What is aging?
How can life be prolonged?
From: An Advanced Treatise in QUANTUM BIOLOGY

As we have proven in our treatise on Quantum Biology, the laws of thermodynamics describe only dead nonliving systems. The rule of life or "organos" is not thermodynamic but is quantic. Only this type of description can be used to understand biology. From this fact we must look at aging in quantic terms, then we will find a rather unique situation that offers us some intriguing solutions or treatments to delay and perhaps one day prevent aging.

Quantitative rules for any type of situation as complex as biology, given the limited knowledge that we possess as human beings, must be taken with a grain of salt. Perhaps we need to develop more guidelines until we understand different rules leading to different laws of the phenomenological actions integral for life.

As Isaacs points out, in developing the uncertainty rule dealing with information handling in cells, we can predict that the existence of informational fields will be developed between very large molecules. Such informational fields will be directionalized and bonded through the long range forces, as we have discussed in previous chapters. These directionalized bonds in long range forces will be very prominent in the role of organization in the DNA/RNA in development of proteins and nucleic acids.

The replicative process of DNA can only be explained to date by these long-range forces. The recognition occurring between cell surfaces and receptor sites, and their attraction to each other can be explained through these long-range forces. Antigen and anti-body reactions are specific, and a lock-and-key type theory of chemicals is not sufficient to explain the different concentration levels of activity in biology. Something must attract the key to the lock.

Enzymes can act because of the large surfaces of favorable collisions that are impacted. In order for high K values to be utilized with the enzyme molecules, there must be enough activation energies with the presence of enzymes. Since enzymes are small dipole magnets, they are brought into the field, and in the presence of base activation energy the enzymatic process can be activated.
Activation energy can happen in the presence of enough heat energy, charge, momentum, or mass. If there is enough of any one of these types of energy, it can start and activate the enzymatic process. Charge would help the attraction of the molecules. Heat would help by increasing the kinetic energy of the molecules. If there is enough mass increased in the field of the enzymatic action, by increasing the substrate and/or the components, then this will increase the probability of striking each other; and can increase the enzymatic effect, if two items, enzyme and substrate, are driven together by momentum.

Thus we can see that the combination of these energies might provide enough activation energy to start and activate the enzymatic process. Since the enzymes are the most prominent things in biology, their action is highly important. Enzymes indeed make life possible. They are small dipole magnets, and in some cases, have some profound magnetic fields. This accounts for some of their heat sensitivity, as they are temperature-viable. In the case of the activation energy needed to supply these different enzymes' interaction, if there is too much of an energy presence, then it can shut down the enzyme action. Here again we are faced with biology's law of dualism, in which too little or too much of an enzyme can cause different problems.

The polarity of enzymes and their magnetic action can be explained only through long-range forces. As we find with the long-range forces, these things will be dependent on virtual photons, and thus will be photic. The electron transport process within biology is photic and dependent on photons.

Such photons, magnetic action, long-range forces, and perhaps even some undiscovered phenomena will account for the informational fields that are set up in biology. Even the polymorphic resonance of vibrational fields of these large molecules could have some dramatic effects in biology. The rather long wavelengths associated with such vibrations, possibly on the order of a thousand angstroms to several microns, would increase the long-range effects. They also can be very specific for reasons that these vibrations assume discrete values, and the strength of coupling depends on the occurrence of like modes in the interacting molecules. Thus the coherence can help overcome some of the distance variables. Coherent radiation is much more penetrating, as it has a longer range of action.

Isaacs describes these forces as an exchange of virtual photons between molecular oscillators under nonconventional statistical conditions.

We can see that there are definitely vibrations in biology, and that these take the place of photons, electrons, molecules, and even cells, which can occupy different vibratory statutes and set off different types of vibration photons or long-range forces. Whenever an electron, photon, or charged particle is moved in space, it sets up a magnetic field. The right-hand rule of electronics says that if an electron travels along the line of your thumb, there is a magnetic field developed at 90° to that line. There is also an electrostatic field developed at 90°, so that the electrostatic, magnetic, and electron-conductive flow make up a three-pole field that is essential to our understanding of energetic phenomena. All of these set up vibrations to the release of photons, in the knowledge that photons are responsible for the autodynamic energies.

Dr. Rife developed a Rife machine that utilized different types of these vibrations to treat different diseases. Just as a sound vibration might shatter a glass at a certain pitch, Rife found that certain vibrations might shatter other crystalline structures. He found that viruses were such crystalline structures. There are many liquid crystal effects; even DNA was found to be crystalline in its structure.

Rife will be discussed later. His work was ahead of his time, and the technological
achievements that he made were disdained by chemical thought patterns; they couldn't accept Rife into modern medicine. Perhaps now, after the advent of more modern theories, Rife may be reanalyzed (see Bio-Quantum Matrix).

VIRUS
CRYSTALLINE
STRUCTURE

A specific frequency can destroy a virus, just as sound can shatter a glass.

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Rupert Sheldrake's book on "Morphic Resonance" outlines the precept for different types of shapes and shape theories in the development of thought, and many other types of chemicals, crystals, etc. In line with these ideas, the current theory of one gene representing one amino acid sequence for one enzyme protein in the form of information storage, information handling is seriously flawed. We must now look at the more large-scale phenomena of these reactions, and how some of these genes can have multiple determinate factors and accomplish different tasks. This is again in the idea of the reductionistic mind of current today thinking in biology, and how seriously flawed that type of thinking might be. Some of this information may be carried on cytoplasmic elements themselves. The proteins might participate in their own code.

Sheldrake's concept of morphic resonance was applied to thoughts, actions and other human events. Even in the animal and insect kingdoms, there were found to be several instances in which morphic resonance explained phenomena that could not be explained by standard chemical entropic thermodynamic science. But as we look more closely at Sheldrake's theories and philosophy, we will find the need for a different perspective, something that might go beyond the dimensions of time and space and into the other dimensions.

Sheldrake challenges us to think about different ideas and entities, and our chapter on onionic forces will perhaps give some insight as to how morphic resonance might be explained. Timing and phasing under quantum rules of information interactions will soon be important in larger functions described now to inducer and suppressor genes. Information can be transferred through timing of operations. The quantic aspects of nonmechanistic interaction would effectively insulate the information from thermal and entropic degradation, while letting some thermal agitation in from time to time to stir the pot, as it were. Thus we can see the need for Brownian motion before mitosis. This is part of the process of biology.

Information transfer for coding and genetics is going to be hard to interpret from chemical intervention only. As we attempt to interrupt through our studies by using DNA crystallization techniques and the like, we are actually studying a very synthetic process, not a natural one. Most of the genotype project, thus, must be taken with a grain of salt. As we find different genes predictive of certain types of diseases, we will have to challenge the idea of the reductionistic simplicity of this claim. Mutual feedback of the other genes, as well as environmental feedback, has a very large interference. This is indeed a multi-body problem. Thus a nonlinear, non-reductionistic, quantic type of thought pattern that we wish to develop through this book would dictate that we must move very cautiously in trying to develop new medical techniques from the results of the genotype project.

The information that we glean from this large statistical challenge called the genotype project, looking at the different genes chemically and what types of processes they are involved with, could be very limited if we rush into an over-reductionistic view. We must be constantly reminded that the process does not work in that simple a form. The process of life is a very complex interchange of information that is happening on many, many channels with feedback.
and dependency involvement. To be over-reductionistic and try to reduce factors into some simple operandi would be making a drastic mistake. Thus the principle of complementarity will lead us to the idea that information is a very complex process of interchanges happening in vast arrays that cannot be calculated without the help of machinery. Even though the human mind in the twentieth century attempts to reduce functioning to its simpler form, it is a mistake to think that this simple map is actually reality.

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In the twenty-first century we will have a different viewpoint; man will evolve the thought processes needed to realize that nature works, and there is a vast complexity of the natural process. As we see the limitations of reductionistic thought, we will finally learn to revere nature for its ability to control life. We will finally realize that nature is smart and man is dumb. Nature is doing; man is guessing. Only nature knows. Instead of developing institutions and prizes that revere man's limited interventions of allopathic medicine or against natural medicine, medicine will recognize nature's vast, awesome, incredible superiority. Then true healing of all of mankind's ailments can proceed.

Thus our new science will need to be built on reverence for the natural process, connection to the environment, and finally, some type of a religion, or at least connectedness; this is what religion truly means. This reverence will guide us to a new level of thought, a new level of respect for the natural process and the environment. As we find, the exchange of biological information in the DNA requires the existence of the total DNA, and requires the rest of the cell to interact with the DNA. To over-reduce the phenomenon is a very chronic mistake.

As we pointed out in Chapter 8 of the Promorpheus, there is a vast intercellular, regulatory process, not only of photons but also of hormones. This interaction between the environment of the cells adds to a very complex situation in determining DNA utilization. Sexual conjugation for cellular life forms is an informational correction device. As Isaacs puts it, at certain times we need new DNA, because the old DNA is starting to give way to thermodynamics, or entropy. Just as in the Navy, we need to periodically have a new captain restored to the ship. When a new captain comes into the ship, he brings order to the ship, making sure that the ropes are tight and that the decks are swabbed at their regular intervals, and everyone performs to the height of their ability. This is the order and control needed to help the ship run. Then as time goes by, the captain will be more lackadaisical in his workings with the sailors on the ship; the decks are not swabbed quite as regularly, and the ropes are not quite as taut as they might be. This type of give-way to entropy and thermodynamics eventually dictates that the Navy must bring in a new captain. The old captain is starting to get too familiar and lax. Such is the necessity for sexual conjugation.

Royal Lee has outlined the build-up of protomorphogens in the cells and the interstitial fluids of the cells, where old types of DNA in broken form accumulate in the cells. We heartily recommend reading of the treatise of "Protomorphology" given by Royal Lee as an understanding for this phenomenon. Thus the breakdown of essential information can be recovered through asexual cell lines over several generations, and reversed by coupling with large molecules of the cytoplasm of a new cell, and thus to overcome the rate of information loss. Sexual conjugation offers advantages in preventing errors of information handling by
one-to-one reproduction, rather than one-to-two reproduction, as in asexual fission, or mitotic division.

So sexual conjugation developed as an alternative to aging, so that the DNA can live on. Aging is the subtle decline of the body's ability to fight entropic thermodynamics. Subtly, certain changes start to happen as the organism loses its quantic information control and subtly allows in more thermodynamic entropy.

As we have shown, the vibrational modes of the different large molecules set up some long-range resonant frequencies through the release of virtual photons. There are also electro-elastic collisions, which are happening as a result of the long-range forces. As we have developed in the early part of this chapter, this is an important part of cellular information, and all cells will need to rely on this type of information transport to regulate their emergent processes of reproduction and metabolism.

The nucleic acids, thus, have more than just chemical components; they have energetic, vibrational and photon activity. This allows the cell to fight against entropy or noise coming into the system. As time passes by, the system gradually loses its fight with the noise. Then the inner cell regulation factors break down, lose their quantic indeterminacy, and fall into determinate Gaussian statistics, and thus, entropy.
Every cell has its unique life span, and if we study the different types of cells, we can see that some of the higher-longevity cells have factors built inside that help to fight against this shift toward thermodynamics.

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To fight against aging we must stimulate intercellular quantic communication

Modern science has found that photon emission from radio waves, television, and from all sources does not follow a simple two-dimensional modality.

It is the fallacy of the educational process that is displayed in two-dimensional books that we think of the photons as just moving through a two-dimensional plane, up and down, left and right. Actually these photons will rotate through a vibrational spiral that includes all four dimensions, including time, and as we understand more and more about other dimensions, we will find that the photons react through the full scan of four-dimensional activity (if not more).

The point here is that these photons do have a three-dimensional spin that is as a clockwise or counterclockwise activity; in other words, if the photon were fired directly at us and we could see the photon approaching, we would notice that the photon is rotating around its indeterminacy axis, clockwise and counterclockwise as it approaches.

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Clock developers through time have developed a universal idea of clockwise vs. counterclockwise. Many ancient traditions talked about walking to the right vs. walking to the left; that walking to the left was bad, and walking to the right was good.

The science of chirality is based on the idea of handedness, and rotations right or left.

It is the speculative point offered by this author that perhaps some ingrained flow of time
in the body that might happen through some of the vortices, which some religions have called chakra, allows for part of this timing action.

Twenty years ago, prior to the writing of this treatise, this author developed a meditative technique in which I would go into a meditative state and rotate the chakras counterclockwise instead of clockwise, with the idea of reversing the aging process. The effects have been profound, as the technique has worked on all the people who have been taught to use it. What we are doing, in effect, is trying to fool the body and reset the clock to run backwards; thus the irreversibility factors inside the body are slowed. Aging doesn't stop, because there are other factors inside the body, which are deteriorating, but aging does slow with this simple technique. We even made a multi media presentation to take us through this meditative state; this tape should be played once or twice a day to help reprogram the clock to go backward.

There are many other factors in the aging process, which we may discuss. Royal Lee and other researchers on the idea of the accumulation of protomorphogens have postulated one such intriguing theory. In his book on "Protomorphology", Royal Lee recounts several experiments that would be very good to review at this time. He found that as organisms age, there is an accumulation of protomorphogens, which are a type of DNA/RNA complex given off by the cells, kind of like a cellular virus which gets into interstitial fluids and starts to attribute to aging. These are rather large complex molecules, seemingly inert inside the system. Accumulations of these protomorphogens can be determined with a simple blood test of the sediment rate of the blood. This sedimentation rate will tell us a lot of different things. Royal Lee found that a high sed rate would be indicative of a possibility of high protomorphogens.

In Royal Lee's literature he found that media containing bacteria for some periods of time would accumulate these protomorphogens, and if they took the bacteria out of the media and tried to re-culture new bacteria, it would be difficult, because of the protomorphogen factors. Bacteriologists have found a very strange phenomenon, coupled with the idea of protomorphology and bacterial growth. The total yield of bacteria per unit volume tends to have
a constant for every given bacterial species. Bail called this population of bacteria the "M concentration".

Organisms may be removed by centrifugation at the M concentration, where growth in multiplication is ceased, and a new inoculum was followed by growth and reproduction. As the bacteria exhaust their food and produce inhibition, the total toxic products cannot be found as the causative factors for the M concentration. Although one may point to a plausible minimal concentration of food per unit on the surface, volume of the organism needed for growth and reproduction, this would lead one to an idea of a geographical space needed by the bacteria in order to reproduce and metabolize.

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Isaacs offers a quantic explanation for this M concentration factor, whereas Royal Lee offers the idea of protomorphology, meaning the release of the excess protomorphogens into the bacterial substrate.

Perhaps the phenomenon of geographical space is also exerted by bacteria. As we find in certain human beings that there is a sense of the amount of space needed to be comfortable, and even in deprivation of the major senses, there still seems to be some geographical sense that allows us to pick up and ascertain when people are in our space.

Isaacs, in the book on "Complementarity", outlines how gram stain and its importance to bacteriology could also be explained in terms of quantic phenomena. This gram reaction may involve the peculiar morphological organization, rather than a true staining.

The specificity and directionality of the gram staining might be explained via the quantic action more readily than any other factor, just as the reactivity of certain hormones can be challenged.

We will find that the lock-and-key idea of chemistry, which is pronounced in pharmacology, does not explain why the lock finds the key. The pursuing of this on a random, entropic, thermodynamic basis would mean that the small amount of hormone needed to make these changes could not possibly have this reaction. Nature would have to work on some other phenomenon including the long-range force to explain this type of interaction. Thousands of examples of biological processes have too small a number to be examined through entropy chemistry. Present-day pharmacology, however, could work on the lock and key precept,
because it overdoes by entering large amounts of a chemical into the system to demand action. The large amounts of this chemical will upset many of the sensitive cybernetic biological controls.

So here again we see some of the fallacy of present-day pharmacological theories, and also why there are billions of dollars each year sought in pharmacological iatrogenic malpractice suits.

Natural hormones act as if they have eyes, and indeed they might, because they might work on a photon basis.

Contrasting Philosophy on Natural Aging defense
Anti-aging and quantum theory

INFLAMMATION

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Recently, in a bar room discussion with an allopathic medical doctor, we tried to discuss just how the white blood cell would find its targeted query, bacteria or fungus, to destroy. I pointed out that for him to find the bathroom he would need his photon receptors, his eyes, to be able to ascertain the photons and home in on the destination. He needs photons to pursue and arrive at such a destination. This, as we point out, is a similar phenomenon to the white blood cell and how it finds its query, on the basis of photons, and how the different types of chemicals and hormones find things on the basis of photons.
It is the natural photon factor, which will help us to understand more of biology in the future. As we do, we will find that the natural photon factor is much more present in natural phenomena than it is in any type of synthetic. Nature indeed is of light, and light is the key to all nature.

The lock-and-key phenomenon and the bathroom, as discussed with our friend the medical doctor, is a good example of how nature has provided him with a photonic receptor ability, vision, to find the bathroom. In such a process one key can find its lock, if there is a "seek" function in a photon receptor process. Sinthetically we could overwhelm the bathroom by putting in a hundred such medical doctors with their keys, looking for the lock, and then of course one will find the lock haphazardly, as with entropic and thermodynamic functioning. This is what is happening in pharmacology.

Where in the synaptic cleft there is a lock-and-key phenomenon, nature has a process wherein the key pursues the lock and homes in on it in a natural way. Synthetic pharmacology plots to put a thousand or more keys into the situation, hoping that one of them will bump into the lock. The presence of these excess keys turns off, or on, many other processes that should not be turned off or on. However, with the focus on the symptom, and not the wholeism, we could see the great success that could be achieved, and the amount of money that a synthetic pharmaceutical complex could earn.

To experimentally define this concept, we would have to see if the hormones are photoreceptive, and also if they are photo-productive, and if there is a difference in their receptor or production ability vs. synthetic or naturally made hormones. To answer this question, we simply need a photon counter.
The one used in this study was the Thorn EMI counter, using an infrared tube, so that we can count photons through the infrared and visible light spectra. In both cases the photon receptor tube was set at a base line of photons to compensate for the photon bath of room temperature.

SIMILAR RESULTS ON ALL HORMONAL TESTS As we can see from the diagram, the natural hormones out-produced the synthetic hormones by almost twenty to one in their production of photons.

In our second test with tissue culture, we can see that the production of photons is dramatically increased by the administration of a natural hormone vs. a synthetic hormone, and the production of hormones goes beyond that which is supplied by the natural hormone. Thus we can see direct evidence that there is a dramatic difference in the photonic ability of natural vs. synthetic hormones. The hormones used in this study were androgenic, catecholic and indolic, as shown in the above diagram.

In a rather long treatise, which we cannot do justice to at this time, he came up with the idea that protomorphogens accumulate in between the cells, and the accumulation of these protomorphogen complexes attribute to aging. The body has to provide enzymes to break up these large protein complex molecules. As aging occurs, the taxation of the enzyme production capacities of the body increases, until the body cannot keep up with the production of the enzymes needed.

This couples well with the research done by Dr. Revici. Revici found that there were two counter-proposing variables in the body; anabolic vs. catabolic. He also found that this anabolic and catabolic process had strong correlates to acid and alkaline, and specifically to sterols vs. fatty acids, and thus Revici developed his theory of lipid balancing in the body, and the concept of dualism. A review of the Revici literature is well advised at this time.

Revici developed a rather large and extensive treatise on this subject, with many different types of experimental challenges, which he performed to prove his theories. He also developed several different modalities, using different types of alcohols and other lipids to treat conditions that were taken out of balance, and were taken to one side or the other of the dualistic matrix.
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Thus Revici found that shifts in acid and alkaline balance, or anything out of the homeostatic control factors, were ones that could be disease provoking, if we took the natural process of life, and could estimate the perfect balance needed to maintain perfect health. Revici found that at certain crisis points or bifurcation points, as outlined in the Promorphes, could produce an alternative for the body to adapt to a new homeostatic condition or try to return to the old pattern. Patients with acute disease were at the early stages of making a choice, and could be guided back to health a lot easier; whereas patients who had chronically adapted to new patterns of balance, and thus patterns of disease, were harder to stimulate, and perhaps they needed a new bifurcation point, or some type of crisis to help provoke them back; hence, the healing crisis, which can be provoked by using too precise a homeopathic. Here we are faced with a decision as to how much of a crisis our patient can tolerate. But let us leave this topic for now and return to that of aging.

Revici attributed part of aging to an accumulation of bad sterols and the body's inability to make good sterols, or hormonal complexes, as a vast part of the problem. Intake of good fatty acid complexes is a must nutritionally to help balance this delicate process. Fatty acids, as we have shown before, are sensitive to heat, and can be destroyed by cooking. In a world of over processing and over-cooking of our nutritional needs, with less emphasis on vegetables and other fatty acid complexes, we can definitely see the link between nutrition and aging.

**FATTY ACIDS < · Battle of Balance, Homeostasis · > STEROLS**

The body's ability to manufacture these sterol compounds, which are hormones,
deteriorates with age. Many researchers have found that there is a deterioration in advanced aging in the ability to manufacture a quantity of hormone. New research has found that there is severe impairment in the ability to manufacture good hormones in later years. Thus two criteria must be met; the quantity and the quality of the hormonal manufacture of the human body. By alleviating the different causes of disease, as outlined in our disease chapter, we can maximize the production of hormones to the ability of the organism. If we take out the environmental variations and get rid of the causes of disease such as radiation, insecticides, heavy metals, environmental pollutions and the like, we will be able to maximize the body's potential (see Natural Repertory of Dr. Nelson).

Small amounts of these toxins, as we have shown in our discussion of hormesis, can be helpful. However, large amounts can be rather toxic. Compensating with hormones is a rather tricky business, especially if we are going to compensate with synthetic hormones, which do not have any of the full-range ability that our natural hormones do. As we learn more about hormonal treatment with natural types of homeopathic sarcodals, we will approach better ways to find just the right amount to help stimulate natural effects and compensate for some of the losses. Large amounts of hormones can actually stimulate more atrophy of the hormonal manufacture mechanisms. So this is a very subtle process in which we need to find just the right amount needed to stimulate more hormonal production by the organism, and at the same time, compensate in small ways. Perhaps the development of natural compensation techniques and different horary clock or circadian rhythms are where we might compensate for a hormone in the morning, and use an energetic homeopathic of it in the evening. Those types of therapy regimes will have the best effects in the future, in light of our new biology.

