THE AVANT GARDE
ON THE CUTTING-EDGE

In this issue:

An antiaging, life-extending aphrodisiac

By Ward Dean, M.D.

Forever Young

An interview with Dr. Bill Lawrence

Peptide technology for dental care

Discover OraltidePRO™

Living longer with an Easter Island discovery

The story of Rapamycin
1. **Declaration:** The Aging Matters™ magazine is intended for IAS private club members (and therefore is not intended for the public). It focuses on the latest international nutritional, hormonal and drug therapies to help combat the signs of aging. These signs include the physical, mental and internal changes consisting of the diseases and disorders such as cancer, arthritis and senile dementias etc. However, the main focus is upon the prevention of such aging diseases and disorders for the ‘healthy-aging’ individual.

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**TESTIMONIALS**

**DR. AUBREY DE GREY  Ph.D.**

“IAS has shown great vision and leadership as an organization focused mainly on the provision of contemporary medical interventions against aging, and in also supporting the SENS Foundation efforts to hasten the development of much more powerful future interventions.”

**NICHOLAS PERRICONE  M.D.**

“IAS is an outstanding resource for the finest, most up-to-date news and information on healthy aging. They also offer products of the highest integrity and efficacy. In fact, IAS is the world’s greatest source, (often the only source) for the most cutting-edge and advanced nutrients to ensure optimum health span and maximum life span.”

**THIERRY HERTOGHE  M.D.**

“IAS have a history of making throughout the world crucial, but difficult to access medications available to patients. IAS is one of the pioneering societies in antiaging medicine that has helped this new medical speciality move forward.”

**JONATHAN V. WRIGHT  M.D.**

“Every adult has the right to take care of his or her own personal health as he or she chooses. In the 20th and 21st centuries this universal human right has been nearly obliterated by an ocean of nanny state regulations and deliberate suppression of information by bureaucracies, with hidden and not-so hidden agendas. International Antiaging Systems is a beacon of useful health care information and a literal island of freedom of health care product choice in our otherwise unfree health care world.”

**DR. WALTER PIERPAOLI**

“I have known IAS for many years and they are a qualified group who provide for me, my family and my patients. Their skill and professional capacity has liberated me from all sorts of problems concerning the search for guaranteed and often rare supplements, or anything which is available but problematic to find. Their service goes far beyond duty and helps in many ways to maintain optimal health.”
Avant Garde translates as ‘new and experimental’ and whilst it is often applied to art and fashion, (hence the model on the cover), in this issue we are applying it to methods and protocols that are becoming popular in the field of antiaging medicine.

Dr. Dean’s article on deprenyl, (otherwise known as seligiline) is a reminder of an agent that enhances dopamine and therefore has been a treatment of Parkinson’s disease for many years. But, at much lower doses it appears to have some intriguing benefits for normal healthy people, including (in animals at least) extending lifespan.

Another article looks at the development of peptides being utilised for dental healthcare, helping to prevent degradation and repair various tissues including teeth and gums.

Once again, we are continuing to highlight how peptides are becoming recognised to have numerous and major health benefits.

Since recent studies have been suggesting that dental healthcare may be related to many types of ailments, including Alzheimer’s, keeping the mouth clean and healthy could have much wider health implications than we ever imagined.

Another of Will Block’s contributions delves into the ever increasingly popular rapamycin, a substance that was originally discovered in the soil of Easter Island, and is now the defector mTOR inhibitor.

We are also delighted to have spoken with Dr. Bill Lawrence; as an American physician he is now halfway through a 3-year trial utilising the Russian peptide bioregulators in his patients.

In an appropriately titled article; ‘Forever Young!’ Dr. Lawrence describes some of his real-world results and experiences, some of which have been remarkable.

Naturally, we’re pleased to share with you what Dr. Lawrence has seen to date. More information will undoubtedly follow and Aging Matters™ will be there to publish it when it happens.

Perhaps not all these agents are brand-new, but when applied in a new way their uses certainly are.

