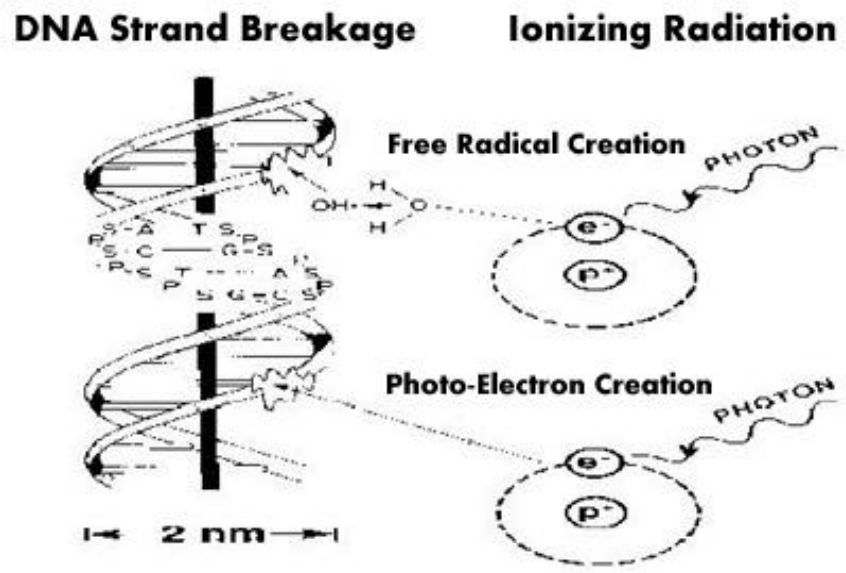

Fig. 5 Two Mechanisms of Strand Breakage Linked to Ionizing Radiation

What is unique about ionizing radiation relative to other mutagens is that it creates clusters of ionizations and reactive chemical agents on the scale of the DNA molecule. Dudley Goodhead has found that "this clustering occurs, even at the lowest exposures, within a pattern of ionized and excited molecules along the path (or 'track') of an individual particle." (7) Goodhead's research has revealed that "a high proportion of the DNA damage is complex, even from sparsely-ionized radiations, including combinations of several strand breaks and base damages (ie considerably more complex than clean double-strand breaks); these severe damages present a special challenge to the cell's repair systems, and it has been hypothesized that they may dominate the long-term consequences of the irradiation." (7) What is beyond question is that double stand breaks produced by ionizing radiation are more difficult to repair than single strand breaks produced by free radicals. It is likely that ionizing radiation impinging upon DNA during the replication phase results in damage similar to a double strand break and is also difficult to repair. Therefore, any factor that slows down the rate of DNA replication could expose DNA strands to a greater chance of corruption from ionizing radiation.

Deuterium, Mitosis, & DNA. It is known that high-levels of deuterium slow down the rate of mitosis, but the exact mechanism remains unknown. In 1989 Jan Lamprecht, Dieter Schroeter, and Niedhard Paweletz conducted a study on deuterium's effect on mitosis at The Institute of Cell and Tumor Biology and German Cancer Research Center in Heidelburg. (8,9) In one test, cells were subject to to 25%, 50% and 75% deuterium oxide for two hours. Another test was performed in which cell were subjected cells to 75% heavy water for two, six, twelve, and twenty-four hours. The percentages of cells in prophase, metaphase, anaphase, telophase, and interphase were then measured. The data showed abnormally high numbers of cells in prophase and metaphase and especially in metaphase. If DNA replication rate is slowed down during the prophase, then ionizing radiation could break strands when they are most susceptible to corruption. However, to verify this effect new tests must be performed with no deuterium present in DNA molecules and enzymes to determine if replication rates are increased over DNA containing normal deuterium concentrations.

Hydrogen and Deuterium Bonds in DNA

Deuterium is thought to have an effect on biological processes through the mechanism of hydrogen bonding. Hydrogen bonds play a role in DNA structure and are partially responsible for the double DNA strand assuming a helical shape. The hydrogen bonds created by a deuterium atom are stronger than a normal hydrogen atom.