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Another profound factor in aging is that of the build-up of certain minerals, the most prolific of which is calcium. Sodium is an extracellular ion that occurs mostly in the extracellular fluids of the body.

![Diagram of potassium in red blood cells](image)

Potassium is an intracellular ion, which occurs inside the cell at a higher percentage. Measuring serum potassium and finding an average of 4.5 is misleading concerning the amount of potassium in the body. Whole blood potassium, as Revici points out, will have a norm of 32. Now we are measuring potassium where it occurs more naturally, within the cells. In whole blood we will use the whole blood cells to determine the amount of potassium, not just the serum, where the cells have actually been separated. Thus, by the Revici system of knowing the whole blood and the serum potassium, we will have a much better idea of just where the potassium is in the body vs. where it belongs. The
norm would be 32 in whole blood, 4.5 in serum. In a case where we might find 6 or 7 in serum, we might find a low value of 22 or 20 in whole blood; this would tell us that the person is actually potassium-deficient inside the cells, where the potassium belongs. But in the medical system, where the serum ratio might be 6 or so, the normal medical doctor would assume that potassium is not the problem; there is plenty of potassium in the system. But the potassium is not where it needs to be. Something has gone wrong with the cellular potassium pump, and the person is actually in a deficient potassium state. Thus we need to know about whole blood sodium as well, to find out if the sodium is really where it should be. Another key balancing act is that involving calcium. Calcium is mostly a membranous mineral that occurs mostly in the membranes of various materials in different cells. Calcium also should be measured with whole blood and serum values to find out its contrasting norm, because when we remove the cells, we remove the membranes of them as well; we will not truly know some of the most needed material about calcium, until we can start measuring calcium, whole blood and serum values.

Calcium is an ion that can have different energetic states of the electrons and protons in it. These imply different energetic capacities. Calcium taken from mineral sources has low energies, as the electrons in the outer states are in tighter quantic states (closer to ground state). The process of photosynthesis and the like is that of elevating these electrons at the higher states; thus there is a higher quantic energy in the calcium taken from a plant or animal source. The idea that calcium is calcium is a fallacy in light of quantum biology. There is a difference between good and bad calcium. Reports released by the AMA, published in 1989 in Reader’s Digest, stated that calcium carbonate, mineral calcium, was found to be a poor source of calcium for the body. This is because it is a low-energy form. Calcium lactate, taken from milk, an animal source, was found to be a much better source of calcium for the body. Here again, modern medicine has tried to over-reduce the factors into just quantity, and does not realize that there are subtle qualitative energy states, subtle abilities of the body to utilize different types of ions. Calcium is used by the body, just as asphalt is used by street crews for fixing roads. When there are problems; potholes, etc., street crews will come by and dump in asphalt to fill them in. Sometimes an over-zealous street crew will put too much asphalt into a spot, and produce a lump in the road. This is basically the type of phenomenon, which is happening in the body, which uses calcium to fix holes within bones and other cellular structures. If too much calcium is put in by an over-zealous body, or if the quality of calcium taken into the body is not right, then, just as if the quality of the asphalt used by the street crew might not be right, the body
might put too much calcium into an area, and thus develop a bone spur, or some other type of calcium accumulation.

As the body ages, calcium accumulates in its different parts and causes lack of flexibility, hardening of the skin and arteries, etc. This gradual accumulation of calcium in the tissues is another hallmark of the aging process.

In 1967 Howard and Associates made some test tube observations regarding the inhibition of the formation of calcium phosphate hydroxyapatite by a polypeptide inhibitor, which is derived from human urine and serum. This is very important in our understanding of the nature of the clinical pathology process of in vitro calcification. These experimenters demonstrated that supersaturated solutions of the calcium and phosphate ions can be prevented from crystallizing by low concentrations of the peptide inhibitor, that no direct involvement can be postulated from peptide blocking of the crystallization sites. Observing the crystallization of the calcium phosphate hydroxyapatite structure by a quantity of peptide, which is sixteen thousand times smaller than the amount of calcium phosphate ions, forces us into the conclusion of how this rather small particle can operate in a quantic way to disrupt the crystallization. This means that it is stepping in at a very small concentration to disrupt the formation of the crystallization and stop the start of the process, operating at a quantic level, rather than at a large, chemical level. They demonstrated that just one nanomole of the peptide inhibitor prevents the binding to cartilage of up to seventeen hundred nanomoles of the calcium ion. Thus the crystallization that might occur with collagen is different from the crystallization of the simple supersaturated calcium and phosphate solutions. The peptide inhibitor can inhibit the crystallization of both.

The collage large molecular sites are a fourth factor, and the system would be more amenable to treatment under long-range forces. The whole matter, when viewed from chemical concepts, seems mysterious and bizarre, and cannot be explained through normal stoichiometric calculations.

This report, taken from the Isaacs research on the inhibition of calcium by different polypeptides, further tells us about how these polypeptides might be declining in their action in the aging process. Calcification is indeed a very crucial procedure in our discussion on aging. Twenty years ago this researcher duplicated several studies on calcium, metabolism and aging. In one such study, a dozen old Norwegian rats, whose life spans were approximately a
year and a half, and which were at approximately a year and four months, were taken, and their coats shaved. The skin of these rats was old, decrepit, wrinkled, and inflexible. Onto their skin was massaged a skin cream containing a high amount of parathyroid hormone. Parathyroid hormone in the body is used to take calcium out of the cells. Thyrocalcitonin is the hormone made in the thyroid, which puts calcium into the bone, and parathyroid hormone is the hormone that takes calcium out of the cells. The skin cream, after being massaged onto the skin of these old rats, was left on, and within six to eight hours, a small bit of dust, or skin residue, exuded from the skin, looking much like the sleep coming out of a person's eyes. Thus the rats were encased in this little bit of residue coming from their skin.

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After twelve hours the residue was brushed away, and lo and behold, the old skin of the rats had been restored to a much more youthful stature. They were very similar to rats who were six months old. After having seen the changes made on the skin of these rats, another very strange phenomenon was found, not only by this researcher who duplicated the research, but by the other researchers from whom the original research was taken. This was quoted in a 1966 issue of Reader's Digest. It was found that not only had the skin of these rats been restored to youthful vigor, but seemingly their entire health had been. Now their life spans doubled, and they lived another year and a half.

Six of the rats in this study, at the end of another year and a half, were readministered the parathyroid cream. Similar results happened; after the dust was removed, their skin was restored to the six-month age, and these six rats lived another year, whereas the other six rats, who just had one administration of the parathyroid hormone, had only doubled their life span. Attempts on the third generation, by taking three of the rats and massaging the parathyroid hormone into their skin, showed little results. After three administrations within the period of two years, we found that six of these rats had tripled the life span of normal rats.

This intriguing research, done at many universities across the country, had profound effects, especially on the FDA, who within two years of the revelation of this research came up with rather strict controls on the sale, utilization, etc. of parathyroid hormone. Extreme care is taken to prevent oral use of parathyroid hormone, as it takes calcium out of the bone. Under the topic of calcium accumulation comes the topic of flexibility. The theory of chiropractic dictates that there are energies flowing through the spine and nervous system, as well as other channels of flow, including acupuncture meridians, chakra points, and other vortices. If the spinal and nervous system should become inflexible, or perhaps subluxated through some small type of pinch (not quite a dislocation, but a subluxation) this would produce a disturbance in the flow, and thus, qualities of disease. Perhaps massage of PHT onto the spine might help.
The development of the oriental arts of yoga, kundalini, and even some of the martial arts, is around the idea of developing the flexibility of the spine on a daily basis, to re-tune the system. If the spine is flexed, and maintained in its flexion for anywhere from half a minute to five minutes, this will help to make the ligaments, cartilage, nervous structures, muscles, etc. more flexible, less prone to injury, and also more conductive of the energy of the body. This is an example of the principle of chiropractic. The spine needs to be bent forward, backward, to the left, to the right, and twisted left and right. This action will help to stimulate flow of the different patterns of the body, to help establish proper hormonal, enzyme, and other life actions, and help the body in its fight against thermodynamics. It is pointed out that simple flexibility also means holding these points; just to touch the toes is not really holding the flexibility. Touching the toes for thirty to sixty seconds would help to build up the flexibility of the spine and joints. So athletes who hold their ankles, with their knees in locked position, and bob up and down, are not developing the same degree of flexibility as the yoga athlete, who is holding the extreme position for thirty to sixty seconds. This helps build the flexibility. The most ancient of cultures have exercises in flexibility, and all attribute these types of exercises to longevity. Other factors in longevity that have existed in ancient literature are factors of good nutrition, of stress reduction, of love, kindness, psychological stability, exercise, using sweat lodges, etc.

An Advanced Treatise in **QUANTUM BIOLOGY**

In Finland and Sweden, where we have probably the longest life spans in the world, we find that the Fins build their saunas before building their houses. Having traveled to the Hunzas tribe in northern Pakistan, we find that these people have long life spans, and they attribute this to the consumption of certain types of herbs, including bee pollen extracts, parsley, and other naturally-occurring herbs, as well as living lives of hard work, good flexibility exercise, and good nutrition.

Another interesting point developed in the concept of aging comes back to our theory of photons; there are certain photons and photic ranges toward the UV range that have been found to accelerate aging, making skin more dry, hard, and inflexible. These things can be found in sunlight, and people who abuse their skin with excess sunlight have been found to age much more quickly. The part of the body that ages the least is often the buttocks, which are exposed to the least amount of sunshine.

In the Old Testament, Methuselah, Adam and many others lived past nine hundred years of age, Methuselah making it to almost one thousand years. It can be supposed that there was a faulty calendar, and that they probably thought that one year was, for example, four months by our calendar. If that were the case, and they thought that every four months was a year, then Methuselah lived to three hundred fifty years by our calendar. If they were off tremendously,
and said that a year was every third month, than he lived over three hundred thirty years. They would have to have made the mistake of thinking that every month was a year, and thus Methuselah would have lived to approximately ninety years old. But in recounting the literature of the time, we see no evidence to that effect. Perhaps there was another variable. Scientists have found many evidences of a global flood. Prior to this flood, they have found that the plant evidence in the fossils and in the plant material recovered, there was some type of difference. These plants seemed to have grown in a different type of light, vs. plants that were discovered in earth strata after the flood. Several scientists have speculated that there was perhaps an extra large amount of water vapor or the like in the Earth's atmosphere prior to the flooding, and this extra large layer of water vapor provided extra protection against infiltration of these harmful UV waves.

This protection could have greatly enhanced longevity. In light of this, we must realize now, as our ozone layer seems to be depleting due to the mistakes of synthetic chemical companies and an over-aggressive polluting economy, that we have destroyed certain amounts of our own protection complexes, and now modern medicine is suggesting the utilization of sun screens and other types of protection to be used daily to protect our skin from aging. This protects the organism from aging. There are also psychological tendencies toward aging, and programmed ideations of what the natural aging picture is like; how we should be, and how we should age. So in developing an anti-aging regime, there are several factors we must consider, including developing enzymatic therapies for protomorphology, calcium therapies, flexibility training, psychological reprogramming of ideations through suggestivity, as well as retraining the direction of our vibration from clockwise to counterclockwise; nutritional techniques, exercise techniques, removing the causes of disease and aging, hormonal techniques, protection from radiations and environmental pollutions, improving the immune system, and other factors. These are all things that must be utilized in our development of a total anti-aging therapy regime.

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ANTI-AGING REGIME
1. Protomorphology Balancing 5. Nutrition Enzyme Therapy
Anti-aging and quantum theory

6. Cardiovascular
2. Calcium Regulation
7. Detox (Xenobiotics) Hormesis
3. Flexibility Training
8. Ultra Violet Radiation
4. Psychological Protection
9. Immune System Fortification

After twenty years of research by this author, we believe that the human life span can be increased to two hundred years without measurable loss of functioning. To do this, the intervention must start at some of the early ages; forty and fifty, rather than waiting until the age of ninety, where there is extremely crippled skin and utilization, and some irreversibility of the process.

This researcher has developed several homeopathic techniques to reverse the aging process and recycle and reconstruct the direction of the aging mechanism. In this document I have attempted to merely include some of the bare tips of the iceberg of the research we have done on aging. I do not want the reader to think that the sparse pages concerning aging are indeed a true, total thesis. Twenty years of research have gone into the development of our different therapeutic regimes, and how they are used in many different ways.

THE GREAT SUBSPACE ATTRACTOR OF LIFE
(What sets the Limits of Biology?)

In Quantum Biology we discuss the possibility of attractors in certain energetic or cybernetic systems which create stability by attracting the subtle energies of the system towards balance. As items are attracted towards balance, and often have subtle controls so that they can be in the plus or minus, they will be involved in producing what is known as a torus in fractal geometry. A simple example would be the thermostat in your house, which, if set at 70 °, will often warm the room to 71 ° or 72 ° before it turns off. The room must cool down to 69 ° or 68 ° before the thermostat turns back on. Thus the thermostat is set at 70 °, but sets a torus of a range between 68 ° and 72 °. Often a person in the room might feel cold at 68 ° and thus turn the thermostat up; whereas if he would be patient, and wait an extra minute or two, the thermostat would likely kick on and warm the room to the appropriate temperature. Now that this person has reset the thermostat to a new temperature of 72 °, the room heats up to 74 °, and thus a new torus is developed.

In our room thermostat, if we also have a humidifier that will add humidity if it gets too dry; and a de-humidifier that will take away humidity if it gets too humid; we now have a second torus affecting the room. In addition the room might also have a regulator of ionic forces, so that positive and negative ions could be controlled. If the room gets too many positive ions, the negative ion generator could kick in, until the room gets too saturated with negative ions, whereupon it would turn off. A very sophisticated room might have control factors for oxygen, and help to control the amount of oxygen versus CO₂ in a room. As we can see, every time we add a new torus, there will be interaction with the old tori, making the room more and more...
When we get to the phenomenon of the unicellular organism, we must understand that there must be a minimum of 600 factors that need to be regulated on just a simple cell. Each of these is interactive in its ability to act on the others. This collective effect adds to the general torus of the cell. In the multicellular organism of the body there are thousands, if not millions, of regulatory events known to medical science, and many that are not known. Thus the torus of the human body is a very complicated factor, and every little change made to every regulatory factor has an effect on the rest of the system.

Such an analogy can be applied to the human body, where millions, if not millions of millions, of entities must have subtle thermostat controls. Often when a patient is at the low or high end of a certain range, he might feel uncomfortable, and thus take some type of synthetic pharmaceutical drug that can interfere and create a new torus. Often sickness can merely be a perceived value of the body trying to adapt to some type of environmental situation, such as increased stress and the like. This increased stress might move the patient towards greater susceptibility to colds and flues. If the patient seeks allopathic treatment, he might get antihistamines or acyclovir, or other compounds that might then upset the torus by allopathic control.

The principle of homeopathy is one of using the torus to create balance. Thus when there is a sickness, those symptoms are seen in homeopathy to be not the problem, but a messenger of the modality of cure, so that the subtle thermostats can be balanced.

After looking at the torus of the human body, we must become aware that reductionism of simple values can produce statistical irregularities.

When practitioners of medicine attempt to reduce a group of patients to one value such as blood pressure, temperature, or whatever, as they do in clinical trials, they are totally robbing the patient of the individuality that the true organism presents. In order to do a true statistical study, we would need to know all the modalities involved in our torus, measure them, and find the most subtle activities that these compounds exhibit. Then we would have to study the compounds and how they effect the human body for generations to totally know what the effects of synthetic compounds would be.

Thus the technology outlined in our books on quantum biology will severely challenge the limited technology presented by synthetic chemical companies in their pharmaceutical and allopathic approach.

In our analogy of homeopathy from Quantum Biology, we can see that if a room’s temperature is set at 70 °F, and the room starts to get down to 67 °F or 66 °F and becomes too cold, sometimes the thermostat may be stuck, and need some type of gentle movement. Allopathic intervention would come in with an outside heater, through a pharmacological intervention which would then attempt to heat the body from an outside source. This would upset the cybernetic regulatory mechanisms. Classically homeopathy would either try to find a subtle heater in a minimal dose that would help to gently jog the body thermostat back on, or the homeopath might choose a compound that would produce coldness in the body, use it in a minute dose, and turn on the heater of the body by activating the thermostat. This produces balance in the torus and returns the patient to self-regulated, internal thermostatic and cybernetic control.

Elsewhere in the Promorpheus we analyze various mechanisms of the torus as we have
looked at heat and cold, and how they can set limits on the amount of thermal activity to which the human body can be exposed. This thermal activity can either be in the form of lack (in the case of cold) or excess (in the case of heat). Earlier we compared values of humidity, dryness, ionic activity through wind, and other climate conditions that help to set tori of the healthy range that the human body needs for maximum health.

From the Merck manual we have indices of the normal values of various blood chemistry ranges. We provide them below. Also from the Merck manual, we show the ranges of the types of diseases that may result in disturbances that could produce highs or lows in these ranges.

**PITUITARY AND HYPOTHALAMUS AS PART OF THE GREAT ATTRACTOR**

**CONDITIONS IN WHICH VARIATIONS FROM SELECTED NORMAL CHEMISTRY VALUES MAY OCCUR**

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Increase</th>
<th>Decrease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alanine Aminotransferase (ALT or SGPT)</td>
<td>Hepatitis, cirrhosis, liver metastases, obstructive jaundice, infectious mono, hepatic congestion</td>
<td>Pyridoxine (vitamin B6) deficiency</td>
</tr>
<tr>
<td>Albumin Dehydration, diabetes insipidus</td>
<td>Overhydration, malnutrition, malabsorption, nephrosis, hepatic failure, burns, multiple myeloma, metastatic carcinomas</td>
<td></td>
</tr>
<tr>
<td>Alkaline phosphatase Bone growth, bone metastases, Paget's disease, rickets, healing fracture, hyperparathyroidism, hepatic disease, obstructive jaundice, hepatic metastases, pulmonary infarction, heart failure, pregnancy Pernicious anemia, hypoparathyroidism, hypophosphatemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspartate aminotransferase (AST or SGOT)</td>
<td>Myocardial infarction, heart failure, myocardiitis, pericarditis, myositis, muscular dystrophy, trauma, hepatic disease, pancreatitis, renal infarct, eclampsia, neoplasia, cerebral damage, seizures, hemolysis, alcohol Pyridoxine (vitamin B6) deficiency, terminal stages of liver disease</td>
<td></td>
</tr>
<tr>
<td>Bilirubin</td>
<td>Hepatitis, bile disease, obstructive jaundice, hemolytic anemia, pulmonary infarct, Gilbert's disease, Dubin -Johnson syndrome, neonatal jaundice</td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
<td>Hyperparathyroidism, bone metastases, myeloma, sarcoid, hyperthyroidism, hypervitaminosis D</td>
<td></td>
</tr>
<tr>
<td>Hypoparathyroidism, renal failure, malabsorption pancreatitis, hypoalbuminemia, vitamin D deficiency, overhydration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cholesterol</td>
<td>Hypothyroidism, obstructive jaundice, nephrosis, diabetes mellitus, familial, pancreatitis</td>
<td></td>
</tr>
<tr>
<td>Hyperthyroidism, infection, malnutrition, heart failure, malignancies</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatinine</td>
<td>Renal failure, urinary obstruction, dehydration, hyperthyroidism</td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>Diabetes mellitus, IV glucose, thiazides, corticosteroids, pheochromocytoma, hyperthyroidism, Cushing's syndrome, acromegaly, brain damage, hepatic disease, nephrosis Excess insulin, insulinoma, Addison's disease, myxedema, hepatic, failure, malabsorption</td>
<td></td>
</tr>
<tr>
<td>Lactate dehydrogenase (LDH)</td>
<td>Myocardial infarction, pulmonary infarction, hemo-lytic anemia, pernicious anemia, leukemia, lym-phoma, malignancies, hepatic disease, renal infarction, seizures, cerebral damage, trauma, sprue</td>
<td></td>
</tr>
<tr>
<td>Phosphorus</td>
<td>Renal failure, hypoparathyroidism, diabetic acidosis, acromegaly Hyperparathyroidism, osteomalacia, rickets, Fanconi syndrome, cirrhosis, hypokalemia, excess IV glucose</td>
<td></td>
</tr>
<tr>
<td>Potassium</td>
<td>Hyperkalemic acidosis, cardiac arrhythmia, diabetic acidosis, hypoadrenalism, hereditary hyperkalemia Cirrhosis, malnutrition, vomiting, metabolic alkalosis, diarrhea, nephrosis diuretics, hyperadrenalism, familial periodic paralysis</td>
<td></td>
</tr>
</tbody>
</table>

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Sodium Dehydration, diabetes insipidus, excessive salt ingestion Excess antidiuretic hormone, nephrosis, hypoadrenalism, myxedema, congestive heart failure, diarrhea, vomiting, diabetic acidosis, diuretics

Total protein Multiple myeloma, myxedema, lupus, sarcoidosis, diabetes insipidus, dehydration, collagen disease Burns, cirrhosis, malnutrition, nephrosis, malabsorption, overhydration

Triglyceride Hereditary, nephrosis, cholestasis, pancreatitis, cirrhosis, diabetes mellitus, hepatitis, dietary Malnutrition

Uric acid Gout, renal failure, diuretic therapy, leukemia, lymphoma, polycythemia, acidosis, psoriasis, hypothyroidism, eclampsia, multiple myeloma, pernicious anemia, tissue necrosis, inflammation Uricosuric drugs, allopurinol, Wilson's Disease, large doses of vitamin C

Urea nitrogen Renal disease, dehydration, G.I. bleeding, leukemia, heart failure Hepatic failure, overhydration, pregnancy