However, they are part of the Avant Garde and we hope that you enjoy reading the Aging Matters™ magazine because, by definition, we report on what is new and experimental in health today.

Phil Micans, MS, PharmB
Editor, Aging Matters™ Magazine

Ward Dean, M.D.
Medical Director

www.aging-matters.com
New research suggests a significant link between the gut, its bacterial population and mental health. For the first time, scientists have explored this connection in humans and have identified the potential reasons for this.

Two types of bacteria; Coprococcus and Dialister are depleted in people with depression, according to researchers in Nature Microbiology. The study also revealed that many gut bacteria can produce compounds that act on the nervous system. If this is confirmed with further investigation, the results could lead to a deeper understanding of the gut-brain link and could even open new avenues for treatment of mental illness.

**THE STUDY**

Scientists analysed faecal microbiome data in conjunction with diagnosis of depression, 1,054 people took part in the Flemish Gut Flora Project.

During the analysis, the research team presented that the two types of bacteria; Coprococcus and Dialister were absent from the guts of people diagnosed with depression. This remained the case in those who take antidepressant medication.

The scientists confirmed the findings in another study where 1,063 people enrolled in LifeLinesDEEP, which collected data on the gut microbiota, the same group of individuals were treated for clinical depression at the University Hospitals Leuven.

The studies co-author Prof. Jeroen Raes states, “The notion that microbial metabolites can interact with our brain- and thus behaviours and feelings is intriguing, but gut microbiome-brain communication has mostly been explored in animal models, with human research lagging behind.”

In the future, Prof. Reas and his research team aim to confirm these results through further experiments. They are already preparing to analyse upcoming samples collected via the Flemish Gut Flora Project.
Further Reading

The Flemish gut flora project.

Life Lines Deep. Lifelines DEEP has already resulted in 14 high-profile publications including several high-impact papers with wide-reaching results. In the April 2016 Science paper, for example, deep sequencing of the gut microbiome showed relationships between variations in the microbiome and 126 exogenous and intrinsic host factors.

Researchers are developing a new pill that administers insulin straight into the stomach wall. Could this mean that injections will soon be a thing of the past?

When type 2 diabetes develops, the pancreas becomes unable to produce enough insulin. This is when doctors usually prescribe daily insulin injections to manage blood sugar levels.

Although, a phobia of needles has presented a significant barrier preventing those with type 2 diabetes from taking the insulin that they need.

Robert Langer, a professor at the Koch Institute for integrative Cancer Research, Massachusetts Institute of Technology and his colleagues are working to make the release of insulin into the bloodstream more flexible.

The research team developed a pioneering new pill design, it consists of a biodegradable capsule containing an insulin micro needle. When the pill is swallowed, the insulin is injected straight into the stomach wall.

The stomach lining does not have any pain receptors; therefore, the researchers believe that this method of delivering the drug will be pain free.

Langer states, “We are really hopeful that this new type of capsule could someday help diabetic patients and perhaps anyone who requires therapies that can now only be given by injection or infusion.”

Further experiments on the capsule system is ongoing. Langer and his colleagues are hopeful that this new pill could replace a host of drugs that are only available by injection.

**Further Reading**


Robert S. Langer. Science + engineering= conquering cancer together. KOCH Institute for integrative Cancer research at MIT
A recent study reveals the bacteria that is caused by bleeding gums is also found in the brain of patients with Alzheimer’s.

Researchers made the discovery after analysing brain tissue, saliva & spinal fluid from dead and living patients diagnosed with Alzheimer’s. Evidence presented that toxic enzymes as well as the DNA from the bacteria were found. Additionally, the bug spread from the mouth to the brain of mice in the study. Tests on the animals confirmed that the enzymes destroyed their brain neurons.

University of Louisville researcher Jan Potempa, Ph.D. the department of Oral Immunology and Infectious Diseases in the School of Dentistry was part of the team of international scientists. According to Potempa, although the infectious agents have been implicated in the development and progression of Alzheimer’s disease, more research needs to be carried out.