Hydrogen Bond Strength. The presence of a neutron in the nucleus of the hydrogen atom doubles the atomic mass and thereby decreases the intermolecular vibration frequency. (10) This has the effect of increasing the hydrogen bonding strength. The physical properties of deuterium oxide differ only slightly from those of hydrogen oxide, or normal water. In aqueous solutions the hydrogen bond strength of deuterium oxide is greater by approximately 0.24 kcal/mol, which represents about a 6% increase over pure water. (10). The hydrogen bond strength in organic compounds is typically lower and is difficult to measure directly. However, by employing vibration mode partition functions from statistical thermodynamics, an accurate model may be constructed to calculate physical and chemical properties of deuterium oxide. Martin Cuma and Steve Scheiner made use of Gaussian codes to calculate in the increase in bonding strength due to substitution of deuterium for hydrogen within common organic groups. (11)

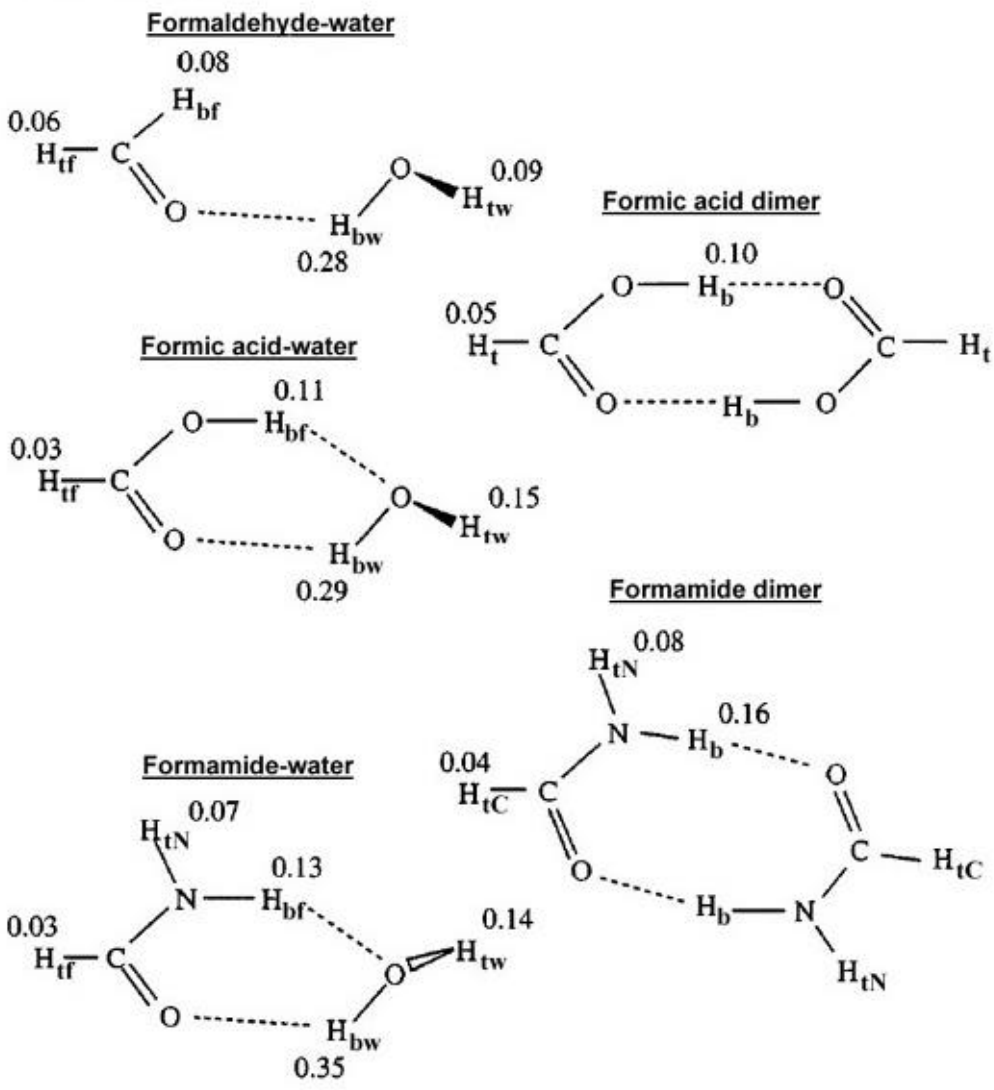

Fig. 6 The effect of deuterium substitution on hydrogen bond strength as appearing in: Influence of Isotopic Substitution on Strength of Hydrogen Bonds of Common Organic Groups (11) Dashed lines are hydrogen bonds. Substitution of deuterium for hydrogen has a comparatively greater effect on the overall bonding strength when the atom directly participates in a hydrogen bond. Values shown are in kcal/mol.

Organic Compound	H-Bonds	Min H-Bond D-Sub	Crit H-Bond D-Sub	Max H-Bond D-Sub
Formaldehyde-water	1.60 kcal/mol	0.06 kcal/mol	0.28 kcal/mol	0.50 kcal/mol
Formic acid dimer	10.84 kcal/mol	0.05 kcal/mol	0.10 kcal/mol	0.29 kcal/mol
Formic acid-water	5.97 kcal/mol	0.03 kcal/mol	0.29 kcal/mol	0.57 kcal/mol