**SELECTED CLINICAL LABORATORY TESTS-- REFERENCE VALUES**

**Reference Values for Blood (B), Plasma (P), and Serum (S)**

**Test Normal Adult Range**

**Conventional Units SI Units**

Acetoacetate plus acetone (B) Aldolase (S) Aminotransferase (S) Alanine (ALT, SGPT) Aspartate (AST, SGOT) Ammonia (B) Amylase (S) Ascorbic Acid (B) Bilirubin (S) Direct (Conjugated) Total Blood volume Calcium (S) Ionized Total Carbamazepine (P) CO₂ content (S) CO (B) Carotenoids (S) Cerulosplasmin (S) Chloride (S) Cholesterol (S) CK (S) Female
Male CK isoenzymes (S) Copper (S) Creatinine (S) Digoxin (S) Therapeutic Toxic Ethanol (B) Glucose, fasting (P) Iron (S) Total Binding capacity Lactate (B) Venous Arterial Lactic dehydrogenase (S) Lead (B) Lipase (S) Lithium (S) Therapeutic Toxic Negative 1.0-8.0 u/L 5-30 u/L 5.25 u/L 11-35 · mol/L 60-160 u/dL 0.4-1.5 mg/dL 0.1-0.4 mg/dL 0.3-1.1 mg/dL 8.5-9.0% of body weight (kg) 2.1-2.6 meq/L 4.25-5.25 mg/dL 4.6-5.5 meq/L 9.2-11.0 mg/dL 3.12 · g/mL 24-30 meq/L 0% of total Hb 0.5-3.0 · g/mL 27-37 mg/dL 96-106 meq/L 120-220 mg/dL 10-70 u/L 25-90 u/L 5% MB or less 70-155 · g/dL <1.5 mg/dL 0.8-2.0 ng/mL >2.5 ng/mL Negative 75-105 mg/dL 50-150 · g/dL 250-410 · g/dL 4.5-20 mg/dL 4.5-14.4 mg/dL 50-115 u./L 0-50 · g/dL 0-1.5 u. (Cherry-Crandall) 0.5-1.4 meq/L 2.0 meq/L 16.6-135 nkat/L* 83-500 nkat/L* 83-415 nkat/L* 11-35 · mol/L 111-296 u./L 23-85 · mol/L 1.7-6.8 · mol/L 5.1-19.0 · mol/L 80-85 mL/kg 1.05-1.30 mmol/L 2.3-2.75 mmol/L 12.75-51.0 · mol/L 24-30 mmol/L 0.9-5.6 · mol/L 1.8-2.5 · mol/L 96-106 mmol/L 3.1-5.68 mmol/L 166-1167 nkat/L* 416-1500 nkat/L* 11-24 · mol/L <133 · mol/L 1.0-2.6 mmol/L >3.2 mmol/L 4.2-5.8 mmol/L 9.27 · mol/L 45-73 · mol/L 0.5-2.2 mmol/L 0.5-1.6 mmol/L 833-1917 nkat/L* 0-2.4 · mol/L 0.15 u. (Cherry-Crandall) 0.5-1.4 mmol/L >2.0 mmol/L

SELECTED CLINICAL LABORATORY TESTS--REFERENCE VALUES (Cont’d) Reference Values for Blood (B), Plasma (P), and Serum (S) Test Normal Adult Range

Conventional Units SI Units

Magnesium (S) · Nucleotidase (S) Osmolality (S) Oxygen saturation (B) Arterial Pco₂ (B) pH (B) Pco₂ (B) Phenobarbital (S) Therapeutic Toxic Phenytoin (S) Therapeutic Toxic Phosphatase, acid (S) Phosphatase, alkaline (S) Phosphorus, inorganic (S) Potassium (S) Primidone (S) Therapeutic Toxic Procainamide (S) Therapeutic Toxic Protein (S) Total Albumin Globulin Electrophoresis Globulin · α · β · Pyruvic acid (B) Quinidine (S) Therapeutic Toxic Salicylate (P) Analgesic Anti-inflammatory Toxic Sodium (S) Sulfate (S) Triglycerides (S) Urea nitrogen (S) Uric acid (S) Vitamin A (S) Vitamin D derivatives (S) 1.25 dihydroxy 25 · hydroxy 1.3-2.1 meq/L 1.8-3.0 mg/dL 1-12 u./L

280-295 mOsm/kg serum water 96-100% 35-45 mm Hg 7.35-7.45 75-100 mm Hg 15-50 · g/mL >50 · g/mL 5-20 · g/mL >20 · g/mL 0.2-1.8 IU/L 23-71 IU/L 3-4.5 mg/dL 1-1.5 meq/L 3.5-5.0 meq/L 5-12 · g/mL >15 · g/mL 4-10 · g/mL >16 · g/mL 6-8.0 gm/dL 3.5-5.5 gm/dL 2.0-3.5 gm/dL 0.1-0.4 gm/dL 0.4-1.1 gm/dL 0.5-1.6 gm/dL 0.5-1.4 gm/dL 0.3-0.9 mg/dL 1.2-4.0 · g/mL >10 · g/mL 20-100 · g/mL 150-300 · g/mL >300 · g/mL 135-145 meq/L 2.9-3.5 mg/dl 35-160 mg/dl 8-23 mg/dl 3-7 mg/dL 20-60 · g/dL 20-45 pg/mL 25-40 ng/mL 0.7-1.1 mmol/L 16.6-200 nkat/L* 280-295 mmol/kg serum water 0.96-1.00 4.7-6.0 kPa 7.35-7.45 10.0-13.3 kPa 65-215 · mol/L >215 · mol/L 20-79 · mol/L >79 · mol/L 3.3-30 nkat/L 383-1185 nkat/L 1.0-1.5 mmol/L 3.5-5.0 mmol/L 23-55 · mol/L >69 · mol/L 17-42 · mol/L >68 · mol/L 60-80 gm/L 35-55 gm/L 20-35 gm/L 1-4 gm/L 4-11 gm/L 5-16 gm/L 5-14 gm/L 0.03-0.10 mmol/L 3.7-12.3 · mol/L >30 · mol/L 145-724 · mol/L 1086-2172 · mol/L >2172 · mol/L 135-145 mmol/L 0.3-0.36 · mol/L 0.40-1.81 mmol/L 2.9-8.2 mmol/L 0.18-0.42 mmol/L 0.7-2.1 · mol/L 48-108 pmol/L 62.5-100 nmol/L

SELECTED CLINICAL LABORATORY TESTS--REFERENCE VALUES (Cont’d)

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This is just a subtle representation of some of the primary values the body needs in balancing its blood chemistry. The little thermostat controls often involve hormone or chemical regulation factors that are activated through the endocrine system. But there are many energetic factors that help this endocrine system to regulate these hormonal and other blood values. Through its matrices every cell must maintain the torus needed to produce metabolism and reproduction consistently. In multicellular organisms we need other controls that still have quantic types of channels. Thus the human body would need an endocrine channel and a hypothalamus gland as thermostats to help control the various interactions. The dramatic vastness and interaction of the torus of the human body makes study of the human body for
medical purposes very difficult. Thus we might see that there are many tori that can be used in biology. If these are accumulated in the precept of the human body and looked at in a holistic fashion, we will see that the body responds as a whole; there is one simple great attractor that dictates the healthy ranges of these blood values. If any one of these values becomes too high or too low, the entire organism tries to make responses towards healing. Adaptation is the highest function in biology. Thus it is not a system-by-system response that dictates biology as far as medical health is concerned. In other words, there is a holistic reactivity to the organism in response to any value that becomes out of touch. Deficiency of calcium, potassium, vitamin C, vitamin A, and so on affects every cell of the body.

Modern science with its reductionistic philosophy has refused to accept this basic dictum of holism. In other words, each and every value, high or low, is treated on an individual level by most modern medical thinkers. This system of reductionism has resulted in the development of many synthetic chemicals from their natural or herbal counterparts. People have tried to reduce these herbs and glandulars to their simplest active ingredients via the theories of chemical reductionism. Since these formulas work in short-term reductionistic studies, they are released to society. Perhaps more consideration will be applied in the future.

The theories of chemical reductionism have thus been developed in the system of medicine and doctors' attempts to reduce their patients to simply one or two measurements, rather than looking at the entire reactivity of the organism. The science of homeopathy attempts to deal with the entire holistic organism rather than with simple types of systems. Early homeopaths would spend four hours going over symptoms and signs. Many homeopaths practice reductionism in that they look for certain symptoms, and then often rely on only one homeopathic remedy to restore the patient to balance. Although the existence of one remedy is possible, it is highly unlikely in today's world, considering the weight of toxic activities that have resulted within our environment from the overload of insecticides, petro-chemicals, and other synthetically-made compounds. These compounds have become prevalent on the scene in the last hundred years or so. Many have come within the last couple of years. This has resulted in a series of compounds for which nature has not produced proper channels of handling.

Thus in dealing with the causes and factors of disease, homeopathy, done in a holistic fashion with a complete nutritional and energetic approach (as outlined in Quantum Biology and the RWC Book), can become a very powerful means of bringing patients back to balance and a healthy torus of peak performance. This torus or great attractor system, along with its cybernetic

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9. Immune System Fortification

Reprogramming

After twenty years of research by this author, we believe that the human life span can be increased to two hundred years without measurable loss of functioning. To do this, the intervention must start at some of the early ages; forty and fifty, rather than waiting until the age of ninety, where there is extremely crippled skin and utilization, and some irreversibility of the process.
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SUMMARY
1. ALL ORGANISMS FIGHT AGAINST RANDOM STATISTICAL ENTROPY.
2. AGING IS THE LOSING OF THIS FIGHT. AS WE AGE, ENTROPY ENTERS THE SYSTEM.
3. THERE ARE MANY FACTORS THAT CAN BE USED TO SLOW AGING.
4. AGING RESEARCH WILL HAVE TO CONSIDER QUANTUM BIOLOGY IN THE FUTURE.
5. HOMEOPATHY AND NUTRITION OFFER MUCH TO AGING PREVENTION.

Anti-aging Diet Tips

STARTS With
What NOT To EAT
1. AVOID Synthetic Foods
2. AVOID Hi Glycemic Foods
3. AVOID Processed Foods
4. AVOID White Sugars
5. AVOID Foods Boiled in Oil
6. AVOID Nitrite/Nitrate meat
Anti-aging and quantum theory

Super ANTI-AGING
WHAT TO EAT

1. Eat Natural Foods with little preservatives
2. Eat more fruits, seed products, leafy greens, salads
3. Let Fruit be your Sweetener,
4. Drink ONLY 100% Fruit juice diluted with water
5. Boil foods in WATER, NOT OIL
6. Use fresh, cold processed UNHEATED olive oil, sunflower oil, safflower oil etc.
7. Less Cooking, Use stir fry well washed veggies
8. Foods made with Love and Nature is Blessed Nutrition, Foods made and eaten with Hate and Anger are poisons.
9. Celebrate each meal with friends, family or at least your joyous self. Celebrate
10. Listen to your inner self what to eat, and when to stop, do not eat with your eyes
Harvard Researchers Successfully Reverse Aging in Mice

by Julie M. Rodriguez, 12/23/13

filed under: Animals, News
In a new study published in the journal *Cell*, Harvard researchers claim to have discovered a new compound that can revive old cells in mice and make them act “young” again. The chemical, called NAD, is found naturally in mouse — and human — bodies, but is gradually depleted from cells as they age. By increasing the amount of NAD in tissue from a 2-year-old mouse, the scientists were actually able to “trick” the cells into acting like those from a 6-month-old animal, the equivalent of a 60-year-old human's cells becoming like a 20-year-old's.
It turns out that as mammals age, the levels of NAD drop by as much as 50%, disrupting communication between cells and their internal energy source, an organelle called the mitochondria. While it's possible that large enough amounts of NAD could theoretically delay or turn back the clock on aging, it's unlikely that it would be able to help cells become immortal.

Dr. David Sinclair, the lead author of the study, has stated very clearly that this is only a measure that could help people “buy more time,” rather than live indefinitely. Because this is a naturally-occurring chemical in the body, hopefully making it available as a supplement won’t have significant adverse effects.

The next step in his research is to find out exactly how much time this compound could potentially add to the average mouse’s life. He plans to add NAD to the drinking water of lab mice and see if it delays the onset of chronic diseases linked to aging, like cancer, diabetes, inflammation, loss of eyesight, and muscle wasting. If that experiment goes well, the next step would likely be to see if NAD has the same effects on human cells in the lab.
The Telomere in Aging

a Brief Review and Towards an QED SCIO/Eductor Stimulation ANTI AGING Therapy

A we have made a treatise for the construct of biology to of a Quantic nature. Life is organized not random or entropic. Thermodynamic describes the laws of death, whereas Quantum theory more fully describes the construct of life. Aging is a decay of the Quantum organization and an acceleration of thermodynamic entropy. This decay of the organization decay makes cellular metabolism more entropic or random. This is aging.

Aging cells are not clueless about their life span: Recent studies show they have a "clock" that reminds them of passing time to alert them to the Quantic decay. Normal human cells replicate a limited number of times before they reach "replicative senescence" and stop dividing. At this point the cells are still alive, breathing and metabolizing food, sometimes for months, until they die. The "molecular clock" that informs the cell of its limited life span is the telomere, a structure at the end of each chromosome that shortens with each cell division. Research shows the mechanism by which a human cell keeps track of its division, by the length of bits of DNA at the end of the chromosome, and their proximity to specific genes. The telomere is at the end of the DNA molecule and thus is susceptible to decay. This decay is similar to the unraveling of a piece of rope. As an organism ages the telomere unravels and thus some of the Quantic organization is lost.

The unraveling could be treated with an Quantum Energetic Dynamic electrical stimulation as in Nelson Biofeedback. A trivector pulse has been developed for stimulating the reconstruct of the unraveled telomere.

A study reported in Science magazine found that in human cells, as in yeast cells, there exists a "telomere position effect" (TPE). TPE is dependent on telomere length and the position of the gene in relation to the telomere. It enables a cell to keep track of its number of divisions, and provides a way to modify gene expression during the lifetime of the cell. According to Dr. Woodring Wright, a senior co-author of the study with Dr. Jerry Shay and colleagues, the telomere position effect suggests that it can "let a cell know how old it is so that it could change its behavior before it became senescent."

Telomeres, telomerase and aging

The hallmark of aging is a gradual loss of functioning cells in the body. But not all cells age at the same rate, even in the same organ. When tested for their ability to divide, normal cells taken from a particular
organ, such as the skin, are happily dividing. Others are incrementally slowing and dividing at a more gradual pace. And then there are those that have reached cell senescence ("old age") and no longer divide or function. On the whole, as tested in cell culture, normal human cells reach senescence after dividing around 60 to 80 times.

The telomere, p53 and senescence

As there are 46 chromosomes in each cell, each with double strands, there are 92 telomeres that dictate its life span. Cells in most growing human tissues and organs gradually slow in growth, in proportion to the shortening of their telomeres.

The telomere is a kind of molecular cap, made of DNA, that protects the ends of the chromosome from damage. Telomere DNA has over 1000 bases (building blocks), with the sequence TTAGG, that repeats over and over. In order to divide, a normal cell has to replicate all the DNA in its chromosomes. But normal cells have difficulty in copying the last few bases on the telomere. As a result, the telomere shortens with each round of DNA replication and cell division. As a cell ages, the telomere keeps shortening until it reaches a finite length. At that point cells stop dividing. This halt in growth is triggered by a gene called p53 that is activated in response to DNA damage. A telomere that has become too short no longer protects the chromosome from DNA damage. When the damage takes place, p53 responds by stopping cell replication and forcing it into senescence. As a telomere gets too short, the finite cell growth prevents DNA-damaged cell growth that could lead to abnormal cells and to cancer.

Telomerase and longevity

As there are 46 chromosomes in each cell, each with double strands, there are 92 telomeres that dictate its life span. Cells in most growing human tissues and organs gradually slow in growth, in proportion to the shortening of their telomeres. Studies have shown that normal cells from old people lose their ability to divide at a faster rate than cells from the young, and that senescent cells increase in the body, with age.
While telomere shortening provides replicative history—a clock that reminds a cell how many times it has divided and how long it yet has to live—elongation of the telomere adds longevity to a cell. This occurs naturally in cancer cells, where a complex protein called telomerase, which has an enzyme component, helps build up and elongate the telomere with each cell division. This allows the cells to continue growing and become effectively "immortal," the hallmark of cancer cells. If one blocks the action of telomerase in a cancer cell by genetic manipulation, the telomere will begin to shorten with each division, as in normal cells, and the cancer cells will stop dividing and die.

In normal cells that are not germ cells, telomerase is switched off at an early stage of development. Telomeres do not elongate and cells must yield to a fate of a limited number of divisions. If one introduces a telomerase gene into normal cells by genetic manipulation, the cell can extend its life span. This has been shown in several studies, including experiments by a team that included Drs. Wright and Shay.

In these experiments telomerase was introduced into telomerase-negative human retina and foreskin cells. The cells began to express telomerase, as actively as cancer cells. Their telomere elongated, and the cells divided vigorously and did not express a cell marker for senescence (beta galactosidase). Furthermore, the cells showed an increased number of cell divisions and a longer life span, compared to the cells that were not treated with telomerase, whose telomere shortened with each division, leading to senescence. Another important observation was that the introduction of telomerase into the cells and their continuous rapid division and longer life span did not make them cancerous. They remained with a normal appearance and normal number of chromosomes.

**Telomere position effect and gene silencing**

Position effect is a term used to describe an event in which a gene's behavior is affected by its location on the chromosome. The changes in behavior can be expressed in various ways, such as differences in the appearance and functions of cells (phenotype), relay of instructions from the gene, and in doubling time of the dividing cells. Position effects have been reported in insects, plants, yeast and mice, and more recently in human cells.

**TPE in yeast cells**

In 1990, Gottschling and colleagues showed in yeast cells that by inserting a gene next to a telomere, it was silenced. The experiments used marker gene ADE2 that produces changes in the color of colonies, depending on whether the gene is expressed (white colonies) or silenced (red colonies). Insertion of ADE2 next to the telomere produced red colonies, (silenced gene). But the red cell colonies had sectors of white colonies, showing the gene was switched back on. Within the white sectors, in the largely red colonies, red sectors appeared. This shows gene reversal; the ADE2 gene was first silenced (red colony), then switched on (white sector), and then silenced again (red within white). The switches may be due in part to neighboring genes influencing the ADE2 gene. This means that while silencing depends on the
gene's proximity to the telomere, competing regulatory factors produced by neighboring genes may modify a gene's behavior.

**TPE in human cells**

The findings that TPE exists in human cells is novel; they show a similarity between TPE in human cells and yeast, and offer clues to cellular aging. In the experiments reported in Science, investigators used a human cancer cell line called HeLa to investigate TPE and the relation between gene activity and telomere length. HeLa cells, which are "immortal," contain telomerase that lengthens the telomere, enabling the cells to keep dividing.

In the experiments, investigators introduced into the cell a gene called luciferase (the gene that makes fire flies glow), linked to DNA. Luciferase, called a reporter gene whose location is identified in the cell by its luminescence, was inserted near a telomere. Its luminescence compared to that of the reporter inserted at internal sites of the chromosome. To test if telomere length influences gene silencing, the investigators then elongated the telomere by telomerase, and examined telomere positional effect on luciferase.

The results showed that luciferase near the telomere produced 10 times less luminescence than luciferase located at internal sites in the chromosome. Increasing the length of the telomere further increased TPE, resulting in an additional two- to 10-fold decrease in luminescence. These experiments showed that the proximity of a telomere to a gene silences the gene: when the telomere is lengthened, and the gene is located further away from the critical end of the telomere, it is silenced even more.

**Telomere position effects and cellular aging**

Telomere position effect sheds light on the role of telomere in cellular aging. According to a simple and older telomere hypothesis of cellular aging, senescent cells have lost an essential gene that allows them to divide. By contrast, immortal cells, including cancer cells, have avoided this loss because they have regained telomerase activity. They continue to maintain their telomeres and press on with cell division.

The existence of telomere positioning effect in human cells offers a different scenario, where there is no need for the loss of a gene to push cells into senescence. It is speculated that, for example, when the cell is young and the telomere long, TPE silences "aging genes" that are located near the telomere, but far away from its end. As the cell divides and the telomere shortens, an "aging gene" would be more affected by its position on the telomere, as it increases its proximity to the end of the telomere. In an old cell where the telomere has shortened to its final length, the "aging genes" are no longer repressed. Silencing is switched off and the "aging genes" activated.

According to Drs. Shay, Woodring and their colleagues, J. Bauer and Dr. Ying Zou, once TPE has been discovered in human cells, there will be a challenge: to identify genes on chromosomes "whose
expression is influenced by telomere length, in order to determine whether TPE actually influences the physiology of aging or cancer."

It is known that certain proteins (gene products), affect cell behavior in different ways, depending on the age of the cell. The genes that regulate these proteins may be important for programming pre-senescent changes in a cell, before telomeres reach their final length.

Take, for example, a cell that needs to alter its energy metabolism to allow for changes in old age. TPE, which keeps track of the "aging gene" in relation to telomere length, will cause mobilization of regulatory genes to help make the needed change before the telomere is too short.

**Telomere, telomerase and age related disease**

Cellular aging contributes to many conditions in the elderly. The skin wrinkles through loss of collagen production by skin cells that have lost function, partly through free radical damage to DNA (sun damage), and senescence. Atherosclerosis is caused by a loss of division-capacity in cells that line blood vessels (endothelial cells). This, in turn, results in overloading of cell factors that increase the risk of atherosclerotic plaques and blood clots. Active cell division is also important in response to injury. For instance, a damaged liver resulting from excess alcohol intake can lead to liver cirrhosis. In this condition, rapid cell division of the normal healthy liver cells, in response to the injury, could replace damaged tissue by supplying functioning liver cells. The shortening of telomeres, however, would limit liver cell replication and prevent tissue renewal. Introducing telomerase into the dividing liver cells, to elongate the telomere, would exert TPE and a silencing of the "aging gene," allowing continuous division that may offer treatment. This was shown experimentally, in a mouse model of chronic liver injury, where inserting the telomerase gene into the injured liver of the mouse prevented cirrhosis.

**Possible therapies**

It is thought that in normal human organs with a capacity for cell replacement, the telomere clock allows enough divisions for normal growth, repair and maintenance. This setting point is not enough, however, to enable additional cell replications needed during chronic disease. Under these conditions, a potential remedy may be found by increasing the life span of tissue cells, by telomerase. Another possibility may involve QED electrical stimulation of cells from an individual, thus extending the life span of the cells in vitro by telomerase, and further re-introducing the QED stimulus into the sardocal trivector signal of the organ that requires help.