Dr David Reynolds, chief scientific officer at charity Alzheimer’s Research UK, states that Alzheimer’s is likely to have multiple causes, one of which may be gum disease bacteria. He adds, “Maintaining good dental health is an important part of a healthy lifestyle, and while we don’t yet fully know the extent to which it can effect our dementia risk, the presence of a single type of bacteria is unlikely to be the only cause of the condition.”

OraltidePRO™ is a mouthwash containing DPR (Dental Bond Peptide) and AGDP (Anti-gingival Degenerate Peptide).

What is the role of peptides in the gum?

Promoting collagen and extracellular matrix (ECM) synthesis, activating growth factors to accelerate mouth healing such as tongue wounds. Healing, promoting growth of shrinking gums and relieving symptoms of inflammation of the gums.

Benefits of using a peptide-based mouthwash:

- Promotes growth of shrinking gums
- Speeds healing of mouth & tongue
- Prevents oral infections (such as gingivitis)
- Helps with enamel remineralization
- Reduces bacteria growth and etching
- Fills slots in damaged enamel

How long does OraltidePRO™ typically take to be effective?

- For sensitive teeth: 1 week of continued use.
- Wound healing/anti-inflammation: 6 times of continual use.
- Growth of shrinking gums: 2 months of continual use.

How to use OraltidePRO™:

1ml OraltidePRO™ mixed with 10ml water, or smaller amounts diluted at a ratio of 1/10. Hold in mouth for 5-10 minutes 1-2 times a day.

Take care of your dental health, if you have any concerns seek the advice of your dentist.
Further Reading


New generation of therapeutics based on understanding of aging biology show promise for Alzheimer’s disease by Alzheimer’s Drug Discovery Foundation.
Deprenyl (later known as selegiline) was developed by Professor Josef Knoll of Semmelweis University in Hungary in the 1950s. It was first used as an anti-depressant and was later used for the prevention and treatment of Alzheimer’s and (especially) Parkinson’s Disease. Deprenyl’s value initially was based on the belief only that it was a monoamine oxidase B (MAO-B) inhibitor. MAO-B is an enzyme that degrades the neurotransmitters dopamine, epinephrine, nor-epinephrine and serotonin in the brain. MAO-B levels rise with age (Fig.1) and it was believed that this caused a decrease in these neuro-transmitters, which resulted in depression, Parkinsonism, and other neurodegenerative diseases. By selectively inhibiting MAO-B, deprenyl was theorized to maintain these neuro-transmitters at more youthful levels.

LIFE-EXTENDING EFFECTS

In 1983, Dr. Walter Birkmayer in Germany reported that Deprenyl, combined with L-dopa, not only improved the on-off phases and rigidity of Parkinson’s disease and reduced adverse reactions to L-dopa, but also prolonged the life expectancy of Parkinson’s patients. Birkmayer’s preliminary report was confirmed by ten-year long studies in nearly 1,000 Parkinson’s patients who added deprenyl to their regimens after L-dopa had lost its efficacy. The L-dopa-plus-deprenyl-regimen significantly delayed the progression of Parkinson’s symptoms, and increased life expectancy compared to those on L-dopa alone.

**Figure 1:** Platelet MAO-B increases with age.  

**Figure 2:** Striatal dopaminergic neurons. The striatum is the main input nucleus of the basal ganglia and a key neural substrate for procedural learning and memory.
Autopsies showed that deprenyl prevented or retarded the degeneration of striatal dopaminergic neurons in the brain (see Fig. 2). Scientists in the U.S. and Finland recommended that Deprenyl treatment be started as soon as the Parkinson’s diagnosis was made.

But it wasn’t just Parkinson’s patients that were living longer with deprenyl. In 1988, Knoll rocked the gerontological community with a study that showed a dramatic extension of maximum lifespan of rats treated with deprenyl (Fig. 3).