Formamide dimer	8.32 kcal/mol	0.04 kcal/mol	0.16 kcal/mol	0.55 kcal/mol
Formamide-water	4.52 kcal/mol	0.03 kcal/mol	0.35 kcal/mol	0.68 kcal/mol

Table 1 The combined strength of the H-Bonds is modified by the presence of deuterium atoms in H-Bonding sites. Substitution of some hydrogen atoms with deuterium atoms have very little effect such as those shown under the column Minimum H-Bond D-Sub. Others have a large effect, such the values shown under the column Critical H-Bond D-Sub. The maximum effect occurs when all hydrogen atoms are replaced by deuterium atoms as indicated under the Max H-Bond D-Sub column.

Organic Compound	Min H-Bond D-Sub Incr	Crit H-Bond D-Sub Incr	Max H-Bond D-Sub Incr
Formaldehyde-water	3.75%	17.50%	31.25%
Formic acid dimer	0.46%	0.92%	2.76%
Formic acid-water	0.50%	4.85%	9.54%
Formamide dimer	0.48%	1.92%	6.61%
Formamide-water	0.66%	7.75%	15.04%

Table 2 The percentage increase of bonding strength over the normal H-Bond strength is shown the table above based upon the values reported in Table 1.

Hydrogen Bonds in DNA. In DNA the hydrogen bonds of interest are the G-C and A-T bonds that form between the strands of the double helix. The exact value for the strength of deuterium bonds in DNA is difficult to assess. Estimates of the strength of individual hydrogen bonds in DNA have been made by Turner and Sugimoto, but there is debate over the accuracy of their model. (12) Griffiths reports that deuterium bonds in enzymes that act upon DNA are typically 0.4 to 1.7 kJ/mol stronger than for normal hydrogen bonds. (13) The substitution of deuterium for hydrogen with DNA undoubtedly affects the bond strength, however, determining the degree of this effect is very difficult and can only be approximated by computationally intensive numerical methods.