The discovery of TPE trivector pattern in human cells provides a mechanism to silence critical genes and change the pattern of cell behavior.

The unraveling of the telomere could be treated with an Quantum Energetic Dynamic electrical stimulation as in Nelson Biofeedback. A trivector pulse has been developed for stimulating the reconstruct of the unraveled telomere.
This finding may lead to further research that uncovers the secrets of cellular aging.

**Clearance of p16\(^{\text{Ink4a}}\)-positive senescent cells delays ageing-associated disorders**

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Nature 479, 232–236 (10 November 2011) | doi:10.1038/nature10600
Received 08 May 2011 | Accepted 30 September 2011 | Published online 02 November 2011

Advanced age is the main risk factor for most chronic diseases and functional deficits in humans, but the fundamental mechanisms that drive ageing remain largely unknown, impeding the development of interventions that might delay or prevent age-related disorders and maximize healthy lifespan. Cellular senescence, which halts the proliferation of damaged or dysfunctional cells, is an important mechanism to constrain the malignant progression of tumour cells\(^1,2\). Senescent cells accumulate in various tissues and organs with ageing\(^3\) and have been hypothesized to disrupt tissue structure and function because of the components they secrete\(^4,5\). However, whether senescent cells are causally implicated in age-related dysfunction and whether their removal is beneficial has remained unknown.

To address these fundamental questions, we made use of a biomarker for senescence, \(p16^{\text{Ink4a}}\), to design a novel transgene, \(\text{INK-ATTAC}\), for inducible elimination of \(p16^{\text{Ink4a}}\)-positive senescent cells upon administration of a drug. Here we show that in the EubR1 progeroid mouse background, \(\text{INK-ATTAC}\) removes \(p16^{\text{Ink4a}}\)-positive senescent cells upon drug treatment. In tissues—such as adipose tissue, skeletal muscle and eye—in which \(p16^{\text{Ink4a}}\) contributes to the acquisition of age-related pathologies, life-long removal of \(p16^{\text{Ink4a}}\)-expressing cells delayed onset of these phenotypes. Furthermore, late-life clearance attenuated progression of already established age-related disorders. These data indicate that cellular senescence is causally implicated in generating age-related phenotypes and that removal of senescent cells can prevent or delay tissue dysfunction and extend healthspan.

**Subject terms:** Cell biology • Organismal biology • Physiology • Health and medicine
Low Vitamin D and High Parathyroid Hormone Levels as Determinants of Loss of Muscle Strength and Muscle Mass (Sarcopenia): The Longitudinal Aging Study Amsterdam

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Received: April 08, 2003
Accepted: September 02, 2003
Published Online: July 02, 2013

Abstract

The age-related change in hormone concentrations has been hypothesized to play a role in the loss of muscle mass and muscle strength with aging, also called sarcopenia. The aim of this prospective study was to investigate whether low serum 25-hydroxyvitamin D (25-OHD) and high serum PTH concentration were associated with sarcopenia. In men and women aged 65 yr and older, participants of the Longitudinal Aging Study Amsterdam, grip strength (n = 1008) and appendicular skeletal muscle mass (n = 331, using dual-energy x-ray absorptiometry) were measured in 1995–1996 and after a 3-yr follow-up. Sarcopenia was defined as the lowest sex-specific 15th percentile of the cohort, translating into a loss of grip strength greater than 40% or a loss of muscle mass greater than 3%. After adjustment for physical activity level, season of data collection, serum creatinine concentration, chronic disease, smoking, and body mass index, persons with low (<25 nmol/liter) baseline 25-OHD levels were 2.57 (95% confidence interval 1.40–4.70, based on grip strength) and 2.14 (0.73–6.33, based on muscle mass) times more...
likely to experience sarcopenia, compared with those with high (>50 nmol/liter) levels. High PTH levels (≥4.0 pmol/liter) were associated with an increased risk of sarcopenia, compared with low PTH (<3.0 pmol/liter): odds ratio = 1.71 (1.07–2.73) based on grip strength, odds ratio = 2.35 (1.05–5.28) based on muscle mass. The associations were similar in men and women. The results of this prospective, population-based study show that lower 25-OHD and higher PTH levels increase the risk of sarcopenia in older men and women.


**Age-related changes in the 25-hydroxyvitamin D versus parathyroid hormone relationship suggest a different reason why older adults require more vitamin D.**

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**Author information**

**Abstract**

Vitamin D requirements are thought to vary with age, but there is little comparative evidence for this. One goal in establishing a vitamin D requirement is to avoid secondary hyperparathyroidism. We studied 1741 euthyroid, thyroid clinic outpatients without evidence of calcium abnormalities, ranging in age from 19 to 97 yr, whose serum and urine had been analyzed for calcium, vitamin D, and parathyroid status. We found no effect of age on the 25-hydroxyvitamin D [25(OH)D] concentration associated with specific vitamin D intakes, and there was no relationship between 25(OH)D and 1,25-hydroxyvitamin D [1,25(OH)2D]. In every age group, serum 1,25(OH)2D declined with increasing creatinine (P < 0.001). What changed with age included creatinine, which correlated with 25(OH)D (r = 0.146, P < 0.001) only in the youngest age group (19-50 yr) but not in the older age groups (P > 0.1). Creatinine did not correlate with PTH in the youngest age group, but the relationship became significant as age increased (e.g. for the elderly, r = 0.365, P < 0.001). Linear regression of log PTH vs. log 25(OH)D agreed with the natural shape of the relationship observed with scatterplot smoothing, and this showed no plateau in PTH as 25(OH)D increased. We compared PTH concentrations among age groups, based on 20 nmol/liter increments in 25(OH)D. Mean PTH in adults older than 70 yr was consistently higher than in adults younger than 50 yr (P < 0.05 by ANOVA and Dunnett's t test). PTH levels of the elderly who had 25(OH)D concentrations greater than 100 nmol/liter matched PTH of younger adults having 25(OH)D concentrations near 70 nmol/liter. This study shows that all age groups exhibit a high prevalence of 25(OH)D insufficiency and secondary hyperparathyroidism. Older adults are just as efficient in maintaining 25(OH)D, but they need more vitamin D to produce the higher 25(OH)D concentrations required to overcome the hyperparathyroidism associated with their diminishing renal function.
The Effects of Age and Other Variables on Serum Parathyroid Hormone in Postmenopausal Women Attending an Osteoporosis Center

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Received: September 03, 2003
Accepted: January 06, 2004
Published Online: July 02, 2013

Abstract

It has been known for many years that serum PTH rises with age, and it has been suggested that this rise may contribute to bone loss in postmenopausal women. It has been variously attributed to declining renal function, declining calcium absorption efficiency, and declining serum 25-hydroxyvitamin D [25(OH)D] levels.

We studied the effects of age, weight, renal function, radiocalcium absorption, serum ionized calcium, and serum 25(OH)D on serum PTH levels in 918 postmenopausal women attending an osteoporosis center. On simple linear regression, serum PTH was a positive function of age ($P = 0.003$) and weight ($P < 0.001$) and an inverse function of serum 25(OH)D ($P < 0.001$) and serum ionized calcium ($P = 0.002$). On stepwise regression, serum 25(OH)D was the most significant (negative) determinant of serum PTH, followed in decreasing order of significance by serum ionized calcium (negative) and body weight and age (positive). Serum PTH was not related to radiocalcium absorption. The reciprocal relation between serum PTH and serum 25(OH)D could not be explained by the serum concentration of 1,25-dihydroxyvitamin D, which did not change with age. After adjustment for serum ionized calcium, body weight, and age, the rise in serum PTH appeared to start when serum 25(OH)D fell less than 80 nmol/liter.

http://www.youtube.com/watch?v=sHQ5siDmcOY

Title: Human Growth Hormone Supplementation and Stimulation Therapy for Anti-Aging - Risks and Benefits
Subtitle: Quantum Electro Dynamic Trivector Stimulation SCIO/Eductor as a viable safe, inexpensive + effective process.

When information from research of human growth hormone (hGH) replacement treatment (GHRT) in elderly men showed that treatment increased muscle mass and decreased abdominal fat, immediate public interest was provoked. Since body composition of hGH-treated old men tended to resemble that of younger persons, the data was over-interpreted to suggest that the hormone could restore youth. The press even went as far as to suggest that the hormone could be a "fountain of youth". Especially important for entrepreneurs was the fact that large numbers of the affluent "baby boom" generation were passing beyond middle age, becoming acutely aware of their mortality and were prepared to purchase the "rejuvenating" hormone, no matter the cost. Making this business opportunity even more tangible was the fact that an unlimited supply of virus free hGH had become available through recombinant gene technology several years earlier. Unfortunately, this link with commercialism and the persisting, exaggerated and unfounded claims of efficacy blunted enthusiasm for continued, legitimate investigation of hGH's true value in opposing the maladaptive effects of aging. Sylvester Stalone’s arrest and conviction in Australia last year has brought further attention to the issue of hGH. The lack of true research should not be overlooked to satisfy greed or vanity. There are risks to using any hormone therapy. Most importantly is dependency. You own production of hormone goes down when you supplement the hormone externally. The amount of hormone needed is actually quite small. The body makes what it needs and this is a very small amount. The supplement of external hormone uses vast amounts that can overwhelm the body and disturb regulating consequences. There is a extremely complex fractal environment in the body and upsetting the balance takes a careful consideration. Using the field of Nelson Bio-Quantum Electro Dynamics can stimulate the production of natural hormone through electro-Dynamic means. By stimulating the trivector signature of the hormone into the body it stimulates a balancing of the hormone production. That stimulates the deficient, while attenuating the excess. The regulatory process of the body is a process of electrical cybernetics. There are sensitive regulating processes that are completely electrical and as such can be accessible to an feedback cybernetic stimulus. Since growth hormone neuroendocrine function declines during aging, and since there is documented evidence showing that poor health and lost vitality can be reversed by GHRT in young adults with pathogenic growth hormone deficiency (GHD), considerable debate over its value in aging continues even today. At the heart of this debate is the question of whether it is reasonable to extrapolate positive data on GHRT from GHD to the aging condition. Research has proven
that electrical stimulus can also be successful in stabilizing hGH and preventing aging.

A Historical Outlook on GHRT
Prior to development of recombinant gene technology, GHRT was restricted almost entirely to children with short stature. Once they reached a height that was considered to be within normal range, therapy was discontinued even though it was known that as adults, GHD children became symptomatic with metabolic "insufficiency." The reason for rigid rationing was limited availability since the only source of hGH at the time was pituitaries from human cadavers. Therefore, children were given first consideration for treatment. However, even the limited supply of hGH was lost in 1983 when Creutzfeldt-Jakob virus was found to be transmitted by the cadaver-derived hormone. Because pooling of pituitary extracts was required, it was not possible to adequately reduce the risk for viral contamination and so the FDA withdrew cadaver derived hGH from the market. However, since GHRT provided an important therapeutic treatment for short stature, alternatives to cadaver-derived hGH were immediately sought. To facilitate discovery, the FDA provided orphan drug status for hGH, which made it significantly easier for companies to register their product(s) for sale. An additional economic incentive for discovery and development of a synthetic hGH was its high cost, which was based at least in part from the precedent established by its original production process, i.e. isolation and purification from large numbers of human cadavers. Furthermore, with an increased supply of hGH, the hormone could be used for more clinical indications such as treatment of adult GHD, thereby creating an even more lucrative market. The basis for this expanded market was that short-stature children who stopped receiving hGH or adults who suffered pituitary dysfunction displayed a higher than normal incidence of cardiovascular disease, diabetes, high blood pressure, bone loss, reduced muscle mass, increased adiposity, reduced immune function, etc. Thus, it seemed that hGH was necessary for more than growth, hGH was in fact, a somatotrophin that contributed to total body health and well being throughout life. This concept was supported by the fact that GHRT resolved many of the problems associated with adult GHD. Accordingly, these economic and scientific incentives resulted in the first major success of the biotechnology sector. Genentech marketed Protropin® in 1985, driving its stock value to unprecedented heights and stimulating immediate competition from other companies. Recognizing that Protropin® contained an additional methionyl group as a result of its production process, the Lilly Pharmaceutical Company successfully entered the GH market with its own product, Humatrope®, which had the exact structure of the human hormone. Currently, all commercially available hGH products- except Protropin® have exactly the same structure as the naturally occurring hormone.

Clinical Effects of GHRT in GHD
1. Increased Morbidity and Mortality in GHD
Adults whose pituitary glands produce insufficient hormones of all types suffer increased overall mortality compared to healthy individuals. In many cases the cause of their premature mortality is cardiovascular disease (5,6). Reports of an association between growth hormone deficiency and cardiovascular disorders showed that when hormone replacement therapy did not include hGH, hypopituitary patients suffered death in greater numbers due to heart disease than expected. This risk is not seen in the Electro Dynamic stimulation. In addition to increased mortality due to cardiovascular disease, other maladaptive changes associated with GHD were found to include premature atherosclerosis, altered lipoprotein metabolism, abnormal body composition characterized by increased weight with reduced muscle and increased central adiposity, impaired glucose homeostasis, fibrinolysis and cardiac function, decreased exercise capacity and quality of life. Thus, individuals that lack hGH have higher than normal risks for developing intrinsic diseases that contribute to their premature deaths. Conversely, GHRT reduced mortality rate in GHD patients to the expected number for the general population. This risk is not seen in the Quantum Electro Dynamic stimulation.

2. Cardiovascular Effects
Cardiac output and oxygen consumption decline in adults who undergo surgical removal of their pituitary glands. Furthermore, the structure of their hearts becomes altered, exercise capacity is reduced and pumping action or diastolic/systolic function becomes impaired. On the other hand, GHRT in GHD patients has an anabolic effect on cardiac structures thereby providing beneficial effects on diastolic and systolic functions. Direct effects on the heart include stimulation of the velocity of circumferential fiber contraction as well as increasing the degree to which they shorten. It was also reported that the maximum tension of cardiomyocyte fibers from hearts of rats with GH secreting tumors was increased as was the sensitivity of myofilaments to calcium exposure. These observations suggest that GH has a beneficial effect on cardiac muscle fiber contractility. Another dramatic demonstration GH cardiovascular efficacy was reported as the ability of GHRT to reverse atherosclerotic lesions and other sequelae of heart disease. This effect and benefit is also demonstrated in the Quantum Electro Dynamic stimulation.

3. Renal Effects and Body Hydration
GH administration causes the body to retain sodium which in turn causes an acute increase in extracellular water content and a slower increase in plasma volume. Increased extracellular volume which results from GH therapy could contribute to improved cardiac function by the Starling effect- which states that increased filling of the heart leads to greater output. Thus, the effect on hydration in concert with those positive effects on the cardiovascular system cited above could contribute to the improved exercise capacity seen in GHD patients receiving
4. Hypertension
Although reports on blood pressure in GHD adults are somewhat conflicting, the hormone deficient patients seem to be predisposed to develop hypertension. The main cause for this effect may be central arousal of the sympathetic nervous system, which has been documented in GHD adults using direct recordings from inside neurons. GH administration was reported to improve left ventricular function without changing mass or thickness of the wall in this part of the heart. This finding suggested that GHRT could also directly increase the strength of heart muscle contraction. Furthermore, GHD is associated with reduced concentrations of vascular system nitric oxide. Since nitric oxide is a locally acting or paracrine vasodilator, multiple factors may contribute to hypertension in GHD. In any event, growth hormone reduces total peripheral resistance, lowers blood pressure and increases nitric oxide production, thereby correcting GHD associated hypertension. In addition to relieving hypertension, GHRT was also shown to actually reverse early signs of atherosclerosis in major blood vessels. This effect and benefit is also demonstrated in the Quantum Electro Dynamic stimulation.

5. Effects on Body Composition
In addition to reversing the negative effects of GHD on cardiovascular structure and function, GH replacement opposed maladaptive changes in body composition associated with the disorder. The most striking effects of GHD on body composition involve the adipose tissue, bone and muscle. Numerous well controlled clinical studies demonstrate that fat especially within the abdomen is increased, while lean body mass or muscle is reduced in association with low hGH. These changes as well as demineralization of bone, which is also marked, are reversed by hGH administration. This effect and benefit is also demonstrated in the Quantum Electro Dynamic stimulation.

a. Lean Body, Fat Mass & Distribution
Numerous well controlled clinical studies demonstrated that growth hormone deficiency is associated with increased body fat, particularly in the abdomen, as well as reduced lean body mass. When growth hormone is administered to patients suffering these symptoms, they are incontrovertibly reversed. As early as 1959, hGH was shown to cause lipolysis or fat breakdown in man. This effect results from hydrolysis of triglycerides, stimulation of fatty acid transport from adipose tissue to the liver and by inhibition of free fatty acid re-esterification into triglycerides by adipocytes.
GHD adults have increased waist/hip ratios consistent with elevated visceral fat volume. Evidence that this dynamic pattern of fat distribution is due to GHD comes from the fact that visceral fat mass increases in patients suffering from acromegaly, a disease in which excessive hGH is produced by the body, after
they receive treatment to reduce GH hypersecretion. Conversely, when adults with GHD receive GH treatment, visceral mass and subcutaneous fat is reduced and this effect is preserved or even increased after prolonged therapy. Besides reducing fat mass, growth hormone replacement therapy initially stimulates whole-body protein synthesis which after a few months returns toward baseline with establishment of a new steady state. This anabolic action of GH is manifested as increased lean body cell mass associated with increased volume of visceral organs and muscle. The changes resulting from GH exposure are potentially significant to GHD patients as well as to the frail elderly because of their reduced muscle mass and strength relative to age-matched or younger controls. Muscle endurance as well as isokinetic muscle strength is also reduced in GHD. In contrast, GHRT increases muscle volume and maximum voluntary isometric and isokinetic muscle strength.

b. Bone Mineralization
The anabolic action of GH on bone is demonstrated by delayed bone maturation and short stature in children with GHD. Adults with GHD experience reduced bone mass and density. Histomorphometric bone data from these patients suggest a prolonged reversal phase, delayed coupling or a delay in the mineralization process indicative of low bone turnover. On the other hand GHRT increases bone mass in animals and humans. For example, data from a two year clinical trial with patients suffering adult-onset GHD, bone mineral density (BMD) increased in lumbar spine and proximal femur after GHRT However, it required a treatment period of 18 months to produce the increase in BMD, explaining why trials of shorter duration were unable to demonstrate similar efficacy of hGH on bone. These findings are consistent with the fact that a single bone-remodeling cycle takes approximately 3 to 4 months to complete and underscores the importance of an adequate period of GH replacement to effectively increase BMD. Cortical bone may respond more slowly to GH than trabecular bone as indicated by the fact that treatment for 18 months with GH increased BMD in the lumbar spine and femoral neck but not in the proximal radius. Increases in BMD following GH treatment may result from direct and indirect actions of GH and insulin-like growth factor -1 (IGF-1) on bone. Indirect actions might include GH enhancement of enzyme activity that increases vitamin D3 concentrations and availability, as well as changes in body weight, fat and mean body mass, and the accompanying sense of well being and exercise performance in response to treatment. Also muscle strength which demonstrates a similar pattern as changes in BMD may be positively associated with the bone changes. A major value of GH on BMD is its potential to reduce fracture risk, especially in the hip and lumbar spine since adults with untreated GHD have increased prevalence of vertebral fractures compared to normal controls. Thus, when GH treatment is administered to GHD patients for two years and BMD increases by approximately 2 - 5%, their fracture rates decrease significantly. This efficiency and utility is also substantiated in the Quantum Electro Dynamic stimulation on
6. CNS Effects and Quality of Life
GH receptors are distributed throughout the brain suggesting that this organ may be a site of the hormone's action. While the binding sites in the hypothalamus undoubtedly involve regulation of pituitary GH secretion, those in the other areas such as the hippocampus could modify psychological and other functions. Exogenous hGH can access the brain as indicated by the fact that concentrations of the hormone increase in a dose-related fashion within the cerebrospinal fluid of adults with GHD after GHRT. Receptors that transduce IGF-1 signals are also found in all regions of the human brain and contribute to brain maturation, neural differentiation, neuroprotection and energy metabolism. Human GH that accesses the brain from the periphery may affect local IGF-1 synthesis since its concentrations in cerebrospinal fluid (CSF) increased nearly 50% during GH administration.
Data from animal studies suggest that GH treatment alters monoamine metabolism in the brain including region-dependent changes in dopamine, noradrenaline, serotonin and 5-hydroxyindoleacetic acid concentrations. Similar changes may occur in humans as indicated by the fact that in GHD patients, GH administration was associated with decreased CSF concentrations of homovanillic acid, a dopamine metabolite and increased €-endorphin. Perhaps these changes in brain neurochemistry play an important role in improving psychological well being that has been observed during GH treatment of GHD patients.
That growth hormone deficiency erodes quality of life is suggested by the fact that people who acquired GHD as adults have higher levels of perceived health problems, are less energetic, less physically mobile, more socially isolated, sleep less well, have impaired cognitive function and mood disturbances compared with normal individuals. In general, these individuals complain of being tired, having low energy, lack of initiative and concentration, memory difficulties and irritability. However, when hGH is administered, they report increased vigor, ambition and sense of well being. The beneficial effects of hGH on quality of life have been confirmed in several studies which reported improvement of cognitive functions including memory, less perceived illness and significant psychological improvements in energy levels and mood. Similar results were derived from clinical trials involving over a hundred patients. Again, energy, emotional reaction and social isolation were improved to the extent that they approached levels similar to those in healthy populations and quality of life improved as early as six months after starting hGH treatment.

Basis for GHRT as an "Intervention in Aging"
Important to the topic of using GHRT as an intervention in aging is the resemblance of GHD clinical characteristics and phenotypes to those that accompany aging, as well as the ability of hGH replacement to reverse or normalize at least in part, the maladaptive changes associated with GHD.
Relevant to these relationships is the fact that spontaneous secretion of hGH decreases with advancing age. The incremental decrease in hGH is greatest between the ages of 20 to 40 years, with variable reductions ranging from 15% to as much as 70% at middle age and beyond. The temporal characteristics of endogenous GH secretion were also evaluated by many laboratories using analyses of frequently collected blood samples. These showed that GH secretion occurs in episodes that vary in amplitude throughout the day with the greatest amounts occurring during sleep. Although spontaneous secretion of GH continues during aging, the frequency and amplitude of the episodes progressively decrease and thus, a decline in mean GH concentrations can be measured across the life span. Notably, a profound decline occurs during the third decade of life preceding the onset of maladaptive changes in body composition and the increased risk for intrinsic diseases that are associated with middle age and beyond. Thus it would seem that reduced hGH in aging, as in pathogenic GHD, is causally associated with physiological and psychological decline.