Knoll and his colleagues treated 132 24-month-old male rats, (equivalent to 60-year-old humans) with injections of saline or deprenyl three times/week. The average lifespan of the saline-treated group was 147 weeks. The oldest rat in the saline group was 166 weeks old when it died. In contrast, the first rat to die in the deprenyl group lived 171 weeks, (five weeks after the last control rat died), and the longest-living deprenyl-treated rat died in its 226th week! The average lifespan of the deprenyl-treated group was 198 weeks—i.e., higher than the previously-estimated maximum lifespan of the rat (182 weeks). Knoll claimed that this was the first time that administration of a drug or nutrient resulted in extension of a species’ known maximum lifespan.

In addition to these dramatic findings, the scientists evaluated sexual functioning in the rats, as a measure of their brain striatal function. Because of the normal age-related decay of sexual function, none of the 2-year-old animals displayed full-scale sexual activity. In the saline group, the last signs of sexual activity had completely vanished by the 33rd week of treatment. In contrast, deprenyl treatment restored full-scale sexual activity in 64 out of 66 rats!

Inspired by Knoll’s groundbreaking lifespan studies with deprenyl, researchers around the world began trying to duplicate his results in a variety of species. Researchers at the University of Toronto in Canada gave injections of deprenyl (0.25 mg/kg) or saline every other day to male Fischer rats starting at 23 to 25 months of age.
The deprenyl-treated animals showed a significant increase in both mean and maximum survival.\textsuperscript{12} Scientists from the Tokyo Metropolitan Institute of Gerontology reported their results with 70 male Fischer 344, (F-344) rats treated with injections of deprenyl or saline 3 times a week from the age of 18 months until they died. Although their results were not as dramatic as Knoll’s, the average lifespan of deprenyl-treated rats after 24 months was 34\% greater than saline-treated controls, lending support to the growing awareness of deprenyl’s life-extending properties (Fig. 4).\textsuperscript{13, 14}

In Germany, scientists treated 14 immunosuppressed mice- beginning at 2 ½ months of age, with half the group receiving deprenyl-laced food. The last mouse in the control group died at the age of 5 months (2.5 months after the study began). In contrast, the last mouse in the deprenyl group died at the age of 14.5 months—1 year after the beginning of the study, having lived nearly three times as long as the longest-living control mouse!\textsuperscript{15}

Scientists at the Jackson Laboratory in Bar Harbor, Maine, conducted studies on 2 strains of mice, starting at mean ages of 26 and 18.5 weeks. In the study that began at 26 weeks, there was a 77-day increase in mean female lifespan, and an 84-day increase in mean male lifespan. For the mice that began treatment at 18.5 weeks of age, the mean longevities were increased only 59 days in females, and 56 days in males. Despite the inconsistencies, the authors concluded that since all studies found increased lifespans in deprenyl-treated mice, further research with deprenyl as a life-extending substance was justified.\textsuperscript{16}

\textbf{Figure 4:} Extension of lifespan of deprenyl-treated rats (open circles) from the Tokyo Metropolitan Institute of Gerontology. Treatment began at 18 months of age, resulted in 34\% increase in the remaining life expectancy after 24 months.\textsuperscript{13}

\textbf{Figure 5:} Survival curves of female (A) and male (B) Syrian hamsters. Female hamsters normally have shorter lifespans than males. After deprenyl treatment, the difference of lifespan between male and female hamsters disappeared.\textsuperscript{17}
Despite the inconsistencies, the authors concluded that since all studies found increased lifespans in w-treated mice, further research with deprenyl as a life-extending substance was justified.\textsuperscript{16}

In 1997, a team from Mannheim, Germany, reported on deprenyl’s effect on the lifespan of Syrian Hamsters. At the age of 13 months, 36 pairs of hamsters were treated—half of which received 0.05 mg/kg deprenyl in their food. The scientists surprisingly found that deprenyl significantly increased life span in the females, but not in males (Fig. 5).\textsuperscript{17} This was especially significant as females of this species normally had a shorter lifespan than males.