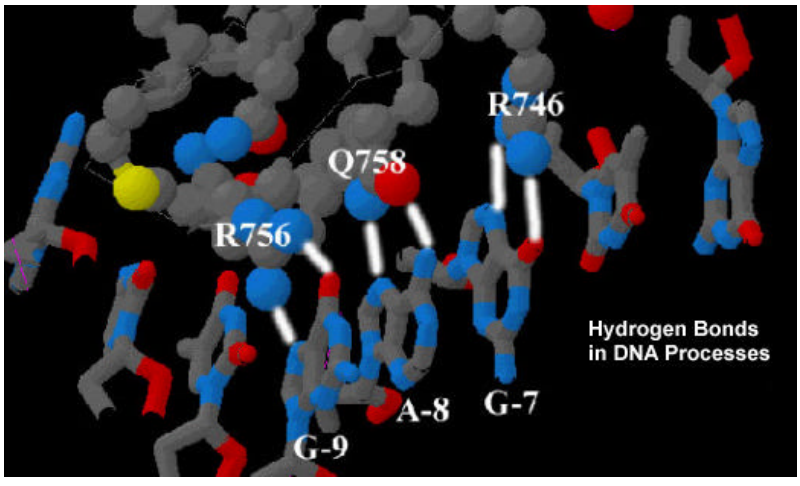

Fig. 7 Example of Hydrogen Bonds in DNA Processes. **"RNA polymerase transcribes specific genes that are found in the DNA. The polymerase recognizes these genes because they have PROMOTERS.**

A PROMOTER is an RNA polymerase binding site in the DNA which comes just before a gene. For T7 RNA polymerase, the DNA sequence that makes up the promoter is TATAGTGAGTCGTATTA in the template strand. RNA Polymerase recognizes the promoter sequence by hydrogen bonds: Arginine 756 makes two hydrogen bonds to Guanine-9, Glutamine 758 makes two hydrogen bonds to Adenine-8, and Arginine 746 makes two hydrogen bonds to Guanine-7. These are only a few of the protein-DNA interactions involved in promoter recognition". (14)

Length of Hydrogen Bonds. Of prime importance in DNA replication and repair is the shape of enzyme molecules that govern these processes. Deuterium shortens the bond length slightly and may inhibit proper functioning of enzymes. However this effect is likely to be small, on the order of perhaps a 1% change.

In general, hydrogen bonding in DNA is a cooperative process that effects stacking interactions and involves the entire molecule. If we use enzymes as a guide, the bond strength of deuterium in DNA is perhaps 0.5% to 2% greater than for hydrogen. When considering the small concentration of deuterium found in nature, and the slight increase in the strength of deuterium bonds over hydrogen bonds, one might be tempted to conclude that deuterium has no appreciable adverse effect on DNA at a concentration of 155 ppm. However, in addition to increased hydrogen bonding strength, deuterium has other potentially adverse effects.

Plausible Mechanisms for Deuterium Adversely Effecting DNA

Deuterated Enzymes: As early as 1974, deuterium was advanced as a possible cause of aging. One notable theory is that deuterium adversely affects the shape of enzyme molecules, which are involved in DNA processes. This is the central concept advanced by Griffiths in: The Possible Roles of Deuterium in the Initiation and Propagation of Aging and Other Biological Mechanisms and Processes

> **"When deuterium is involved in a chemical reaction, consideration must be given to a slight change in the inductive effect, as deuterium is more electronegative than hydrogen. Hyperconjugative effects are also involved since CD3, for example, is less delocalized than CH3, and, more important, the effective size of a C-D bond is smaller than the effective size of a C-H bond. Thus steric effects have a part to play, reinforcing our contention that any highly stereospecific enzyme molecule containing a deuteron in an important position has a potential for participating in an error reaction." (13)**

Deuterium Compromised DNA Repair Enzymes. A large class of enzymes and proteins play a role DNA replication and repair. (15) Some enzymes used during DNA replication and repair make extensive use of hydrogen bonds. These enzymes are potentially highly susceptible to adverse effects from deuterium contamination. One notable protein is p53, which plays a significant role in DNA repair. "Several different types of DNA damage can activate p53, including double-strand breaks in DNA produced by gamma-irradiation and the presence of DNA repair intermediates after ultra-violate irradiation or chemical damage to DNA."(16) It is interesting to note that over 50% of human cancers contain mutations in the gene that produces the p53 protein.