Concerns About GHRT in the Elderly
There are at least 3 areas of concern about using GHRT in the elderly:
1. It may be unsafe and have toxic direct effects.
2. It may cause physiological perturbations that accelerate rather than delay the onset of intrinsic disease and aging itself.
3. It may be ineffective, causing risk and undue expense without offering any tangible benefit.
These risks are not demonstrated in the Quantum Electro Dynamic stimulation.

1. General Safety Concerns
Extensive study of GH replacement in children with GHD resulted in the accumulation of 15-years data showing that recombinant GH has an excellent safety profile. Because the history of treating GHD adults is shorter, there are fewer studies documenting adverse drug reactions in this population. The most common effect noted during early clinical trials was fluid retention which is now recognized as being the result of using too high doses of GH. Because hGH is not yet approved by the FDA for treating age-related decline in body structure and function, correspondingly less information is available on adverse events in normal elderly populations. Furthermore in many studies of GHRT in the elderly, the subjects suffered from diseases such as osteoporosis, or were malnourished or experienced significant weight loss of unknown etiology. Nonetheless, in these small number of studies using dosages of hGH ranging from 0.010 to 0.3 mg./kg. at intervals of a few days to several months, adverse events including hyperglycemia, hypertension, carpal tunnel syndrome, edema, glucose intolerance and hyperinsulinemia were occasionally observed. However, it should be noted that these doses are quite high and not those usually associated with routine GHRT for aging, i.e. 0.002 - 0.005 mg./kg./day.
2. Possible Acceleration of Aging
A few investigators have expressed concern that increased concentrations of GH and IGF-1 associated with GHRT may accelerate the maladaptive changes associated with aging. This view derives mostly from animal studies, which may have little or no relevance to the human condition. However, in one clinical study, men with the highest levels of plasma IGF-1 had a 4.3-fold greater risk for prostate cancer than those with the lowest IGF-1 concentrations. In another study, high concentrations of IGF-1 were associated with increased breast cancer. In contrast, IGF binding protein-3 (IGFBP-3) which reduces tissue exposure to free IGF-1 was inversely correlated to risk such that when the relationship between the two was considered, the predictive value of IGF-1 for cancer was greatly increased, especially for colon malignancies. Whether these relationships are causal or simply correlative must still be determined. These dangers are not displayed in the Quantum Electro Dynamic stimulation.

3. Cost of Treatment and Risk: Benefit
Declining health, increased illness and dependency is a growing social burden of aging. The value of these aging parameters in terms of direct, indirect and intangible costs, including pain or suffering can be significant, so it is reasonable to explore the possibility that GHRT has the potential to reduce such cost by compressing morbidity into the later and perhaps final stages of life. As a result, GHRT has captured the imagination of the general public and of entrepreneurs alike because it has been promoted, especially in the lay literature as being effective in aging, even though actual proof of efficacy has not been forthcoming. Instead, support for GHRT in aging has been extrapolated from dissimilar models, such as adult GHD and from animal studies. The concerned practitioner should recognize that the effects of GHRT in GHD and aging are not the same. The basic difference is that in GHD, GH is therapeutic, i.e., it is administered to relieve changes in specific symptoms that in turn can be objectively measured and evaluated. On the other hand, GH administration to healthy people for the purpose of preventing maladaptive changes and diseases of aging requires negative data to prove efficacy, i.e., the absence of change is the proof. Without formal studies of the outcomes of hGH administration over periods of years, the proof will not be forthcoming.

Conclusion
The purpose of this article was to provide some explanation for employing GHRT as an intervention in aging. A review of both chemical and electrical utility has shown the greater prospect for safety and efficacy with Quantum Electro Dynamic stimulation. Although the basis for its use for that purpose is extrapolated from GHD, there is a continuing craze for hGH administration to healthy people during aging to minimize the ravages of senescence and thereby promote good quality of life until death. The aging population is definitely increasing and without intervention to
minimize the social impact of senescence, the phenomenon could be overwhelming to the health care systems and economies of nations. Perhaps partial relief can be realized by hormone replacement, and specifically by the use of hGH which seems to have potentially positive, global influence over bodily structure and function. Although not as significant as in GHD, GHRT in aging has already been shown to have beneficial effects, at least in the short term. Future studies should evaluate the minimal dosages required to sustain health and vitality without causing side effects or perturbations in bodily function that in the long term might produce the opposite effects from those desired.

Aging and Inflammation

- Causes of Age-Related Inflammation

Chronic systemic inflammation is an underlying cause of many seemingly unrelated, age-related diseases. As humans grow older, systemic inflammation can inflict devastating degenerative effects throughout the body (Ward 1995; McCarty 1999; Brod 2000). This fact is often overlooked by the medical establishment, yet persuasive scientific evidence exists that correcting a chronic inflammatory disorder will enable many of the infirmities of aging to be prevented or reversed.

The pathological consequences of inflammation are well-documented in the medical literature (Willard et al. 1999; Hogan et al. 2001). Regrettably, the dangers of systemic inflammation continue to be ignored, even though proven ways exist to reverse this process. By following specific prevention protocols suggested by the Life Extension Foundation, the inflammatory cascade can be significantly reduced.

The Causes of Age-Related Inflammation

Aging results in an increase of inflammatory cytokines (destructive cell-signaling chemicals) that contribute to the progression of many degenerative diseases (Van der Meide et al. 1996; Licinio et al. 1999). Rheumatoid arthritis is a classic autoimmune disorder in which excess levels of cytokines such as tumor necrosis factor-alpha (TNF-a), interleukin-6 (IL-6), interleukin 1b [IL-1(b)], and/or interleukin-8 (IL-8) are known to cause or contribute to the inflammatory syndrome (Deon et al. 2001).

Chronic inflammation is also involved in diseases as diverse as atherosclerosis, cancer, heart valve dysfunction, obesity, diabetes, congestive heart failure, digestive system diseases, and Alzheimer's disease (Brouqui et al. 1994; Devaux et al. 1997; De Keyser et al. 1998). In aged people with multiple degenerative diseases, the inflammatory marker, C-reactive protein, is often sharply elevated, indicating the presence of an underlying inflammatory disorder (Invitti 2002; Lee et al. 2002; Santoro et al. 2002; Sitzer et al. 2002). When a cytokine blood profile is conducted on people in a weakened condition, an excess level of one or more of the inflammatory cytokines, e.g., TNF-a, IL-6, IL-1(b), or IL-8, is usually found (Santoro et al. 2002). (See the Suggested Reading reference list for additional citations.)
Protecting Against Inflammatory-Related Disease

The New England Journal of Medicine published several studies in the year 2000 showing that the blood indicators of inflammation are strong predictive factors for determining who will suffer a heart attack (Lindahl et al. 2000; Packard et al. 2000; Rader 2000). The January 2001 issue of Life Extension Magazine described these studies and explained how individuals could protect themselves against these inflammatory markers (such as C-reactive protein, homocysteine, and fibrinogen).

A growing consensus among scientists is that common disorders such as atherosclerosis, colon cancer, and Alzheimer's disease are all caused in part by a chronic inflammatory syndrome.

Seemingly unrelated diseases have a common link. People who have multiple degenerative disorders often exhibit excess levels of pro-inflammatory markers in their blood. Here is a partial list of common medical conditions that are associated with chronic inflammation:

<table>
<thead>
<tr>
<th>Disease</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergy</td>
<td>Inflammatory cytokines induce autoimmune reactions</td>
</tr>
<tr>
<td>Alzheimer's</td>
<td>Chronic inflammation destroys brain cells</td>
</tr>
<tr>
<td>Anemia</td>
<td>Inflammatory cytokines attack erythropoietin production</td>
</tr>
<tr>
<td>Aortic valve stenosis</td>
<td>Chronic inflammation damages heart valves</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Inflammatory cytokines destroy joint cartilage and synovial fluid</td>
</tr>
<tr>
<td>Cancer</td>
<td>Chronic inflammation causes many cancers</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>Chronic inflammation contributes to heart muscle wasting</td>
</tr>
<tr>
<td>Fibromyalgia</td>
<td>Inflammatory cytokines are elevated</td>
</tr>
<tr>
<td>Fibrosis</td>
<td>Inflammatory cytokines attack traumatized tissue</td>
</tr>
<tr>
<td>Heart attack</td>
<td>Chronic inflammation contributes to coronary atherosclerosis</td>
</tr>
<tr>
<td>Kidney failure</td>
<td>Inflammatory cytokines restrict circulation and damage nephrons</td>
</tr>
<tr>
<td>Lupus</td>
<td>Inflammatory cytokines induce an autoimmune attack</td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Inflammatory cytokines induce pancreatic cell injury</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>Inflammatory cytokines induce dermatitis</td>
</tr>
<tr>
<td>Stroke</td>
<td>Chronic inflammation promoted thromboembolic events</td>
</tr>
<tr>
<td>Surgical complications</td>
<td>Inflammatory cytokines prevent healing</td>
</tr>
</tbody>
</table>

A critical inflammatory marker is C-reactive protein. This marker indicates an increased risk for destabilized atherosclerotic plaque and abnormal arterial clotting. When arterial plaque becomes destabilized, it can burst open and block the flow of blood through a coronary artery, resulting in an acute heart attack. One of the New England Journal of Medicine studies showed that people with high levels of C-reactive protein were almost three times as likely to die from a heart attack (Ridker et al. 1997).

The Life Extension Foundation long ago advised members to have an annual C-reactive protein blood test to detect systemic inflammation that could increase the risk of heart attack, stroke, cancer and a host of age-related diseases. In fact, on January 28, 2003, the American Heart Association and Centers for Disease Control & Prevention (CDC) jointly endorsed the C-reactive protein test to screen for coronary-artery inflammation to identify those at risk for heart attack.
What Causes Elevated C-reactive Protein?

- Elevated C-Reactive Protein and Interleukin-6 Predict Type II Diabetes

While some doctors are finally catching on to the fact that elevated C-reactive protein increases heart attack and stroke risk, they still know little about its other dangers. Even fewer practicing physicians understand that pro-inflammatory cytokines are an underlying cause of systemic inflammation that is indicated by excess C-reactive protein in the blood.

In an abstract published in the March 6, 2002 issue of the Journal of the American College of Cardiology (JACC), tumor necrosis factor-alpha (TNF-a) levels were measured in a group of people with high blood pressure and a group with normal blood pressure (Verdecchia et al. 2002). The objective of this study was to ascertain if arterial flow mediated dilation was affected by hypertension and chronic inflammation as evidenced by high levels of the pro-inflammatory cytokine TNF-a.

The hypertensive subjects taking anti-hypertensive medications had about the same blood pressure as the healthy test subjects. Arterial flow medicated dilation, however, was significantly impaired in the hypertensives and this group also showed higher levels of TNF-a, indicating persistent inflammation despite blood pressure control. This study showed that even when blood pressure is under control, hypertensives still suffer from continuous damage to the inner lining of the arterial wall (endothelial dysfunction) caused by a chronic inflammatory insult. The doctors who conducted this study concluded by stating:

"Antihypertensive therapy alone may be insufficient to improve endothelial dysfunction in hypertensives with high plasma levels of inflammatory markers. Additional therapy to target inflammation may be necessary to improve endothelial function and to prevent progression of coronary atherosclerosis in high-risk hypertensives with subclinical inflammations."

A sensitive index to evaluate how much endothelial damage is occurring is the measurement of TPA (tissue-type plasminogen activator), a clot-dissolving enzyme found in the blood. This same study showed elevated TPA levels in hypertensives, indicating continued endothelial damage despite blood pressure reduction. These findings indicate that hypertensives should have their blood tested for both TNF-a and TPA to assess how much inner wall (endothelial) arterial damage is occurring (Verdecchia et al. 2002). If TNF-a and/or TPA levels are high, aggressive therapies to suppress the inflammatory cascade should be considered.

Elevated C-Reactive Protein and Interleukin-6 Predict Type II Diabetes

In a study published in the July 18, 2001 issue of the Journal of the American Medical Association, a group from the famous Women's Health Study was evaluated to ascertain what risk factors could predict future development of Type II diabetes (Pradhan et al. 2001). The findings showed that baseline levels of C-reactive protein and interleukin-6 (IL-6) were significantly higher among those who subsequently developed diabetes compared to those who did not.

When comparing the highest versus lowest quartile, women with the higher IL-6 levels were 7.5 times more likely to develop diabetes while those in the higher C-reactive protein ranges were 15.7 times more likely to become diabetic. After adjusting for all other known risk factors, women with the highest IL-6 levels were 2.3 times at greater risk, while those with the highest C-reactive protein levels were 4.2 times more likely to become diabetic. It should be noted that these other diabetic risk factors (such as obesity, estrogen replacement therapy and smoking) all

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sharply increase inflammatory markers in the blood. The doctors who conducted this study concluded by stating:
"Elevated C-reactive protein and IL-6 predict the development of Type II diabetes mellitus. These data support a possible role for inflammation in diabetogenesis."

C-Reactive Protein and IL-6 Predict Death

- **Frailty in Elderly Linked to Inflammation**

It is well established the elevated C-reactive protein, IL-6 and other inflammatory cytokines indicate significantly greater risks of contracting or dying from specific diseases (heart attack, stroke, Alzheimer's disease, etc.).

A group of doctors wanted to ascertain if C-reactive protein and IL-6 could also predict the risks of all-cause mortality. In a study published in the American Journal of Medicine, a sample of 1,293 healthy elderly people were was followed for a period of 4.6 years (Harris et al. 1999). Higher IL-6 levels were associated with a twofold greater risk of death. Higher C-reactive protein was also associated with a greater risk of death, but to a lesser extent than elevated IL-6. Subjects with both high C-reactive protein and IL-6 were 2.6 times more likely to die during follow up than those with low levels of both of these measurements of inflammation. These results were independent of all other mortality risk factors. The doctors concluded by stating:
"These measurements (C-reactive protein and IL-6) may be useful for identification of high-risk subgroups for anti-inflammatory interventions."

Frailty in Elderly Linked to Inflammation

In a study of almost 5,000 elderly people, scientists discovered that frail seniors were more likely to have signs of increased inflammation than their more active counterparts. This study was published in the Archives of Internal Medicine (Walston et al. 2002) and showed that these frail seniors with elevated blood inflammatory markers also tended to show more clotting activity, muscle weakness, fatigue and disability than active elderly people.

Findings from these studies should motivate every health conscious individual to have their blood tested for C-reactive protein. If it is elevated, then the Inflammatory Cytokine Test Panel is highly recommended. Those who suffer from any type of chronic disease may also consider the Inflammatory Cytokine Test Panel in order to identify the specific inflammatory mediator that is causing or contributing to their problem.

Glycation's Role in Inflammation

- **Cooking and Aging Have Similar Biological Properties**

Eating high temperature cooked food is another contributor in the production of inflammatory cytokines. In fact, it has been shown that eating high temperature cooked food leads to the formation of advanced glycation end (AGE) products. Glycation can be described as the binding of a protein molecule to a glucose molecule resulting in the formation of damaged protein structures. Many age-related diseases such as arterial stiffening, cataract and neurological impairment are at least partially attributable to glycation. These destructive glycation reactions render proteins in the body cross-linked and barely functional. As these degraded proteins
accumulate, they cause cells to emit signals that induce the production of inflammatory cytokines.

The glycation process is presently irreversible, though an important study indicates a drug in clinical trials may be partially effective. According to a Proceedings of the National Academy of Sciences study, consuming foods cooked at high temperature accelerates the glycation process, and the subsequent formation of advanced glycation end products.

A more succinct descriptive term for "advanced glycation end products" is "glycotoxin," since "advanced glycation end products" are toxic to the body. We will use the word "glycotoxin" from here on to describe the term "advanced glycation end products."

**Inflammation: Chronic**

**Cooking and Aging Have Similar Biological Properties**
Cooking foods at high temperatures results in a "browning" effect, where sugars and certain oxidized fats react with proteins to form glycotoxins in the food. Normal aging can also be regarded as a slow cooking process, since these same glycotoxins form in the skin, arteries, eye lenses, joints, cartilage, etc. of our body.

The Proceedings of the National Academy of Sciences study shows that consuming foods high in glycotoxins might be responsible for the induction of a low-grade, but chronic state of inflammation. In addition, the glycotoxins in food cooked at high temperatures also promote the formation of glycotoxins in our living tissues. The implication of these findings is profound.

What one eats plays a major role in chronic inflammatory processes. Consuming low glycemic foods prevents the insulin surge that contributes to chronic inflammatory processes. It is also important to avoid over consumption of foods high in arachidonic acid (beef, egg yolk, dairy, etc.).

We now know that eating too much over-cooked food causes an increase in inflammatory cytokines. Since most "junk" foods are cooked at extremely high temperatures, it makes sense to avoid French fries, hamburgers, potato chips, fried food and other snacks. These foods not only contain lots of glycotoxins, they also create other metabolic disorders that can induce degenerative disease.

Consuming at least 1000 mg a day of carnosine, and/or 300 mg of the European drug aminoguanidine can inhibit pathological glycation reactions in the body. Eating high temperature cooked foods also induces the formation of glycotoxins. Avoiding foods cooked at high temperature not only reduces pathological glycation processes, but also prevents the formation of numerous gene-mutating toxins that are known carcinogens.

Food is cooked to destroy bacteria and other pathogens that could cause a serious illness. It is important not to eat undercooked food, but avoiding food unnecessarily cooked at higher temperatures is desirable. Certain foods (like fried foods) have to cook at high temperatures. Health conscious people are increasingly avoiding fried foods because they are associated with many health risks.

With the availability of cytokine blood profile tests, it is now possible to ascertain the underlying cause of chronic inflammatory disease. The appropriate drugs, nutrients, dietary change(s) and/or hormones can then be used to suppress the specific cytokines
(such as IL-6 or TNF-a) that are promoting the inflammatory cascade.

The Detrimental Effects of Sleep Deprivation
On June 22, 2002, researchers at the annual meeting of the Endocrine Society held in San Francisco reported that sleep deprivation markedly increases inflammatory cytokines. This finding helps explain why pain flare-up occurs in response to lack of sleep in a variety of disorders. According to the researchers, even modest sleep restriction adversely affects hormone and cytokine levels. In this carefully controlled study, sleep deprivation caused a 40% to 60% average increase in the inflammatory marker IL-6 in men and women, while men alone showed a 20% to 30% increase in TNF-a. Both IL-6 and TNF are potent pro-inflammatory cytokines that induce systemic inflammation (Vgontzas et al. 1999; Vgontzas et al. 2001).

The study results were presented by Dr. Alexandros Vgontzas, professor of psychiatry at The Pennsylvania State University in Hershey. Dr. Vgontzas stated that the findings indicate that getting a full night's rest of eight hours is not just a nice bonus, but a necessity. He stated that people who are missing even two to three hours of sleep function poorly the next day.

Dr. Vgontzas added that the finding that lack of sleep may stimulate an increase in chronic inflammatory response is worrisome because inflammation has been linked to the most common lethal conditions affecting humans today. Vgontzas warned: "Restriction of sleep a few hours is a major risk for public safety."

This study has significant implications for the treatment of chronic pain and inflammatory disorders. For many, following the recommendations in Life Extension's Insomnia Protocol could provide considerable relief from pain and other disorders by preventing the increase of pro-inflammatory cytokines.

The Dangerous Pro-Inflammatory Cytokines

- Reducing Inflammation
- Lowering Elevated C-Reactive Protein
- Blood Testing
- The Importance of Cytokine Testing
- Pentoxifylline Studies
- When to Avoid PTX and Other Anti-Inflammatories
- Sources of Pentoxifylline
- Diet and Inflammation

The following acronyms represent the most dangerous pro-inflammatory cytokines. Health-conscious persons should become familiar with these terms because excess levels of these cytokines cause or contribute to many diseases states:

- TNF-a tumor necrosis factor-alpha
- IL-6 interleukin-6
- IL-1(b) interleukin-1 beta
Reducing Inflammation

Scientists have identified dietary supplements and prescription drugs that can reduce levels of the pro-inflammatory cytokines. The docosahexaenoic acid (DHA) fraction of fish oil is the best documented supplement to suppress TNF-a, IL-6, IL-1(b), and IL-8 (Jeyarajah et al. 1999; James et al. 2000; Watanabe et al. 2000; Yano et al. 2000). A study on healthy humans and those with rheumatoid disease shows that fish oil suppresses these dangerous cytokines by up to 90% (James et al. 2000).

Other cytokine-lowering supplements are DHEA (Casson et al. 1993), vitamin K (Reddi et al. 1995; Weber 1997), GLA (gamma linolenic acid) (Purasiri et al. 1994), and nettle leaf extract (Teucher et al. 1996). Antioxidants, such as vitamin E (Devaraj et al. 2000) and N-acetyl-cysteine (Gosset et al. 1999), may also lower pro-inflammatory cytokines and protect against their toxic effects.

Prescription drugs like Enbrel ($10,000 a year) directly bind to TNF-a and block its interaction with TNF cell surface receptors. Enbrel has demonstrated significant clinical improvement in rheumatoid arthritis patients, as have high-dose fish oil supplements (Kremer 2000). High levels of TNF-a may persist even in people receiving Enbrel drug therapy. Even if Enbrel brings TNF-a down to a safe range, other inflammatory cytokines such as IL-6 and IL-1(b) may continue to wreak havoc throughout the body. High levels of tumor necrosis factor (TNF-a) are destructive to many vital tissues such as joint cartilage (e.g., rheumatoid arthritis) and heart muscle (e.g., congestive heart failure).

Excess IL-6 and other inflammatory cytokines attack bone and promote the formation of fibrinogen that can induce a heart attack or stroke (di Minno et al. 1992). To prevent and treat the multiple diseases of aging, it is critical to keep these destructive immune chemicals (cytokines) in safe ranges.