Two months later, the same journal published a study by scientists at the VA Medical Center in Denver, CO, using male Fischer 344 rats. They administered deprenyl in drinking water to 14 rats, beginning at 54 weeks of age (16 rats served as controls) (Fig. 6). Although there was no difference in the maximum lifespan due to deprenyl, the mean lifespan was significantly longer for the deprenyl-treated rats (110 vs. 103.5 weeks).\textsuperscript{18}

In the same year, thirty-three elderly beagle dogs between the ages of 10-15 years old were given 1 mg/kg of deprenyl or a placebo each day for 104 weeks by researchers in Overland Park, Kansas. Twelve of 15 (80%) dogs in the deprenyl group survived to the conclusion of the study, in contrast to only 7 of 18 (39%) of the dogs who received placebo. By the time the first deprenyl-treated dog died on day 427, five placebo-treated dogs had already succumbed—the first on day 295 (see Fig. 7). The authors concluded that daily administration of 1 mg/kg deprenyl could prolong the life of relatively healthy 10-15-year-old dogs.\textsuperscript{19}

The German team that three years previously had tested deprenyl on immunosuppressed NMRI mice (cited above) did another series of experiments using various combinations of deprenyl and lipoic acid in ten groups of 14 mice. They obtained the best results with doses of 75 mcg/kg deprenyl plus 9 mg/kg of R-lipoic acid.

\textit{This combination resulted in nearly 200\% increased maximum lifespan}.\textsuperscript{20}

**Figure 6:** Extension of lifespan of male rats treated with deprenyl in their drinking water, compared to controls. Treatment was begun at 54 weeks of age. Deprenyl-treated rats exhibited higher survival rates at all time points after 62 weeks of age.\textsuperscript{18}

**Figure 7:** Survival of dogs between 10 and 15 years old at the start of the study, treated with deprenyl for at least six months. Note that by the time the first deprenyl-treated dog died on day 427, five of the placebo-treated dogs had already died.\textsuperscript{19}
**MULTIPLE MECHANISMS FOR DEPRENYL’S BENEFITS**

Besides its known MAO-B inhibiting properties, scientists in the UK reported that deprenyl also induced increased levels of superoxide dismutase (SOD) and suggested this as the basis of deprenyl’s neuroprotective and life-extending effects. The Japanese team led by Kitani has probably done more ongoing research with deprenyl’s effect on lifespan than any other group. Kitani speculated that the life-extending effects of deprenyl were not due solely to its MAO-B inhibiting effects, but to a multiplicity of mechanisms. These included elevations of catalase as well as SOD (as found by the British team mentioned above). Other benefits attributed to deprenyl included immune system enhancement characterized by increased concentrations of cytokines such as interleukin-1beta (IL-1beta), tumor necrosis factor-alpha (TNF-alpha), interferon-gamma, and natural killer (NK) cell functions. Kitani speculated that these humoral factors were responsible for deprenyl’s ability to prevent malignant tumors in rodents and dogs, and for its diverse antiaging and life-prolonging effects by enhancing homeostatic regulation of the neuro-immuno-endocrine axis.

Researchers from the Department of Pharmacology, University of North Dakota School of Medicine and Health Sciences expanded upon deprenyl’s potential life-extending mechanisms by noting that deprenyl also enhances cyclic AMP; restores insulin-like growth factor I (IgF1); possesses neurotrophic-like actions and enhances the synthesis of nerve growth factor; and protects against peroxynitrite- and nitric oxide-induced apoptosis.

**LESS MAY BE MORE**

When deprenyl was first clinically used, the recommended dose for Parkinson’s disease was 5 mg in the morning, followed by a second dose at noon. Prof. Knoll early-on recommended that those at risk of (or diagnosed with) Alzheimer’s disease “should take 10 mg daily for the rest of their lives.” This is probably still good advice for those suffering from these dementing illnesses.