Slowing of DNA Replication. Deuterium could also inhibit an enzyme such as DnaB, which is responsible for unwinding and separating DNA during replication. Other enzymes such as Primase and polymase play key roles in synthesizing RNA and in adding nucleotides to the DNA chain during replication. If any of these enzymes are inhibited, the rate of DNA replication could be slowed appreciably when the DNA is most susceptible to damage by radiation. Thus deuterium could also act as a catalyst for DNA degradation when coupled with common levels of radiation exposure.

Bonding Site Inhibition of DNA Repair. The process of DNA replication is in some ways similar to the repair of a double strand break. Complex enzymes detect breaks and rejoin DNA strands based upon specific bonding sites. Hydrogen bonds are often employed within these binding sites. If deuterium is present in these sites, steric effects and increased bonding strength could also inhibit DNA repair. Once again, if the rate of DNA repair is severely curtailed, ionizing radiation could further disrupt the repair process while the strand is broken and is susceptible additional radiation damage.

Deuterium Studies on Organisms

Fully deuterated water, known as heavy water, is toxic. It is primarily used as a moderating agent in nuclear power plants. Largely due to the fact that heavy water is readily available, but deuterium depleted water (DDW) is scarce, relatively few studies have been conducted on the biological effects of DDW verses heavy water. It would normally stand to reason that if increasing deuterium levels above those found in nature has no measurable effect, then decreasing deuterium levels will have no effect either. However, one must considered that deuterium has been in the environment for a *long time*, and therefore it is possible that through evolution, humans and other organisms have developed mechanisms to protect against it, or eliminate it. In a manner somewhat analogous to a pH buffer, cells may be able to moderate the adverse effects of increased levels of deuterium. That the human body has some means of eliminating deuterium is evidenced by the fact that deuterium levels in the body are typically 80% of that found in the nature. It stands to reason that if deuterium had no harmful effect on the body, the body would not attempt to expel it. Finally, one must considered that all organisms on the planet are exposed to low levels of deuterium throughout their entire lifetime. In the absence of a control group, the effects may not be obvious.

Deuterium Studies with Algae. Much of the work studying deuterium's effect on biological organisms was performed at Argonne National Laboratory in the 1960s. Work continues to this day primarily on various strains of blue-green algae. Most algae may be grown in 100% heavy water, but at a significantly reduced rate as compared to within tap water.(17) An organism's success in adapting to growth in pure heavy water is to some degree tied to the complexity of its DNA. Organisms with relatively small genome sizes, (on the order of 3-4 million base pairs for algae and bacteria) and that lack sophisticated DNA repair mechanisms, (18) can successfully adapt to growth in pure deuterium oxide. Mammals, such as mice, with genome size on the order of 3 billion base pairs cannot have more than 25% of their body water replaced with deuterium before going into convulsions. (19)

Mice Studies with High Levels of Deuterium. Studies have been performed on mice in which they consumed deuterium at a 20% to 30% concentration, after which they were irradiated with at near lethal levels. (20) In one series of tests mice were deuterated for 12 days after which they were exposed to 8.5 Grays of radiation. In one case mortality was significantly less in deuterated than in non-deuterated mice. In another test "mortality from whole-body neutron-boron radiation, unlike mortality from whole-body x-radiation, was not lowered by deuteration." (21) These test were done to evaluate the use of deuterium and radiotherapy in the treatment of malignant human tumors. It is possible that by inhibiting cell division high levels of deuterium could protect against short-term radiation exposure. However, there are adverse side effects from such elevated levels of deuterium, which limit its clinical application.

Hungarian and Romanian Deuterium Studies

Only in the last ten years has a program been developed to study the effects of less deuterium on the human body. Most of these studies have been performed in Hungary and Romania where low cost deuterium depleted water is available. The studies have involved cancer in both humans and animals.