Methods of Lowering Elevated C-Reactive Protein

Those who are in relative good health, but have elevated C-reactive protein, can try to lower it using a variety of diet modifications, supplements and/or drugs. Supplements such as vitamin E, borage oil, fish oil, DHEA, vitamin K and nettle leaf extract can lower C-reactive protein. Diets low in arachidonic acid, omega-6 fatty acids, saturated fats, high-glycemic food and overcooked food can suppress inflammatory factors in the body.

If diet and supplements fail, drugs such as ibuprofen, aspirin, pentoxifylline or one of the statins (such as Pravachol®) should be tried. If the modified diet, nutrients and/or drugs lower C-reactive protein to below 1.3 (mg/L) of blood, then this is an indication that the underlying inflammatory fire has been extinguished. (The high-sensitivity C-reactive protein blood test is recommended to measure this indicator.)

For those whose blood tests reveal persistently high inflammatory cytokine levels despite taking the supplements mentioned above, a low-cost prescription drug may be of enormous benefit.

The generic name of this low-cost prescription drug is pentoxifylline (PTX); the brand name is Trental. This drug was first used in Europe in 1972 and long ago was removed from patent status (meaning it is not cost-prohibitive). PTX is prescribed to improve...
blood flow properties by decreasing its viscosity. It works by improving red blood cell flexibility, decreasing platelet aggregation, and reducing fibrinogen levels (de la Cruz et al. 1993; Gara 1993; Gaur et al. 1993). PTX has fallen from favor because no drug company has the economic incentive to market it to physicians. PTX is primarily prescribed to patients with peripheral artery disease, although it may have potential efficacy in treating a wide range of diseases relating to chronic inflammation.

Numerous studies show that pentoxifylline (PTX) is a potent inhibitor of TNF-α, IL-1b, IL-6, and other pro-inflammatory cytokines (Neuner et al. 1994; Noel et al. 2000; Pollice et al. 2001; Ventura et al. 2001). Similarly, studies also show that DHA fish oil suppresses these same cytokines (Das 2000; Yano et al. 2000). In people who have a chronic disease involving elevated levels of the inflammatory cytokines, the daily administration of 400-800 mg of PTX and/or 1000-2000 mg of DHA fish oil could be of enormous benefit.

Individuals with chronic disease sometimes find it difficult to suppress C-reactive protein. In these cases, it is important to identify the specific inflammatory cytokines that are responsible for the destructive inflammatory processes that is causing or contributing to the underlying disease state. This enables a custom tailored program to be implemented, and its success measured by suppressing the pro-inflammatory cytokine culprits. For instance, if levels of TNF-a levels are elevated, and natural approaches fail to lower it, the prescription drug Enbrel should be considered.

**Inflammatory Cytokine Blood Testing**

People suffering from chronic disease often have elevated levels of C-reactive protein in their blood. C-reactive protein indicates an inflammatory process is going on in the body, but does not identify the specific pro-inflammatory cytokine that may be the underlying cause.

Testing for pro-inflammatory cytokines has been prohibitively expensive because there has been so little demand for it. The Life Extension Foundation offers an inflammatory cytokine profile at an affordable price. Below is the cytokine panel for this test along with the optimal anti-inflammatory ranges:

<table>
<thead>
<tr>
<th>Pro-Inflammatory Cytokine</th>
<th>Optimal Anti-Inflammatory Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tumor necrosis factor alpha (TNF-a)</td>
<td>Quest: 0-25 pg/mL</td>
</tr>
<tr>
<td>Interleukin-1 beta (IL-1b)</td>
<td>0-150 pg/mL</td>
</tr>
<tr>
<td>Interleukin-6 (IL-6)</td>
<td>2-29 pg/mL</td>
</tr>
<tr>
<td>Interleukin-8 (IL-8)</td>
<td>10-80 pg/mL</td>
</tr>
</tbody>
</table>

**Note:** Quest and LabCorp are blood testing facilities. Other blood testing laboratory methods may have different ranges.

As stated earlier in this chapter, an inexpensive C-reactive protein (high-sensitivity) blood test (CRP-hs) can help reveal if you have systemic inflammation. If your C-reactive protein level is over 1.3 (mg/L), this is an indication that you have an inflammatory event occurring in your body. Those with elevated CRP-hs levels (and who have a disease associated with chronic inflammation) should consider using a supplement protocol and/or prescription drugs known to suppress elevated pro-inflammatory cytokines.
The Importance of Cytokine Testing for Those Suffering From Chronic Illness

There are many chronic disease states that can now be managed by the proper utilization of the Inflammatory Cytokine Blood Panel. If you are elderly, or suffer from any serious disorder, these cytokine tests can enable your doctor to prescribe therapies that specifically target the inflammatory cytokine responsible for your poor state of health.

From a practical standpoint, if you suffer from congestive heart failure, and your levels of TNF-a remain persistently high, you may ask your doctor to prescribe the drug Enbrel®, which specifically counteracts the destructive effects of TNF-a.

If you suffer from cancer and your levels of IL-6 remain persistently high, you may consider high dose DHEA or asking your doctor to prescribe a bisphosphonate drug (such as Zometa® that protects against bone destruction that releases excess IL-6 into the body. Those with prostate, certain types of breast cancer, and other hormonally driven cancer should consider other IL-6 lowering therapies (such as high dose DHA fish oil extract) in lieu of DHEA.

Some cancer and patients display elevated levels of IL-8, which induces cancer cells to express growth factors that fuel their propagation. In hepatitis C, elevated IL-8 signals interferon drug resistance. An IL-8 suppressing therapy will soon be available to Americans (it is already used in Japan).

Those with systemic inflammatory disease often manifest high levels of IL-1b. If diet, the anti-inflammatory supplements (fish oil, borage oil, DHEA, etc.) and cytokine-suppressing drugs (pentoxifylline, 400 mg twice a day) fail to suppress this destructive cytokine, then ask your doctor to prescribe the drug Arava (leflunomide), starting at the low dose of 10 mg a day.

Diet and Inflammation

In addition to toxic cytokines, there are other inflammatory pathways that can be mediated via diet modification. A common problem involves overproduction of pro-inflammatory hormone-like "messengers" (such as prostaglandin E2) and underproduction of anti-inflammatory "messengers" (such as prostaglandin E1 and E3).

The good news is that omega-3 fatty acids found in fish oil help to suppress the formation of undesirable prostaglandin E2 and promote synthesis of beneficial prostaglandin E3 (Kelley et al. 1985; Watanabe et al. 2000). Gamma-linolenic acid (GLA) induces production of the anti-inflammatory prostaglandin E1 (Das et al. 1989; Fan et al. 1997). What you eat can significantly affect whether you have more of the beneficial prostaglandins (E1 and E3) as opposed to the pro-inflammatory prostaglandin E2.

Because prostaglandin E2 is a culprit in inflammation, reducing the consumption of foods that are high in omega-6 fatty acids and increasing the consumption of omega-3 rich foods, such as salmon and other fish, can be beneficial. Limiting foods that convert to arachidonic acid can help reduce inflammation. Arachidonic acid is a precursor to both prostaglandin E2 and the pro-inflammatory cytokine leukotriene B(4) (Brock et al. 1999). Another dietary factor that can lead to high levels of arachidonic acid is the overconsumption of high-glycemic index carbohydrates that cause excess production of insulin (Kreisberg et al. 1983). These quickly digestible foods include fruit juices or rice cakes. Food heavy in polyunsaturated fats or saturated fats can also increase prostaglandin E2.

Additionally, a study of elderly patients with heart disease requiring elective surgery (Tepaske et al. 2001) found that nutritional supplements containing omega-3 polyunsaturated fatty acids (as well as yeast and L-arginine) improved the outlook for high-risk patients when given a minimum of 5 days prior to surgery.
The number of inflammatory-related diseases that could be successfully treated with cytokine-lowering therapy is staggering. PTX and supplements such as fish oil, nettle leaf, DHEA, and vitamin K possess mechanisms of suppressing inflammatory cytokines. Unfortunately, there are no side-by-side comparisons to enable us to categorically state whether PTX or natural agents (such as DHA fish oil) work better.

Foods cooked at high temperatures can produce a browning effect in which glycotoxins are formed from the reaction of sugars and oxidized fats with protein. Glycotoxins may contribute to low-grade chronic inflammation. High glycemic foods may also contribute to the inflammatory process. Dietary modifications to reduce inflammation should include elimination of foods and cooking processes that contribute to a chronic state.

For those who have multiple degenerative diseases, the cytokine profile blood test and the C-reactive protein blood test are highly recommended. This may be done through your own physician or the Life Extension Foundation. If your cytokine test reveals excess levels of cytokines such as TNF-a, IL-1(b), or both, nutritional supplementation, dietary modifications, and low-cost prescription medications such as PTX are advised.

The following supplements are suggested:

- The docosahexaenoic acid (DHA) fraction of fish oil may be the most effective nonprescription supplement to suppress pro-inflammatory cytokines. Gamma-linolenic acid (GLA) is a precursor of PGE1, a potent anti-inflammatory agent. A product called Super EPA/DHA provides 1400 mg of EPA and 1000 mg of DHA in 4 capsules.

- DHEA is a hormone that decreases with age. DHEA has been shown to suppress IL-6, an inflammatory cytokine that often increases as people age. Typical doses of DHEA are 25-50 mg daily, although some people take 100 mg daily. Refer to the DHEA Replacement protocol for suggested blood tests to safely and optimally use DHEA.

- Nettle leaf has been shown to suppress the proinflammatory cytokine TNF-a. Take 1000 mg daily.

- Vitamin E and N-acetyl-cysteine (NAC) are protective antioxidants with anti-inflammatory properties. Vitamin E that contains gamma-tocopherol and tocotrienols provides the most broad-spectrum protection. Take 1 capsule daily of Gamma E Tocopherols with Sesame Lignans and Tocotrienols with Sesame Lignans. NAC is an amino acid with antiviral and liver protectant properties. One 600 mg capsule daily is recommended.

- Vitamin K helps reduce levels of IL-6, a pro-inflammatory messenger. Vitamin K also helps in the treatment of osteoporosis by regulating calcium and promoting bone calcification. One 10 mg capsule daily is recommended for prevention purposes. Do not take vitamin K if you are taking Coumadin or some other type of anticoagulant medicine.

- Consuming at least 1000 mg per day of carnosine and/or 300 mg of the European drug aminoguanidine can inhibit pathological glycation reactions in the body.

An Advanced Treatise in QUANTUM BIOLOGY control factors, is part of a new science of fractal dynamics and chaos theory in which we can now understand how many different systems can meld into different reactivities. The stiffest challenge for biology and medicine is yet to come, as we start to realize that there are many ways of seeing the body, and that nature has many more secrets than those
originally supposed. Mitchell Feigenbaum wrote a landmark paper on "The Qualitative Universality for a Class of Nonlinear Transformations". This was presented to the Journal of Statistical Physics, Vol. 19, #1 in 1978. This set up the major intellectual point that led into fractal and chaotic theories, which have now come to challenge reductionistic and deterministic theories of mathematics.

Statistical entropy and thermal dynamics were thought to be paramount, and to not have indeterministic or chaotic effects. But chaos theory in fractals is proven to have effects on supposedly chaotic events. This paper by Dr. Feigenbaum was one of the first to catalog the bifurcation points and set the criteria for limit cycles in tori.

Dr. Fiegenbaum's paper shows that if we have a function of x which has infinite bifurcation or trauma points, there can be a limit cycle oscillating around the attractor which sets the limits of the torus. The population dynamics, through the universality, will allow the bifurcation points as they approach the limit cycle by a ratio of \( \cdot = 2.5029078750957 \), as a consistent point of departure bringing a dispersion point into the torus which will disrupt the torus cycle.

Feigenbaum found on his pocket calculator that this period building number, 2.5029078750957, was crucial in normal entropic situations in which the bifurcation would exceed that number, and would induce shifts in the cycle which would allow for fractal development. This same number, when applied to a limit cycle, such as that within the torus, could also be used to set the crisis or bifurcation point, in which biology would produce severe diseases. Thus if one of the modalities within the torus of life which produces the stability in a patient's health would exceed this 2.5 deviation from the norm, it would be disruptive, and could present a problem to the organism.

In analyzing the pluses and minuses from the norm of our biological matrices we will see, when a patient exceeds the 2.5 deviation from the norm, that it is life-threatening. We refer now to the article from Dr. Feigenbaum, which is supplied in New Biology.

The type of torus we are discussing also comes akin to Rupert Sheldrake's morphic resonance, in which we can look at various attractions from the field of forms. Plato, in his analysis of the field of forms, outlined another world where the proper form of things existed. We have explored this as a subspace consciousness effect. This form thus created an attractor towards other items in the world as we know it. In the world of forms this type of shape created a kind of perfection that the world as we know it could never quite accomplish; yet, the world we know, in its tendency toward this form, was very much like this system of attractors. If we review Plato's world of forms, we can see that Rupert Sheldrake's idea of morphic resonance, and An Advanced Treatise in QUANTUM BIOLOGY

\( M(n) \) is called a linear manifold if, for every pair of vectors \( u \) and \( v \) is also in \( M \). With every linear combination of \( u \) and \( v \) also in \( M \). It is easily verified that a linear manifold in \( M(n) \) is a subspace in the sense of possessing common properties. Visualize this as a connecting plane through the origin of the first space. A set of independent vectors is called a finite basis for \( M \) if it spans \( M \) so that every vector \( u \) in \( M \) is a linear combination of all the vectors \( u \), where it is easy to show that the numbers \( k \) are uniquely determined.

\[ u = k(1)U(1)+...+k(r)u(r) \]

The manifold spanned by vectors all \( u \) is often noted by the symbols \( M[u] \). It can be proved that every linear manifold \( M \) in \( M(n) \) has a finite basis and that every basis of \( M \) contains
the same number of vectors. This number is called the dimension of M. The vector of M(n) are n-dimensional.

The universe is intertwined with congruent space and subspace.

Every basis v of a linear manifold can be orthonormalized. Meaning it can be replaced by another basis u of the same manifold in such a way that all the u's are an orthonormal set. Divide the first vector set by its norm and set a new vector set. Consecutive norming of the various dimensions is called the Schmidt orthonormalization process. If a set of vectors spans the whole space it is said to be closed. If the zero vector is orthogonal to every vector in the set it is called complete. A complete set of vectors that is orthonormal is a c.o.n.s. (complete orthonormal set).

A linear manifold which is not closed cannot itself be a Hilbert space, since such a space would not be complete. Every infinite dimensional theoretical closed infinite dimensional space satisfies Hilbert space. Every set of subspaces have at least two well defined parameters, their intersection and tier closed sum such that:

a) The interaction is the greatest subspace common to all the spaces,

b) There must be at least one base space dimension, which contains an intersection of all other spaces.

By using Fourier series analysis of Bessel's inequality (a form of Parsavals Equality) and Pythagorean theorem we can relate the spaces. This is an abbreviated form of mathematical analysis of invariant subspace. The flexibility of our variant space will follow similar rules. Hermann Weyl's book on "The Theory of Groups and Quantum Mechanics" by Dover outlines the theoretical aspects.

For our brief purposes we need to

1. recognize the mathematical nature of our theory
2. Recognize the mathematical data of our theory.

The matrix we have supposed can be extrapolated through 10 dimensions to cover the extra dimensions of our existence. The base dimensions we live in and perceive most are but a reflection of the other dimensions that fill the universe. As Isaacs surmised "The human being could be the solution for the universe.". If this is true then in the human genes there would be the mathematical solution for the universe, in the mind would be the potential for solving this solution, and in the hearts and guts there would be the ability to feel the grandeur and appreciate the what God hath done. If the universe is connected by God and we are made in his image then An Advanced Treatise in QUANTUM BIOLOGY

Plato, saw this world of forms as an existing phenomenon that had some place in the heavens. We might see that this world of forms actually could be locked into the small which reflects the large, or the DNA structure. The DNA structure might offer a type of energetic form which could be expounded as the organism grows from one cell into many cells. Thus as the egg and sperm unite and produce another organism, this type of form might be locked into an energetic blueprint. This energetic blueprint, thus, would create the pull and push of the various cells, which then would grow into organs, organ systems, and other bodily structures. This is not a random process, but has a quantic relation, which is the basic treatise of Quantum Biology.

In the movie "Awakenings" we see Robin Williams playing the part of a doctor who has found one type of modality that affects a catatonic patient. He uses dopamine in a large quantity exceeding natural reactions in a patient population that has deficiencies and the inability to handle this dopamine. The large quantity of dopamine that he gives them comes into the cycle and, by interrupting the limits, brings the patient back to normality. Continued use of the large quantity of dopamine without sensitivity to this type of cycle produces dramatic results, which
ends up taking the patient back to the old torus of catatonia. If the doctor could have developed a more sensitive means of adjusting the dosage before the symptoms returned, as well as the sensitivity of the whole organism to the dopamine overload, he could have possibly caused a long-term effect on these patients and brought them back to normality. Thus holism could have been a directed answer to the doctor's dilemma in the movie.

In a system of medicine we must now be able to look for systems of intervention that will help to guide the patient back to the balance of the health torus. Allopathic, in its use of compounds working against the body and often in large quantities, will produce much difficulty, whereas homeopathy might offer plans for a cure. Allopathic often also is ill-equipped to deal with some of the true ideologies and causative factors in disease. These are also mechanisms that need to be alleviated before we can get back to balance.

Thus concepts such as the minimal dose, like treating like, hormonal isodes, nosodes, sarcodes, and other types of homeopathic intervention will offer medicine true curative factors for the future.

In conclusion, we can see that the complexity of biology as presented in this treatise offers us a theory of reverence that must be adopted in our system of medicine. We must respect the natural sensitivity of this process and realize our inability to duplicate it. The system of homeopathy with its proposition of bringing the patient back to balance, and the system of naturopathy, observing nature and using natural modalities, will become the forefront of medicine. We realize that there will be much resistance, as this will threaten many intellectual egos. We can only pray that these egos find the reverence in their hearts to make the change.

The biochemicals discussed have distinct electrical patterns. Each Chemical system has a trivector signature of voltage amperage and resistance profile. This sets up a band of capacitance and inductance bands for each system. These systems act as electron and subspace transport An Advanced Treatise in QUANTUM BIOLOGY chemical (enzyme) will also thus be more specific for each point versus the more general pattern of the chemical pathway.

To measure these patterns we need to first measure the overall electrical pattern of the patient’s reactance to the chemicals. We then must chart the trivector, resonant frequency, and regae field reaction of the chemicals. This includes the resistance, impedance, voltage, amperage, capacitance, inductance, resonant and harmonic frequencies, ph, eh, reactance, polarity, evoked potential, etc. Evoked potential is the reactance pattern of a subject to an applied stimulus. Then we measure the individual reactions of these patients in the context of the individual patterns. Then the specific chemicals can be measured in the same fashion. Attempts to measure just one parameter such as resistance or resonant frequency will be grossly inaccurate. Instead a fractal dynamics of non linear data analysis must be used for the best results. Then thousands of subjects need to be analyzed for pattern similarity. After 12 years of analysis a computer program capable of performing the vast numbers of individual analysis has been developed. (see Int. Jou. of Med Sci of Hom)

The end resulting computer program can now analyze and treat pathways and specific enzymes. Only by systemic analysis of the electrical trivector signature can the patterns be best analyzed. The computer can set up an interactive handshake analysis. A cybernetic link can be established where the computer can treat check and retreat in a consistent loop till the energetic imperfection is abolished, corrected, or till the system refuses to respond. Any more therapy would be unwise. The old style systems where just one way therapies without cybernetic feedback. Simply put this computer can interact during therapy with the patient to adjust the
Anti-aging and quantum theory

therapy for individual needs.
This computer was coined the 'Butterfly system' in an article in 1990 in an English medical Journal. This was because of its' fractal analysis capacity. The theory of chaos fractals is often associated with the butterfly flapping its' wings in Singapore and causing a rain in central park. This fractal system is the key of our computer.
To accomplish this task in just twelve years took tremendous dedication and extreme sacrifice. When others were out enjoying the weekend, I worked on electrical parameter testing. The nonlinear systems of fuzzy mathematical analysis and Fourier dynamics had to be learned and some new systems developed. A trinary system of subspace interaction had to be developed with very little help. A quantic matrix of energetic relations and frequencies had to be posited, tested, refined retested, etc. Clinical experimentation and laboratory analysis have paid off in results with patients.
The fuzzy analysis for the torus was developed to analyze the patient data in such a way so to notice the first sign of disease. Rather than let patients get crisis breakdown of systems, a early warning system needs to be developed. We should try to detect the risk and the earliest sign of a problem rather than wait for heroic synthetic medication and surgery medicine. The nonlinear system of analysis is designed in the Biophysics section.
AGING AS A TREATABLE DISEASE
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as to make a perfect energetic medicine machine. This makes the QXCI device able to correct the aging trend of loss of electro chemical control and enable a daily stabilizing and balancing force to slow the aging process while stimulating restoration of the electrochemical balance. In studies done on 100 mice in Budapest, doctors have found the QXCI capable of extending the life span by a factor of almost 2. Meaning the mice given the QXCI Rx live almost twice as long as those given placebo. The placebo used was the Bicom therapy.
In humans the results have been extremely favorable, Users have reported extreme satisfaction and very good results. There has not been an organized study, however. The sophistication and technology of the QXCI cybernetic loop allows for anti-aging to become a greater possibility for the future. A Quantic field can be developed to cybernetically stabilize and normalize deviancies in the Quantic stability field of the patient. This restores balance and prevents decay. It cannot restore damage already done by aging, but can limit decay. Below are some of the anti-aging compounds used today and the resistance scores shown by the QXCI.
Glucose in high concentrations is toxic to cells and tissues. One aspect of this toxicity is the ability of glucose to form covalent links with proteins, nonenzymatically, and so alter protein structure and function. Such changes may be important in the aging process.

As people age, their cells and tissues change in ways that lead to the body’s decline and death. The cells become less efficient and less able to replace damaged materials. At the same time the tissues stiffen. For example, the lungs and the heart muscle expand less successfully, the blood vessels become increasingly rigid and the ligaments and tendons tighten. Older people are also more likely to develop cataracts, atherosclerosis and cancer, among other disorders.