However, Kitani and his associates found that with continued use, lower doses of deprenyl provided better effects with regard to optimizing antioxidant enzyme levels in the brain and maximizing the life expectancy and lifespan of experimental animals. They determined that the dosage used for any life span study was a critical factor, with the dosage differing widely depending on sex, age of the animal, and duration of therapy. In fact, Kitani’s team proposed that long-term treatment with deprenyl decreased the optimal dose by a factor of at least 5 in terms of upregulation of antioxidant enzymes. Although Dr. Kitani had conducted more studies with deprenyl than any other scientist except Prof Knoll, there is no indication that he used deprenyl himself. He passed away unexpectedly on October 15th, 2008 at the age of 73 from colon cancer.

Kitani’s conclusions agree with Knoll’s later recommendations. Whereas Knoll initially recommended dosages of up to 10 mg per day for those with Parkinson’s and Alzheimer’s diseases, he explained in his later work that deprenyl has enhancing qualities in “femto-picromolar concentrations,” which leave MAO-B activity unchanged. He believed that the activity of the catecholaminergic neurons in the brain stem and this previously unrecognized ‘enhancer effect’ is responsible for the unique therapeutic benefits of deprenyl. Consequently, Knoll suggested doses of as little as 1 mg/day as optimal for disease-prevention and life-extending purposes.
Prof. Knoll ‘practiced what he preached.’ Since January 1989, at the age of 64, he took 1 mg Deprenyl every day. At the age of 92, he continued his daily consumption of what he referred to as a catecholaminergic activity enhancer (CAE) substance. I first discussed this with him at the IAS-sponsored Monaco Anti-Aging Conference in 2002. He revealed that although deprenyl’s activity as a MAO-B inhibitor was its first-discovered and most highly-promoted mechanism, he described its most important mechanism as a “catecholaminergic receptor sensitizer”--sort of like a; “metformin for the brain” (he winked at me as he said that, as I had just given a talk on the multi-hormone receptor-sensitizing effects of metformin).

Until his death on April 17th, 2018 at age 94, Prof. Knoll continued to maintain his emeritus position as a Professor at Semmelweis University, conducted ongoing research and wrote books and articles. His only limitation was that he traveled less internationally than he had formerly done. He commented slyly in his book that his ongoing “self-experiment (1 mg of Deprenyl daily) augers very well.”

References

FOREVER YOUNG

Phil Micans interviews Dr. Bill Lawrence

Dr. Lawrence has a Jurisprudence Degree (UCLA-Law), an MS in Psychology and a Ph.D. in Nutrition. Since 1990 his focus has been on slowing and reversing human biological aging. He has developed numerous science-based protocols focused on optimal aging for health professionals and individual clients.

Presently Dr. Lawrence is the administrator of two clinical studies to determine if human biological age can be reversed.

NB- Dr. Lawrence is chronologically 72. His ‘biological age,’ based on telomere length is 45.

Q. Dr. Lawrence, before we get into specifics regarding what you have achieved with your research and clinical studies, can I ask you why are you involved in anti-aging research? I’m asking because often these personal stories can be most enlightening.

A. It’s self-preservation. I am nearly 73, but unfortunately, my family genealogy records show, in the last two hundred years, no male lived beyond seventy. One could say; “I am in overtime!” (hopefully, not the sudden-death type!)

Most of my ancestors died between the ages of forty and sixty-five, including my father at age 65. In the last two years three of my siblings, all in their sixties died. When people greet me with; “How are you?” my response is always; “I’m doing great- I woke up breathing this morning!”

Q. Some authors use early photos of themselves. How recent is this photo of you?

A. Four months.

Q. Impressive, you certainly look closer to your telomere age and thank you for your personal story. I guess that the obvious question is- can human biological age be reversed?

A. It can, but I want to clarify this though, as I believe both ‘biological age’ and ‘age reversal’ are misnomers and I generally avoid using those terms. More accurately, we can now slow down and reverse the average ‘cellular age’ of humans which results in significant life and health-span extension.