Radiation and Low Level Deuterium Studies on Mice. Of particular note is a study performed by W. Bild, et al at the Romanian University of Medicine and Pharmacy. (22) In this study mice were fed DDW at a 30 ppm concentration for a 15 days period during which they were exposed to a sub-lethal dose of 8.5 Grays of radiation. A control group of mice were fed tap water and exposed to same level of radiation. The test group had a survival rate of 61% while the control group's survival rate was 25%. The test group also maintained normal white blood cell and red blood cell platelet counts as compared to the control group. Test group mice that were infected with K. pneumonia 506 and S. pneumonia 558 in addition to being irradiated or treated with cyclophosphamide showed increased non-specific immunity parameters. Test results generally showed an intensification of the immune defenses and increased proliferation of the peripheral blood cells over the control group, which may accounted for some of the radiation protective effects. This test was done to evaluate the effects of DDW on chemotherapy patients. However, the results may also point to a relationship between the adverse effects of radiation and deuterium as they relate to aging.

Possible Mechanisms for Observed Effects on Mice. As outlined earlier, deuterium slows down the rate of mitosis and can conceivably have adverse effects during the S phase of cell division when DNA is most susceptible to radiation. A radiation dose of 8.5 Grays would have resulted in double strand breaks as well as greatly disrupted DNA processes during replication. It is conceivable that mice with lower levels of deuterium in their systems would have been benefited from less error prone cell division and more effective repair of radiation damaged DNA. Such effects could be viewed as a greatly accelerated case of what happens over a lifetime of exposure to solar radiation. Even at very low levels, deuterium could slow down DNA replication processes, or otherwise interfere with the repair of DNA damaged by solar radiation. Over a lifetime of exposure to low levels of *both deuterium and solar radiation*, errors could accumulate in DNA and contribute to aging.

Deuterium Depleted Water Trials on Cancer Patients in Hungary

Although there is relatively little data on the effects of DDW on healthy people*, there is a wealth of data on the effects of DDW on cancer patients. Gabor Somlyai has been successfully using DDW to treat cancer in Hungary for the past ten years. (23) Results have come from double-blind clinical trials, and compassionate use of DDW as an adjuvant treatment.

Clinical data appearing in: "The Biological Effects of Deuterium Depletion" by Gabor Somlyai

"Interim evaluation confirmed a significant difference between the control group and treated groups with respect to the examined parameters that indicated the anti-tumor effect of the preparation.

- **a) At the time of the 5th and 6th visits, the ratio of patients showing an increased efficacy (PR) was significantly higher statistically (5th visit: p=0.0096; 6th visit p = 0.021 in the treated group.**
- **b) The volume of the prostate decreased significantly (p =0.043) in the treated group, whereas it could be regarded as unchanged in the control group.**
- **c) The number of patients with a decreased prostate volume was significantly higher (exact Armitage-test: p=0.015; exact Fisher-test: p =0.011).**
- **d) Significantly more patients reported a positive chance in symptoms in the treated group (exact Armitage-test: p=0.0009; exact Fisher-test: p=0.0018).**
- **e) The survival rate in the treated group was significantly higher (p=0.030)**

After the consumption of more than 10 tons of Dd-water no event endangering life occurred. We did not experience any deterioration in blood counts, irritation of the mucous membrane, nausea, headache, etc., that could have been attributed to Dd-water consumption.

Compassionate use and as an Adjuvant Treatment:

Prior to and parallel to the above trials, between October 1992 and the spring of 1999, we provided Dd-water to approximately 1200 patents. Our knowledge concerning the efficacy and application methods of Dd-water comes mainly from the follow-up of this patient population … During the last 8 years we provided about 350 tons of Dd-water to the patients and some 12-14 thousand pages of documentation records the data of the meticulous follow-ups. Recommendations, comments, dosage advice, and results are based on these observations.