Few investigators would attribute such diverse effects to a single cause.

Nevertheless, we have discovered that a process long known to discolor and toughen foods may also contribute to age-related impairment of both cells and tissues. That process is the chemical attachment of the sugar glucose to proteins (and, we have found, to nucleic acids) without the aid of enzymes. When enzymes attach glucose to proteins, they do so at a specific site on a
specific molecule for a specific purpose. In contrast, the nonenzymatic process adds glucose haphazardly to any of several sites along any available peptide chain. On the basis of recent in vitro and in vivo studies in our laboratory at Rockefeller University, we propose that this nonenzymatic "glycosylation" of certain proteins in the body triggers a series of chemical reactions that culminate in the formation, and eventual accumulation, of irreversible crosslinks between adjacent protein molecules. If this hypothesis is correct, it would help to explain why various proteins, particularly ones that give structure to tissues and organs, become increasingly cross-linked as people age. Although no one has yet satisfactorily described the origin of all such bridges, many investigators agree that extensive cross-linking of proteins probably contributes to the stiffening and loss of elasticity characteristic of aging tissues. We also propose that the nonenzymatic addition of glucose to nucleic acids may gradually damage DNA.

The steps by which glucose alters proteins have been understood by food chemists for decades, although few biologists recognized until recently that the same steps could take place in the body. The nonenzymatic reactions between glucose and proteins, collectively known as the Maillard or browning reaction, may seem complicated, but they are fairly straightforward compared with many biochemical reactions. They begin when an aldehyde group (CHO) of glucose and an amino group (NHZ) of a protein are attracted to each other. The molecules combine, forming what is called a Schiff base. This combination is unstable and quickly rearranges itself into a stabler, but still reversible, substance known as an Amadori product.

If a protein persists in the body for months or years, some of its Amadori products slowly dehydrate and rearrange themselves yet again-into new glucose-derived structures. These can combine with various kinds of molecules to form irreversible structures we have named advanced glycosylation end products (AGEs). Most AGEs are yellowish brown and fluorescent and have specific spectrophotographic properties. More important for the
body, many are also able to cross-link adjacent proteins. The precise chemical structure of advanced glycosylation end products and of most AcE-derived cross-links is still not known. Nevertheless, some evidence suggests that AcE's are often created by the binding of an Amadori product to glucose or another sugar. Such end products would form bridges to other proteins by binding to available amino groups. In some instances two Amadori products may instead merge, creating an ncE that is also a cross-link. The one glucose-derived cross-link whose chemical structure is known appears to be just such a combination. It is 2-furanyl-4(5)-(2-furanyl)-1H-imidazole, or FFI. First isolated in the laboratory (from a mixture of the amino acid lysine, the protein albumin and glucose), FFI has since been found in the body.

The realization that the browning reaction could occur in-and potentially damage-the body emerged from studies of diabetes, a disease characterized by elevated blood-glucose levels. In the mid-1970's one of us (Cerami) and Ronald J. Koenig examined a report that the blood of diabetic individuals contained higher than normal levels of hemoglobin A1c: a variant of the protein hemoglobin, which is the oxygen-carrying component of red blood cells. Curious about why the levels were elevated, the two investigators attempted to determine the molecule's structure. Hemoglobin A1c is an Amadori product. Moreover, as is true for the amount of Amadori product formed in foods, the amount of hemoglobin A1c formed is influenced by the level of glucose in the blood: when the glucose level is high, the amount of Amadori product is also high. (Workers in our laboratory and elsewhere have since identified more than 20 Amadori proteins in human beings and have consistently found two or three times as much product in people with diabetes as in nondiabetics.)

The hemoglobin findings reveal that glucose, which bathes tissues and cells throughout the body, is not the inert biological molecule most biologists thought it was. Although the sugar does not react while it is in its usual ringlike formation, the ring opens often enough to enable Amadori products and other substances to form. Glucose remains the least reactive sugar in the body, but it has the greatest potential effect on proteins because it is by far the most abundant variety.
The fact that glucose is reactive suggested to Cerami that excess blood glucose in people with uncontrolled diabetes might be more than a marker of the disease. If the sugar could bind nonenzymatically to proteins in the body, he reasoned, excessive amounts could potentially contribute to diabetic complications: the host of disorders, ranging from impaired sensation to kidney failure, that often disable people with diabetes and shorten their life. In particular, it seemed possible that high levels of glucose could lead to an extensive buildup of advanced glycosylation end products on long-lived proteins. The accumulation of acE's in turn might undesirably modify tissues throughout the body.

Such musings soon led to a suspicion that glucose could also play a role in the tissue changes associated with normal aging. The effect of diabetes on many organs and tissues is often described as accelerated aging because several of the complications that strike people with diabetes—including senile cataracts, joint stiffness and atherosclerosis—are identical with disorders that develop in the elderly; they merely develop earlier. If excess glucose does in fact hasten the onset of these ills in people with diabetes, normal amounts could conceivably play a role in the slower onset seen in nondiabetics as they age.

Our laboratory's studies of senescence (which complement our ongoing studies of diabetes) began with an attempt to determine whether advanced glycosylation end products do in fact accumulate on, and form cross-links between, long-lived proteins in the body. Major constituents of the lens of the eye—the crystallin proteins—became the first objects of study because once these proteins are produced they are believed to persist for life; they therefore fit the profile of proteins that could amass advanced glycosylation end products. Also it seemed likely that a buildup of such AcE’s and of AcE-derived crosslinks could help to explain why lenses turn brown and cloudy (that is, develop senile cataracts) as people age. In support of this idea, workers elsewhere had previously found two types of cross-link in aggregates of crystallin proteins from human senile cataracts. One bridge was pigmented, suggesting that it could be an ncE. The other type was a disulfide bond formed between sulfhydryl (SH) groups of the amino acid cysteine.
In test-tube experiments Cerami, Victor J. Stevens and Vincent M. Monnier showed that glucose could produce a cataractlike state in a solution of the proteins. Whereas glucose-free solutions containing crystallins from bovine lenses remained clear, solutions with glucose caused the proteins to form clusters, suggesting that the molecules had become cross-linked. The clusters diffracted light, making the solution opaque. Analysis of the links between the molecules confirmed that both the disulfide and the pigmented types were present. The group has also discovered that the pigmented crosslinks in human senile cataracts have the brownish color and fluorescence characteristic of advanced glycosylation end products. In fact, some crosslinks can be chemically identified as the advanced glycosylation end product FFI.

Combined with other evidence, the above data suggest that nonenzymatic glycosylation of lens crystallins may contribute to cataract formation by a twostep mechanism. Glucose probably alters the conformation of proteins in ways that render previously unexposed sulfhydryl groups susceptible to combination with nearby sulfhydryl groups. Hence disulfide bonds develop, initiating protein aggregation. Later Amadori products on the proteins become rearranged, enabling FFI and other pigmented cross-links to form, discolor the lens and make it cloudy.

Convinced that at least one class of proteins undergoes the browning reaction and forms undesirable cross-links, we and our colleagues turned to the body's most abundant protein: collagen. This longlived extracellular protein glues together the cells of many organs and helps to provide a scaffolding that shapes and supports blood-vessel walls. It also is a major constituent of tendon, skin, cartilage and other connective tissues. In the past 25 years various investigators have shown that collagen builds up in many tissues, becoming increasingly cross-linked and stiff as people age. Studies of the dura mater, the collagen sac separating the brain from the skull, provided early evidence that advanced glycosylation end products could collect on collagen. Monnier, Cerami and the late Robert R. Kohn of Case
Western Reserve University found that the dura mater from elderly individuals and from diabetics displays yellowish brown pigments whose fluorescent and spectrographic properties are similar to those of advanced glycosylation end products formed in the test tube. As would be expected, protein from people with diabetes had accumulated more pigments than the protein of nondiabetics. In nondiabetics the amount of pigment attached to the protein increased linearly with age.

Evidence suggesting that glucose induces collagen not only to form AGE’S but also to become crosslinked comes from several studies. On the basis of work by other investigators, it has long been known that fibers from the tail tendons of older rats take longer to break when they are stretched than fibers from younger animals, indicating that the older fibers are more cross-linked and less flexible. Monnier, Cerami and Kohn therefore attempted to mimic the effects of aging by incubating tendon fibers of young rats with various sugars.

The fibers gradually accumulated advanced glycosylation end products and showed a concomitant increase in breaking time.

More recently we have evaluated the cross-linking of both purified and aortic collagen. In the first instance the protein was incubated with glucose in a test tube; in the second instance it was essentially incubated in the body of diabetic animals that had high blood-glucose levels. In both conditions our chemical tests unequivocally showed that the glucose led to extensive crosslinking.

Although we suspect that the formation of glucose-derived cross-links between long-lived proteins helps to account for many symptoms of aging and for many complications of diabetes, such bridges are not the only ones that can potentially damage the body. We have shown that ncE's on collagen in artery walls and in the basement membrane of capillaries can actually trap a variety of normally short-lived plasma proteins. Even when collagen is incubated with glucose and then washed so that no free glucose is present, the long-lived protein can still covalently bind such molecules as albumin, immunoglobulins and low-density lipoproteins.

This binding may help to explain why both people with diabetes and
the aged are prone to atherosclerosis: a buildup of plaque in arterial walls. The plaque includes smooth-muscle cells, collagen (which is produced by the smooth-muscle cells) and lipoproteins (the cholesterol-rich proteins that are the primary source of fat and cholesterol in atherosclerotic lesions). No one yet understands the exact processes leading to atherosclerosis. It is conceivable that glucose contributes to plaque formation by causing advanced glycosylation end products to develop progressively on collagen in the vessel walls. Once those substances form, collagen may trap low-density lipoproteins from the blood—which in turn can become attachment sites for other lipoproteins.

In theory, glucose-altered collagen could also trap von Willebrand factor, a protein that is believed to promote the aggregation of platelets (sticky bodies involved in blood clotting). The platelets may release a factor that stimulates the proliferation of smooth-muscle cells, which produce extra collagen. Other glucose-related events may further promote plaque formation. More studies are needed to determine the extent to which any of the postulated events take place and how they might interact with various other processes that contribute to atherosclerosis.

Protein trapping and cross-linking may also help to explain the thickening seen in the basement membrane of capillaries as people grow older (and the more rapid thickening in people with diabetes). In people with diabetes, thickening of a specialized basement membrane in the kidney, the mesangial matrix, promotes renal failure. In nondiabetics the consequences of renal basement-membrane thickening are less clear, although we suspect the process may help to decrease the aged kidney's ability to clear wastes from the blood. Elsewhere in the body thickened capillaries become particularly narrow or occluded in the course of time in the lower extremities, where gravity increases the rate of protein trapping by vessel walls. Such narrowing can contribute to the impaired circulation and loss of sensation often found in the feet and legs of both diabetics and older nondiabetics. In order to function properly, the sensory nerves need an adequate supply of blood. Because aging takes place at the level of the cell as well as of tissue,
our laboratory has recently begun to examine the effects of glucose on the material that controls cell activity: the genes. At least in resting cells, the nucleic acid DNA, which contains amino groups, is long-lived. It therefore can potentially accumulate advanced glycosylation end products. These ncE's might then contribute to known age-related increases in chromosomal alterations or to declines in the repair, replication and transcription of DNA. Such genetic changes are believed to impair the body's ability to replace proteins critical to normal cell function and survival. Nonenzymatic glycosylation might also cause mutations that affect the activity of the immune system or lead to some types of cancer.

Richard Bucala, Peter Model and Cerami have found that incubating DNA with glucose does indeed cause fluorescent pigments to form. The pigments do not build up as quickly as they do on proteins, because the amino groups of nucleic acids are significantly less reactive than the amino groups of proteins.

No one has yet investigated the effects of acÉ's on the nucleic acids of mammalian cells, but the group's studies of bacteria suggest that nonenzymatic glycosylation may well interfere with the normal functioning of human genes. When a bacteriophage (a bacterial virus) with a DNA genome was incubated with glucose and then inserted into the bacterium Escherichia coli, the phage's ability to infect E. coli cells was shown to be reduced. The degree of reduction depended on both the incubation time and the concentration of the sugar.

Bucala and his fellow workers also found that adding the amino acid lysine to a mixture of DNA and glucose hastened the loss of viral activity. Presumably the sugar reacted with the amino acid, forming an "ncE-lysine" that quickly bound to the DNA. Because both protein and glucose are present in mammalian cells, it seems likely that a similar reaction might account for the finding that protein covalently attaches to the DNA of aged cells. The effects of such protein binding to genetic material are not known.

Just how the attachment of glucose or a glycosylated protein to DNA interfered with the bacteriophage's normal activity is also not clear. In another
study, though, sugar was shown to cause a mutation in DNA. The workers isolated plasmids (extrachromosomal pieces of bacterial DNA) carrying genes that make E. coli resistant to the antibiotics ampicillin and tetracycline. Then they incubated the plasmid with glucose-6-phosphate, a sugar that reacts more quickly than glucose, returned the DNA to bacterial cells and exposed the cells to an antibiotic. Most of the cells exposed to tetracycline died, whereas most exposed to ampicillin lived. Clearly some of the incubated plasmids kept the ampicillin-resistance gene but had lost the activity of the tetracyclineresistance gene.

Further study showed that most of the tetracycline-resistance genes had been altered by deletions or insertions of DNA. We suspect that those genes had collected advanced glycosylation end products and that the resulting mutations arose when the bacteria attempted to repair the DNA altered by AGE'S. This conclusion is supported by the finding that bacterial cells lacking a DNA-repair enzyme did not have mutations in the DNA.

In order to better determine the effects of advanced glycosylation end products on the DNA of human cells, we are developing new methods for measuring both ncE's and glycosylated proteins on DNA. In addition we need to learn more about the cell's mechanisms for repairing glycosylated nucleic acids.

The ultimate goal of our research into both aging and diabetes is to find ways of preventing or delaying their debilitating effects. If our glycosylation hypothesis is correct, such effects might be mitigated either by preventing the formation of glucose-derived cross-links or by increasing the activity of biological processes that remove ncÉ's.

On the first front we, along with Peter C. Ulrich in our laboratory, have developed a promising drug called aminoguanidine. This small molecule, in the class of compounds called hydrazines, reacts with Amadori products. It apparently binds to carbonyl groups and in so doing prevents the Amadori products from becoming advanced glycosylation end products. In test-tube studies of the drug we incubated albumin either with glucose alone or with glucose and aminoguanidine. Advanced glycosylation and products formed in the first mixture within half a week and increased
progressively with time. In contrast, the aminoguanidine mixture produced an equal amount of Amadori product but resulted in marked inhibition of AGE formation. Similarly, when we incubated collagen with glucose, the protein became extremely cross-linked, whereas the addition of aminoguanidine blocked nearly all glucose-derived intermolecular bridges. Parallel findings come from studies of diabetic rats. Animals treated with aminoguanidine amassed fewer advanced glycosylation and products in the aorta, and fewer cross-links, than untreated rats. In a separate group of diabetic rats we have shown that aminoguanidine prevents both the trapping of immunoglobulin in the basement membrane of renal capillaries and the trapping of plasma lipoproteins in the arterial wall.

We are now planning trials of aminoguanidine in human subjects. If the drug shown to be safe, we hope to conduct long-term trials of its ability to prevent diabetic complications. Because diabetes is in some ways a model of aging, success in such trials might eventually help to justify studying the ability of aminoguanidine (or similar compounds) to prevent disorders related to age in nondiabetics.

We are also studying the other approach to treatment: increasing the activity of the body’s AGE-removal system. Even if the production of advanced glycosylation and products could not be prevented, an effective AGE-removal system might help to counteract any dangerous buildup on proteins. Macrophages, the scavenger cells that remove debris from tissues, apparently constitute one such removal system. This property of the scavenger cells became clear about three years ago when we examined peripheral-nerve myelin: the complex mixture of long-lived proteins that forms an insulting sheath around nerve fibres. We incubated isolated myelin with glucose for eight weeks to mimic the effects of long-term exposure to glucose in the body. Then we introduced macrophages into the mixture. The cells ingested more myelin than they did when the substance had not been exposed to sugar. They also took up more myelin from diabetic animals than from nondiabetic ones, presumably because the diabetic animals had a greater amount of advanced glycosylation end products. More recent evidence indicates that the signal for protein uptake by
macrophages is specifically the advanced glycosylation end product. We have found, for example, that a mouse macrophage has an estimated 150,000 receptors for the AGE=s that form on albumin. Macrophages attempt to ingest any protein attached to the advanced glycosylation end product FFI, but the cells= receptors do not appear to react with any non-AGE substances that accumulate on proteins, including Amadori products.

The affinity of macrophages for FFI, and for advanced glycosylation end products in general, became dramatically apparent when we attached FFI and other ncÉ s to membrane proteins of normal red blood cells. Mouse macrophages took up the altered cells much more avidly than they take up normal cells. (In addition to supporting the contention that macrophages are an ncE-removal system, this discovery suggests that advanced glycosylation end products have at least one constructive role in the body: they may indicate that a cell is aged and should be removed.)

Why do acE's build up on proteins if the body has a system for removing them? We do not have an answer, but a few explanations seem likely. For one thing, the end products may generally form in locations that are not readily accessible to macrophages. Moreover, the highly cross-linked proteins that eventually accumulate appear to be increasingly difficult to remove. Also, as people age, their macrophages may become less efficient as a disposal mechanism. In support of this last notion we have very recently discovered that the number of AcE receptors on mouse macrophages declines as mice grow older. We are currently seeking drugs that increase the removal rate of unwanted advanced glycosylation end products, but a successful treatment will have to dissolve the end products without excessively damaging irreplaceable proteins. In the case of myelin, for instance, excess ncE-stimulated uptake of old or damaged protein could erode the myelin sheath, which is essential to nerve functioning.

Additional evidence must be gathered before we can say with certainty that nonenzymatic glycosylation of proteins contributes to the cell and tissue changes characteristic of aging. The data collected so far do indicate that our
hypothesis is a promising one. More important, the findings raise the exciting possibility that treatments can one day be developed to prevent some of the changes that too often make "aging" synonymous with "illness."

Health and aging: Scientists discover key to longevity in hypothalamus

The hypothalamus, an almond-sized structure located deep within the brain, is known to have fundamental roles in growth, development, reproduction, and metabolism.

Credits:

Albert Einstein College of Medicine

Scientists at Albert Einstein College of Medicine of Yeshiva University may have found the “Fountain of Youth” or rather, the “fountain of aging”.

Anti-aging and quantum theory
In a paper published on May 1st, 2013, in the online edition of *Nature*, Hypothalamic programming of systemic ageing involving IKK-β, NF-κB and GnRH, scientists pinpointed a protein complex in the hypothalamus that controls the aging process.

"Scientists have long wondered whether aging occurs independently in the body's various tissues or if it could be actively regulated by an organ in the body," said senior author Dongsheng Cai, M.D., Ph.D., professor of molecular pharmacology at Einstein. "It's clear from our study that many aspects of aging are controlled by the hypothalamus. What's exciting is that it's possible — at least in mice — to alter signaling within the hypothalamus to slow down the aging process and increase longevity."

Dr Cai noticed that inflammation in the hypothalamus gives rise to some components of metabolic syndrome which can lead to heart disease and diabetes. He said that when people age, there are inflammatory changes in the tissues when age-related diseases of the cardiovascular system are present and in neurological disorders and some cancers.

The scientists studied a specific protein complex called NF-κB (nuclear factor kappa-light-chain-enhancer of activated B cells).

"Even though inflammation involves hundreds of molecules, NF-κB sits right at the center of that regulatory map," Dr Cai said.

When the team activated the NF-κB pathway in the hypothalamus of mice, they aged significantly faster, lost muscle strength, skin thickness and the ability to learn. On the contrary, when they blocked the NF-κB pathway in the hypothalamus of mouse brains, the aging process slowed and longevity increased by about 20 percent.

The researchers also found that when they activated the NF-κB pathway in the hypothalamus levels of gonadotropin-releasing hormone (GnRH), (which is synthesized in the hypothalamus) declined. GnRH in the blood is usually associated with reproduction.

The researchers injected the hormone into a hypothalamic ventricle (chamber) of aged mice and found it protected the mice and aided in the creation of new neurons in the brain.

“According to Dr. Cai, preventing the hypothalamus from causing inflammation and increasing neurogenesis via GnRH therapy are two potential strategies for increasing lifespan and treating age-related diseases.”
The title of the paper is "Hypothalamic Programming of Systemic Aging Involving IKKβ, NF-κB and GnRH." The other contributors are Guo Zhang, Ph.D.; Juxue Li, Ph.D.; Sudarshana Purkayastha, Ph.D.; Yizhe Tang, Ph.D.; Hai Zhang, Ph.D.; Ye Yin, Ph.D.; Bo Li, Ph.D. candidate; and Gang Liu, Ph.D.; all at Einstein.

Tantric and Kundalini sex

Orgasm is an opening or stimulation of the chakra. Most people can only open their base chakra for sex. But when you learn to open the other chakra for sex then the door to pleasure is intensified. There are at least seven chakra points in the human body as told in the age old texts of the powers of the body. The ancient cultures knew of these power points which correspond to endocrine hormone glands and other things.

Tantric and Kundalini yoga teach techniques of opening and controlling these energy points. Years ago I had the pleasure of working with a Patrick Flanagan who taught about opening the chakra energy centers. He observed a group of yoga people who lived to old old age and used yoga daily. He found that they had developed a sexual technique of
absorbing the life force of their partners during sex. A type of energy vampirism, this had anti aging effects for the one absorbing the energy.

Tantra is a spiritual tradition that originated in India some 4,000 years ago. It is a way of life that celebrates and strives for the union of body, mind and spirit. Tantra is a form of yoga. Yoga means union. The vital principle of Tantra is a union of lovers, and union with the divine, with God. In the Tantric practice, sexuality and spirituality are joined. Lovers actually attract God right into their bed!
Most people spill their life force out during sex. The energy is released but not reabsorbed. These people could not only reabsorb their own energy release but could absorb the excess release of others. If very practiced they could steal the energy of others and use it to keep themselves alive longer. I learned the technique and I can teach you the basis of it now.