Today there is a scientific consensus that cell aging, the number of times our cells can replicate (the Hayflick Limit), determines human longevity and it also drives age-related disease.
Q. So what causes cell aging?

A. There are numerous factors including lifestyle, diet, free radicals, glycation, pathogens, toxic environmental exposure, as well as ‘wear and tear.’ Telomere shortening is one of the primary causes. Methylation issues are also a recently discovered cause.

Telomeres are sections of genetic material at the end of our chromosomes.

Telomere function is to preserve cell integrity and prevent chromosomal ‘fraying’ as cells replicate. Cell replication is necessary for life and each replication results in telomere loss.

Eventually, with enough telomere loss, cellular function, and maintenance decline.

With enough cellular damage tissue repair slows, organs and systems deteriorate and become dysfunctional. Gene expression is then altered, resulting in age acceleration, vulnerability to age-related diseases, disability and finally, death. This cascade of inevitable events can be significantly delayed by restoring telomeres.

Q. Are there clinical studies which confirm the telomere theory of aging is an accurate explanation of why humans age?

A. Yes, there are several thousand studies referenced on PubMed confirming the relationship between telomeres and aging. Many of those clinical studies have shown the all-cause mortality rate of people with shorter telomeres was significantly higher compared to those with longer telomeres.

The Nobel Prize in Medicine was awarded in 2009 for the discovery of the telomerase enzyme and how telomeres protect chromosomes, which results in increased cell replication, and longer healthier lifespans.

Michael Fossel, M.D., Ph.D., who you interviewed several years ago, has written one of the most comprehensive books on aging, The Telomerase Revolution. In evaluating the various aging theories, he concludes: “Telomerase activation is the single most promising approach to reversing the effects of aging.” Dr. Fossel is also the author of the telomere textbook used in most American medical schools.

Q. Are there clinical studies which confirm the telomere theory of aging is an accurate explanation of why humans age?

A. Yes, numerous studies. For instance, a study supported by the National Institutes of Health reported in a study of over 100,000 people 65 or older, those with the shortest telomeres had a more than 20 percent higher risk of death in the following three years than those with longer telomeres.

Q. Are short telomeres associated with disease?

A. Yes. Many studies confirm people with longer telomeres not only live longer but experience less disease. PubMed has over 4,000 clinical studies referencing telomere length and disease. These include cancer, heart disease, Alzheimer’s and Parkinson’s disease, cognitive decline, COPD, stroke, arthritis, osteoporosis, etc.

A 2003 study published in The Lancet, reported mortality as a result of heart disease was over three times higher in individuals with shorter telomeres and eight times higher from infectious diseases.

The Journal of the American Medical Association (2010) reported an international team of doctors compared telomere length against instances of cancer in 787 patients. The patients with the shortest telomeres had three times greater risk of cancer than patients of the same age who had slightly longer telomeres. Short telomeres increased the risk of cancer and also seemed to make cancer more virulent. Short telomere length doubles the mortality rate of cancer.
Dr. William Andrews, one of the world’s leading authorities on telomerase biology, has written: “Almost every known disease can be attributed to the shortening of telomeres.”

Progressive telomere shortening is likely the primary cause of skin aging. It is possible management of telomere length could slow, stop and, possibly reverse skin aging.

**Q.** Is it possible to measure the length of a person’s telomeres?

**A.** Yes. Testing has improved over the years and a reasonable approximation of ‘telomere age’ or ‘cellular age’ is now possible.

A newborn has an average telomere length of 10,000 bps (base pairs). A young adult of 20-30 years has an average telomere length of approximately 8,000 - 9,000 bps. Humans lose approximately 35 or more base pairs per year. When telomeres reach a level of fewer than 7,000 or so bps, cell replication begins to be impaired, and the risk of a cascade of severe health issues increases together with an increased risk of death.

Specific telomere length is less important than changes in length over time. A person who loses 50 bps compared to a person losing 100 bps per year is aging much slower and is at a decreased risk of disease and mortality.