Between October of 1992 and December of 1997, 887 patients began to consume Dd-water. Among them, 134 patients (15 percent) were diagnosed with breast cancer. The ratio show that patients with breast cancer were represented in an approximately equal ratio among patients consuming Dd-water, to that of the entire population (in the US, for example, 13 percent of all cancers is that of the breast)" (23)

* Note: For the past three months the author has been consuming 36 liters of DDW per month at a 105 ppm concentration with no ill effects. The author has experience a slight increase in stamina during strenuous exercise, but this effect is hard to quantify or verify. Trials with DDW-25 will begin soon.

Statistical evaluation covered 55 patients in an advanced state (A).

Figure II. 4 shows the survival data of breast cancer patients in an advanced state and consuming Dd-water.

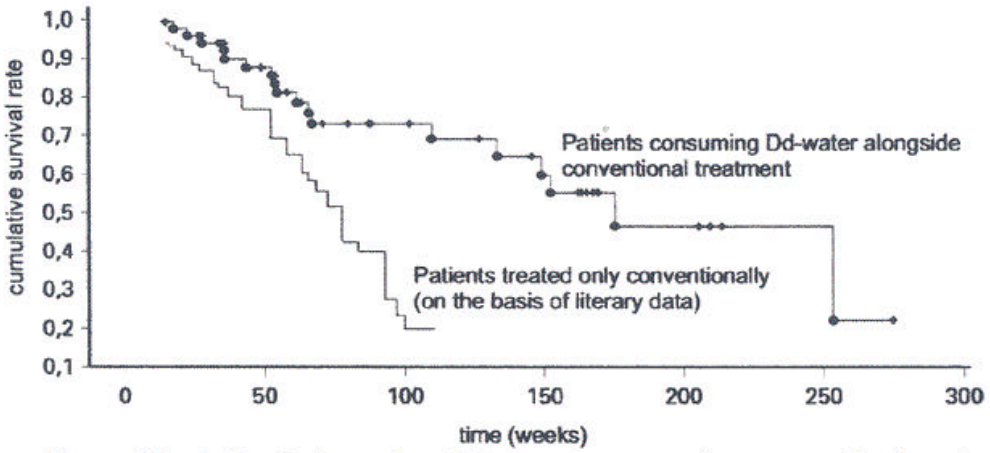

Figure II. 4 Survival graph of breast cancer patients treated after the appearance of distant metastases

"It is clear from Figure II. 4 that 85 percent of the above population survived one year after the beginning of the Dd-water consumption in an advanced state, 56 percent survived 3 years, 47 percent 4 years, and 25 percent 5 years after the beginning of consumption of Dd-water." (23)

Thus there is plenty of clinical evidence that DDW consumption does have a measurable effect on cancer and since cancer and aging are related processes, there is a good chance that DDW consumption could have an anti-aging effect.

Discarded Hunza Data Revisited

Although deuterium levels in glacier fed streams is known to be less than what is found in surface water, the effect is diminished by rainwater entering streams as they flow down to cultivated areas. There have been numerous claims of people in mountainous regions living exceedingly long lifetimes, even approaching 150 years, but few of these reports have been validated. There was interest in the Vilcabamba region of Ecuador, the Caucasus region of Russia, and the Hunza region of Pakistan due to an article by Dr. Alexander Leaf in the January 1973 issue of National Geographic. (24) However, the Vilcabamba cases were refuted by R.B. Mazess in 1979 and in 1982. (25,26) Likewise, the longevity claims of the Caucasus region arose due to age exaggeration on the part of individuals attempting to avoid military service. The politics of the former Soviet Union also fostered an image of superiority of people from the region where Stalin was born. (27) Indeed, the geography of these regions does not promote low deuterium levels in melt water runoff and does not fit the model proposed here. However, the Hunza region is different in this regard. Unlike the Vilcabamba and Caucasus regions, the Hunza region receives very little annual rainfall, only about 4 inches. (28) The Hunza people receive their drinking water from glacier runoff, which is also used to irrigate their crops. The area is in may ways a high altitude desert. Although published deuterium levels for the Hunza region could not be found, we can make a very good estimate based upon "Deuterium Content of Stream Waters of Glacier Origin in the Himalayas" by Hisao Wushiki. (29)