First recognize that all clocks worldwide are designed to go clockwise. This is because the photon energy of aging and the degeneration of life from quantum control to thermodynamic decay rotate clockwise. The bio photon energy of life is counterclockwise and is defies entropy and resists aging. So meditating on a clock rotating counter clockwise is a start.

Then when you feel the energy release of orgasm, don’t let the energy just spill out, channel the energy with your mind up the spine. Feel each chakra center and open them with your mind and channel the energy up the spine allowing it to flow outwards from the top of your head the
Anti-aging and quantum theory

crown chakra center. Then the energy will return like a magnets energy and flow back to the base of your spine to be rechanneled.

Feel the whole planet Earth, sense the solar system, sense the galaxy, sense the universe, and sense the endless list of the multi-verses. Expand your energy and circle of compassion to include all things. This is best done with a stable sex relationship where there are no rushing and uncomfortable feelings. On a one night stand there is too much tension usually. As you open each chakra center with practice your sexual pleasure is increased. As you widen your circle of energy and compassion while feeling the energy up your spine, out of your head and back to your base, your pleasure will increase.

You will absorb any free floating energy around you and watch out you will be able to steal others energy. But this has karmic implications. Please try to resist the urge, or at least use it wisely. If you do not your partner will age too fast and you will outlast them.

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<thead>
<tr>
<th>Chakra</th>
<th>Color</th>
<th>Endocrine organ</th>
<th>Sexual activation tip</th>
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<tbody>
<tr>
<td>The Crown Chakra</td>
<td>Violet</td>
<td>total and Brain</td>
<td>complete focus of all</td>
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<tr>
<td>The Brow Chakra</td>
<td>Indigo</td>
<td>Pineal Pituitary, Hypothalamus, Hypothalamus</td>
<td>low lights pleasurable sights</td>
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<td>The Throat Chakra</td>
<td>Blue</td>
<td>Thymus, Thyroid, ParaThyr</td>
<td>Music, Sounds, Mantra</td>
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<td>The Heart Chakra</td>
<td>Green</td>
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<td>Romance, Compassion</td>
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<td>The Stomach Chakra</td>
<td>Yellow</td>
<td>Stomach, Intestine</td>
<td>Good food, will power</td>
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<td>The Spleen Chakra</td>
<td>Orange</td>
<td>Adrenals, Kidneys</td>
<td>Adrenal tap, scratch</td>
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<td>The Base Chakra</td>
<td>Red</td>
<td>Gonads</td>
<td>Proper Genital stimulation</td>
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Tantric sex is not just a means of physical intimacy between sexual partners. It is also an emotional and philosophical way to gain a deeper level of understanding with your mate and to add to your witch powers. Though the art of Tantra is thousands of years old, you can learn from self discovery and networking with friends.
1. **Step 1**

*Take a seminar, class, or retreat. Or find a mentor who can teach you and coach your technique. As they say “When the student is ready the Master will Appear”.*
2. **Step 2**

Find a tutor. If you look online or in the classified sections of alternative newspapers, New Age magazines, or bulletin boards at wellness centers and fitness establishments, you will most likely find at least one or two listings for private Tantra tutors. Many of these tutors also offer group classes, but if you do not feel that you would be comfortable in a group setting than try to set up one-on-one time with the instructor. Be careful when setting up private instruction, as some people who claim to be professional Tantric Sex tutors could be looking for other kinds of encounters. Before you go in for an actual session, set up a meeting with them just to get to know them and see their teaching facility. Ask if you can get the names and numbers of some of their previous clients so that you can call and ask them if the tutor is legitimate. Be careful.

3. **Step 3**

Learn from a book. There are many books available on the subject of Tantric sexuality. You can start looking for one by going online and doing a search at places like Amazon.com or Barnesandnoble.com. The books that come up from your search will probably cover every aspect of Tantra you could think of, from the religious and philosophical side to the relationship side to the physical side. Once you find one that sounds right for you, either order it online or go into a bookstore with the author and title in hand so you can find it on the shelf.
4. **Step 4**

Watch an instructional video. These can be found in a number of places, including catalogues, Internet stores, and some video and bookstores. Like books on Tantric sex, they cover all of the aspects of Tantra, from physical postures and positions to philosophical meditations. For people who learn better from seeing things than just reading them, learning from a video can be a lot better than trying to learn from a book, as you will be able to see everything you are being instructed on.

5. **Step 5**

Research on the Internet. There are numerous websites available to learn Tantric sexuality from. Some have instructional videos and audio lectures, others have pictures and diagrams, and still others are comprised of mostly text and images. Like videos and books, these websites contain a variety of information on all aspects of Tantra and can be very helpful if you wish to learn in the privacy of your own home and are looking for as much information as possible. Some
websites will ask you to pay a fee, and will give instruction in the form of modules and class lectures as if you were taking a course at a school. Others will contain their information in essays and quotes from experts in the field.

6. Teach yourself, for the best teacher is within.

Never mistake Tantra for lust! Some Tantrikas undergo years of training so that they can experience sacred sexuality without the least trace of lust ever crossing their minds! "I also love this part, and can attest to the truthfulness of it~ "In ordinary sex, people rush to get to the end...But in Tantra, the process of getting there is even more delightful~ Tantrika adepts can make love for several hours or even longer~
The popular understanding of Tantra in the West represents only a part of the vast system. "Tantra" means a loom or weaving. In Tantric teachings, the universe is perceived as a fabric where everything is woven together, connected and related seamlessly. Tantra is also translated as "web". We are all connected to each other in this infinite web. The main initiators of Tantra into the esoteric world were originally female adepts—These women were seen as manifestations of the divine Shakti, the female aspect of the ultimate reality Devi created the universe and the whole universe is her body—The sun and moon are Her eyes—The wind is Her breath—The mountains are Her bones—The rivers and oceans are Her veins—Devi is omnipresent and all-knowing...Electricity, magnetism, power, heat, light, and the five elements are Her eternal manifestations—She is the primal force of life which underlies all existence and the Tantrik seeks union with Her—
By use of mantras a person is able to create and experience the vibration of the Goddess. Mantra is so important in Tantra that it has been called "Path of the Mantra." Mantra is a chant, a prayer, an invocation, a magical syllable, living words made of power, and a manifestation of the Goddess. A Mantra is a combination of sacred syllables which form a nucleus of spiritual energy. It serves as a lens to focus spiritual vibration. The potency of mantra is released through repetition and this may entail many thousands of repetitions or just one, depending upon the receptivity of the individual. OM is the supreme mantra and the easiest to remember. The mantra of the Hindu and Buddhist goddess Tara is OM HRIM STRIM HUM PHAT. The universe and every being are made of these occult sounds, letters of light, the little mothers of existence. The mantra helps to focus the mind and awaken the supernatural energies within the body and to evoke the presence of the Goddess. Mantra is used to bring oneself into harmony with the universe, uniting individual and cosmic consciousness (the Goddess). The devata is the presiding deity of the mantra, a very personal aspect of the goddess. Mantra is Goddess of the spoken word. Tantra views the body as a living temple. A mysterious force is active in the body. It is in the brain, the cells, everywhere. Consciousness is not found in the brain alone, but exists throughout the body. Raising the Kundalini (life force) is a primary goal of Tantra...
The Kundalini awakens in the lower back and when it reaches the brain through Tantric methods, bliss and enlightenment result. In Tantra there is always an emphasis on personal experimentation and experience. Tantrikas experience enhanced sensory and mental capacities. Though our ordinary perception is confined to a narrow range, Tantrikas can often feel an intense variety of universal vibrations. Tantric yogis use the senses as a means of elevating their consciousness. Tantric yoga is the only system of yoga which deals with human sexuality and focuses on the unification of male and female energies. By merging with a partner, one can experience the blending of male and female essences. Within each gender lies the seed of the opposite sex which creates attraction between two people. The Taoist sages call these opposites yin and yang (feminine and masculine respectively). Both yin and yang exist in men and women, with the yen predominantly in women, and the yang in men. These polarities complement each other and it is important to learn how to balance them. Tantra endeavors to combine yin and yang into blissful union and although one can work with a sexual partner to accomplish this, the inner yoga practices of Tantra can enable an individual to unfold and unite the male and female energies.
and female energies without a partner. In fact, it is better for a person to do Tantric spirituality alone than to attempt it with the wrong type of sexual partner. In Tantric sexual practice, to see your partner as divine, to actually and deliberately worship and adore the divine in her/him, initiates the process of transformation. Tantra teaches that men and women are manifestations of god and goddess. Two of the ancient Hindu Tantric models for this are Shiva and Shakti, Krishna and Radha. These Divine couples demonstrate the possibility, through Tantric sexual union, of achieving a fully conscious awareness of themselves as God and Goddess. Tantric partners should maintain eye contact and become aware of each other's breathing until their breaths come simultaneously. With practice, they'll eventually match their heartbeats, energy, and consciousness. In Tantric sex the couple becomes one Being and they are able to experience the ecstasy of sexual union for an hour or more. In this sense, Tantric sex is used as a vehicle to expand consciousness. Experiencing oneness with each other will enable them to experience the ultimate oneness with the Goddess. Sexual foreplay, which should last at least an hour, is used to build energy and increase a couple's awareness of each other.
In ordinary sex, people rush to get to the end—But in Tantra, the process of getting there is even more delightful—Tantrika adepts can make love for several hours or even longer—In Tantric sex the climax is avoided as long as possible or even dispensed with entirely so as to build energy which can be used for psychic experiences or even for spiritual enlightenment—*~Tantra is not about giving into lust~Many people have been led astray in Tantric sex because they only fell into lust~*~Never mistake Tantra for lust! Some Tantrikas undergo years of training so that they can experience sacred sexuality without the least trace of lust ever crossing their minds! Suggestions for creating a Tantric environment include keeping the bedroom clean, as well as oneself, use incense and candles, and meditative music. Eat a light meal and either drink just one glass of wine to attain a slight glow and to help relax or abstain from alcohol completely for the evening. Use massage to begin foreplay. Dancing, feeding one another and taking a bath or shower together can also begin Tantric foreplay. Loving conversation is good as long as it’s not of the past or future...Keep your thoughts in the present! Tantric sex requires you to fully engage all of your senses—Be aware of what you hear, of what you smell, of what you see—Be aware of the sensations on your skin—Try to be totally cognizant of as many sensations as possible occurring at once—In this heightened state of awareness, your body will give you messages—Listen to them—Tantra seeks to animate and control the life force—When Tantric lovers connect deeply and their energies join, they magnify the life force in each other—If one person constantly drains the other, then this is conscious or unconscious vampirism—Lovers can exchange and magnify energies by holding one another and breathing together—This Tantric practice can lead to a pure and enlightened love that is beyond sex—Ultimately love is Divine...Love is God/dess—Tantra uses love’s energies to awaken the soul’s light, power, and awareness—Several forms of Hinduism teach that the world is an illusion, but Tantra teaches that our world is real—Tantra celebrates the physical aspect of creation—The Tantrika enjoys all aspects of life and some of the skills s/he develops are composing poems, drawing, singing, chanting, writing, gardening, cooking, playing musical instruments, dancing, tattooing, sewing, solving riddles, carpentry, meditation, sports, gymnastics and sexuality—Those who practice Tantra know that all seemingly ordinary activities can lead one to higher mystical awareness if these things are done mindfully and skillfully—Basic Tantric mind training teaches one how to control thoughts and moods—We must always learn to think consciously without daydreaming, thus...
do our thoughts become focused and powerful. Eventually we can even experience the primal cosmic realm that has eternally existed beyond all thoughts and symbols. Without mental discipline, Tantra, nor any form of yoga or spirituality can be effective...The mind is our basic tool—A strong mind saves us while a weak mind dooms us! Tantra teaches that there is a constant exchange of energies between people, animals, plants, planets, stars and universes—It is the blessing of a human being to be able to feel things more deeply and consciously. The human being can sink lower than the animals or rise higher than the angels—Tantra is a way of life, a path of discovering the ecstatic in everyday life. Everything is to be experienced as a gift from the Goddess—Passion is important in all aspects of life and in Tantra it’s understood that our search for passion and pleasure is really a search for God/dess—Sex is only one doorway to the Divine—Each flower is a doorway—Each smile of a child is a doorway—For the Tantrika adept, there is nothing but doorways—Even eating, drinking, or listening to music can be a communion with God/dess—God/dess is in a finger, a nut, or a glass of water—A feeling of warmth is felt. The soul becomes a fire—An inner light is seen...When God and Goddess unite within us, we experience the true inner marriage—
Anti-aging and quantum theory

10 Foods That May Improve Your Appearance and YOUR WELLNESS

Get skin glowing and hair shining the natural way, while making your body more well.

Collagen is a natural protein in your skin and muscles that provides resiliency, shape and texture. Unfortunately, collagen production decreases with age—but you can fight back with dark fruit. "Blood oranges, cherries and blueberries are full of antioxidants, which decrease aging and disease by lowering inflammation. Antioxidants also increase collagen production and thicken the skin, making you look younger and healthier," says Julia Tatum Hunter, M.D., of Skin Fitness Plus in Beverly Hills. "Antioxidants also decrease [the severity of] rosacea." Blackberries, raspberries, plums, pomegranates, cranberries, Asian dragon fruit and kiwis also.

A recent Canadian study concluded that getting more potassium might help lower your weight and blood pressure. Levels measured in study participants were proportional to their diet and weight. "That makes sense," says Blatner. "The richest sources of potassium are beans, vegetables, and fruit, so the person with high potassium levels is consuming a lot of these foods, which are low in calories and are the most filling." You should aim for 4,700 milligrams of potassium each day. Supplements may help you hit that target, but doctors don’t recommend them for everyone. Try filling up on white beans (1 cup: 1,000 mg. potassium), winter squash (1 cup: 494 mg.), spinach (1 cup: 840 mg.), baked potato with skin (926 mg.), yogurt (1 cup: 600 mg.), halibut (4 ounces: 566 mg.), and orange juice (1 cup: 473 mg.).
2. Shellfish, sunflower seeds and sardines
These foods may not taste great together, but individually they offer a powerhouse of essential fatty acids. Steven Chang, M.D., staff physician for RightHealth.com, says fatty acids nourish the skin, help maintain skin integrity and keep skin cells performing optimally. "Essential fatty acids, a component of all cell membranes in the body, regulate the flow of nutrients, waste materials, and water in and out of cells—which keeps you looking young." Flax seeds, tuna, walnuts, canola oil, soybean oil and pumpkin seeds are more good sources of essential fatty acids. Foods are better than powders, powders are better than pills, and pills are better than no supplements.

3. Dandelion, turnip and mustard greens
"Foods that keep our livers cleansed of toxins, heavy metals and fats make our whole body function more efficiently," says Dr. Hunter. "This makes us happier, which affects how we look. Plus, a healthy liver brightens our eyes and tightens our skin." She recommends dense green foods such as broccoli, spinach and arugula—as well as turnip, mustard and dandelion greens. Eating these slightly bitter greens has been shown to lessen your sweet tooth. Hunter warns: "Simple and refined sugars, high-glycemic carbohydrates, and refined, manufactured foods age us." Excess sugar has been linked to a process called glycation, in which sugar molecules bond to protein molecules, which has been linked to sagging, wrinkled skin.

4. Oregano, thyme and parsley
"If you have puffy bags under your eyes in the morning, you are almost certainly consuming much more salt than you need," says Doris Day, M.D., author of Forget the Facelift: Turn Back the Clock with a Revolutionary Program for Ageless
Skin (Avery, 2005). "Another problem is alcohol: It dehydrates you and can make your skin sag. The worst combination is alcohol and salt, which causes puffy dark circles under your eyes." Dr. Day recommends reducing your sodium intake to eliminate bloating. Instead of salt, season your meals with herbs and spices such as oregano, thyme, rosemary, parsley and garlic.

5. Crunchy vegetables
Fresh raw veggies are as good for your grin as they are for your skin! Celery, carrots, string beans and cauliflower contain cellulose, which helps scrub stains from your teeth—giving you a whiter, brighter smile. "Both the cellulose and the [other] fiber in these foods act as abrasives that clean and remove bacteria from teeth," says Mickey Bernstein, M.D., president of the American Academy of Cosmetic Dentistry. Crunchy veggies are especially effective for recent discolorations. If you've just consumed blueberries, coffee, mustard, red wine or cranberry juice, follow it up with fresh cucumber slices or an apple. Foods are better than powders, powders are better than pills, and pills are better than no supplements. Water in your glass is good, but water in your food can have serious slimming power. In a new American Journal of Clinical Nutrition study, obese women ages 20 to 60 were told to either reduce their fat intake or increase their intake of water-rich foods, such as fruits and veggies. Although they ate more, women in the water-rich group chose foods that were more filling—yet had fewer calories—so they still lost 33 percent more weight in the first 6 months than the women in the reduced-fat group. Fill up on food that's high in H2O. Some good choices in addition to fruits and veggies: broth-based, low-sodium soups; oatmeal and other whole grains; and beans.
6. Sea vegetables

"Polluted cells can't function at their optimum level. When our cells are functioning optimally, not only do we have more energy—we look and feel great," says nutritionist Carol Wasserman. "Sea vegetables are one of our richest sources of minerals and phytochemicals." These veggies help detoxify, rebuild and nourish all the cells in our body. Unhealthy foods, stress and environmental pollutants cause cells to age prematurely, potentially leading to thinning hair and premature wrinkles. "Sea vegetables reverse this process," says Wasserman. "For example, spirulina is a ‘detox powerhouse.’ Hijiki, kelp, arame, wakame, and dulse also work wonders." Foods are better than powders, powders are better than pills, and pills are better than no supplements.

7. Meat, cheese, lentils, and sprouts

It may take 10 pounds of milk to make a pound of cheese, but fortunately you don't need to eat that much dairy or protein to repair your cells. As you age, your hair and skin cells become damaged, making you appear older. The protein in meat, chicken, low-fat cheese, cottage cheese, and certain vegetables promotes cell growth and repair, which translates to younger-looking skin, fewer wrinkles, less hair loss and a glossy mane. To take a break from meat or dairy, try soybeans and lentils instead (they contain more protein than any other legume). The need for protein is over rated. We should not absorb protein, but should break down the protein to its’ amino acids. Sprouts are rich in the amino acids we need and thus supply the needed factors. When you get a meat craving it is because you are craving amino acids. A small handful of sprouts will supply the amino acids you need. And the meat craving that is left is just addiction.
8. Egg yolks, organ meats
Dr. Chang says, "Vitamin A is especially important for skin repair, and decreased levels can lead to dry, flaky skin." Dr. Day adds that a lack of vitamin A may cause your skin to heal poorly and wrinkle easily. The main sources of this vitamin are foods from animals, such as liver, eggs and whole-milk dairy products. Some plants—carrots and broccoli, for example—supply beta-carotene, which your body converts to vitamin A as needed. Apricots, nectarines, plums and cantaloupe are more great sources of beta-carotene.

9. Almond or hemp "milk"
Almond milk is a nutritious dairy alternative because of its high levels of magnesium, potassium, manganese, copper, vitamin E, selenium and calcium. Licensed medical esthetician Tina Seitz says, "Hemp milk is a delicious, nutty-tasting non-dairy beverage that provides essential balanced nutrition. It's a fantastic alternative to soymilk or dairy, and has a natural well-balanced ratio of omega-3 and omega-6 essential fatty acids to keep your mind sharp, your immune system strong and your skin glowing." Both almond and hemp milks are plant-based, and don't contain lactose. They offer high-quality protein that can give hair a radiant, healthy shine and helps keep skin soft.
10. Wild salmon with avocado and mango dressing

This is more than a delicious meal—it’s an anti-aging feast! Stephen Sinatra, M.D., of the University of Connecticut School of Medicine says, “Wild Alaskan salmon has precious omega-3 essential fatty acids, which enhance blood flow. The pink/orange color of wild salmon is an anti-aging carotenoid called astaxanthin that protects cell membranes.” Salmon also contains dimethylaminoethanol (DMAE), which improves facial muscle tone and reduces wrinkles. Add avocado for its antioxidant properties and mango (for vitamin E and anti-inflammatory carotenoids) and you’ll be sitting pretty after dinner!

MOST IMPORTANT

Eat good sugars and good oils. Avoid bad sugars and bad oils.

Dextrose sugars are bad because they have a high glycemic index and go into fat very quickly while weaken the immune system the nerves and the hormone production. Dextrose sugars are white, sugar cane, sugar beet, corn sugar. Levulose or fructose (fruit sugar) as it is called makes less fat, more hormones and strengthens the immune system.

Cold processed plant oils are best. They contain unsaturated fatty acids which are carbon chains. They make the cell membranes of all cells. Thus the visible skin is made of fatty acids. Once a fatty acid is boiled or cooked the fatty acids become trans fatty acids and acrylamides are formed by cooking that cause cancer. Avoid any food boiled in oil. And avoid any trans fatty acid containing processed food.

Avoid nitrates especially in processed meats. Hot dogs, bologna, lunch meats, convenience meats, nitrate sausage and other processed meats are more of a cancer risk than anybody has suspected. The nitrates produce age acceleration and a host of other problems. Avoid at all costs.

Avoid smoking and exposure to smoke. This is an age accelerant. Also most synthetic drugs accelerate aging.

Avoid excess or disturbing stress. At age forty, life has given you your face. If you have been over stressed and over reactive your face will show the life.
WHAT ABOUT CHOCOLATE

Cocoa can lower blood pressure; reduce the risk of heart attack, stroke, diabetes, and dementia; and possibly even prevent cancer. But the research isn’t as delicious as it seems. The cocoa-bean products used in the studies are a far cry from the highly processed chocolate candy you find on the shelves of your local store. “Milk chocolate contains about 150 calories and 10 grams of fat per ounce,” says Campbell. The key here is small doses. Dark chocolate, which retains more of the bean during processing, generally has slightly less fat and fewer calories than milk chocolate—plus, it’s richer, so less goes a longer way.

COMMON SENSE IS SO RARE THESE DAYS, IT SHOULD BE CLASSIFIED AS A SUPER POWER

Armed with Clarity Of Mind, Dedication to Truth and the Ability to Leap over a Bigot with a Single Bound SUPER DESI fights against the Forces of Greed and Lies for Truth Justice + the American Gay