Himalayan Geological Studies Involving Deuterium. In his 1977 Glaciological Expedition to Nepal, Hisao Wushiki measured the deuterium content in a number of streams that feed the Sun Kosi River. The levels vary by time of the year and location. Streams at higher elevations during the winter and post-monsoon seasons have the lowest deuterium levels. The values ranged from –66 SMOW (Standard Mean Ocean Water, this scale ranges from 0 at 158 ppm to –1000 at 0 ppm.) for the Sun Kosi river to -170

SMOW for the Ronabuk tributary located on the backside of Mt. Everest, farthest away from India. Much of the snow in the Himalayas arises from moist air that flows up from India during monsoon season. This water is rich in deuterium, but as it passes over the mountains the heavy water precipitates out first, leaving deuterium depleted water, which falls as snow in the higher elevations.

Fig. 8 Terraced Hunza Fields Irrigated by Glacier Water Containing Lower Levels of Deuterium

Inferred Hunza Deuterium Level. Based upon geography and altitude we can infer that the deuterium content of the Hunza region is comparable to the runoff from the glaciers associated with Mt. Gosainthan which is at an altitude of 8013 meters and located approximately 700 km inland. Runoff from the Gosainthan glaciers is at –160 SMOW, which corresponds to 133 ppm or about a 16% reduction over normal surface water. The Hunza people receive their water from the glaciers of Mt. Ultar, which is at an attitude of 7398 meters and located 1600 km inland. For this reason Hunza water most likely has a deuterium concentration at or below 133 ppm. A 16% reduction may not seem significant, however, the Griffiths theory predicts that the adverse biological effect of deuterium goes by the square of the concentration. (13) Although the true age of the Hunza people is difficult to verify, there is ample evidence to support the claim that the elderly people of the region are vigorous and long lived. This has been loosely tied to diet and exercise, but it could also be attributed to lower deuterium levels in the water and food of the region.

Effectiveness of Consuming DDW

Consumption of DDW differs from antioxidant formulas, HGH stimulants, vitamins, and other anti-aging remedies in one key aspect: DDW does not change its chemical composition when digested. All DDW consumed has a direct effect at the cellular level. However, for DDW to have a measurable impact, existing deuterium must first be leached out of the body. Through thermal substitution reactions deuterium atoms are replaced over time by regular hydrogen atoms. This process proceeds fastest when the concentration of deuterium in drinking water is at the lowest possible level. Unless food that is consumed is also grown with DDW, there will always be deuterium in the diet. Consumption of DDW can conceivably protect DNA from damage and assist DNA repair mechanisms, but it does not directly repair DNA. It is therefore questionable whether DDW consumption will “rejuvenate” the body, but it certainly could serve to protect the body and enable it to function more efficiently.

Work in Progress & Suggested Research

At this writing a sample of Hunza water is being collected from streams close to the glacier source. Within 60 days this sample should be available in the United States for deuterium level testing by an independent lab. This will help to establish actual deuterium levels in glacier runoff within the Hunza region.

When the theory of enzymes compromised by deuterium was advanced in 1974, many of the tools to explore the DNA at the molecular level were not available. A highly desirable experiment would be to deuterise key enzymes involved in DNA replication and repair and then determine if the rate of these processes is significantly inhibited.

As of July 2000 researchers at Brookhaven National Labs published reports highlighting new technology for assessing damage done to DNA by double strand breaks. Special enzymes are used to cut DNA at sites exhibiting specific kinds of damage. These segments are then separated and counted on electrophoretic gels to measure clusters of damaged DNA. Such tests could be repeated using partially deuterated DNA, or fully deuterated DNA taken from algae grown in heavy water.

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