**Q.** Can telomeres be restored or ‘lengthened’?

**A.** Yes. The key to health-span and extended longevity is to activate an enzyme, telomerase, which restores telomere length.

American scientists erroneously claim they discovered the telomerase enzyme. They are still looking for a safe and effective way to activate it.

More than thirty years ago, Russian scientists discovered how to ‘turn on’ telomerase using specific short-chain amino acids, referred to as ‘Peptide Bioregulators.’ Their published results confirm that these Russian peptides activate the telomerase enzyme to ‘turn on’ relevant genes, stop telomere loss, and lengthen telomeres resulting in a longer and healthier lifespan.

**Q.** Are there clinical studies that confirm the Peptide Bioregulators can restore telomeres and reduce all-cause mortality?

**A.** Yes. Both Russian studies (2003) and the interim results of my American clinical study, Telomerase Activation Protocol (TAP), confirm the Peptide Bioregulators restore telomere length.

In the ongoing TAP telomere study, of 22 subjects with two or more telomere tests, 96% experienced lengthened telomeres over baseline while 4% maintained their baseline telomere length even though a year older at their secondary test. The average decrease in their ‘telomere’ or ‘biological age’ was 16.9 years over two years.

In the 1990s Prof. Vladimir Khavinson, Director the St. Petersburg Institute of Bioregulation and Gerontology conducted a human clinical study, published in Russia in 2003, to determine if Peptide Bioregulators could enhance the health and extend the lifespan of humans.

The accompanying graph reports on those results, (see figure 1).

In the Russian study, there were two groups of subjects based on age, ‘Elderly and Old’ (the Russians are not the most diplomatic). In each group, there was a ‘peptide’ group and a ‘control’ group of non-peptide subjects. In the ‘Elderly’ group (60-74 years of age) over twelve years, the control group (non-peptide) experienced higher all-cause mortality (44.1%) while the peptide group experienced significantly
less mortality (22.3%). In other words, the peptide group reduced death by fifty percent!

In the ‘Old Age’ group (75-89 years of age) the results were even more dramatic. The non-peptide control group, during the six years of the study, experienced all-cause mortality of 81.8%. The peptide group had significantly less mortality of 33.3%. In other words, the peptide group had a survival rate of 66.7% compared to 18.2% in the non-peptide group. (See Figure 2 that details the precise statistics).

Unfortunately, these studies are either ignored or generally unknown outside of Russia and eastern Europe. If a USA pharmaceutical company produced these results with a drug, it would be broadcast on every media in America and considered to be one of the most important scientific discoveries of all time!

In addition to the dramatic decrease in all-cause human mortality, the studies reported improvement of many biomarkers including memory and physical performance, bone tissue density, immune enhancement, and telomere lengthening.

**Table:**

<table>
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<tr>
<th>Group of patients</th>
<th>Indices</th>
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<td>Old people (75-89 years)</td>
<td>Initial mean age, years</td>
<td>80.2 ± 1.6</td>
<td>81.5 ± 2.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mortality rate in the course of 6 years, %</td>
<td>81.8</td>
<td>45.8*</td>
<td>33.3*</td>
</tr>
</tbody>
</table>

**Figure 2:** The statistical results of the study that was conducted over 12-years shows that patients who consumed the pineal and thymus peptide bioregulators, whether they started at age 60-74 or 75-89 lived significantly longer than the controls who only took multivitamins.

**Figure 1:** Overall mortality was significantly decreased in patients who took the pineal peptide bioregulator, and mortality was further reduced when patients combined both the pineal and thymus peptide bioregulators- as compared to the controls.
I understand you are engaged in a second biological-age clinical study having to do with epigenetics and methylation. So far, we’ve managed to publish two books about the technology, one scientific reference titled; peptides in the epigenetic control of ageing and the other a public booklet, by Dr. Marios Kyriazis titled; the peptide bioregulator revolution.

**Q.** What can you tell us about this new clinical study?

**A.** The new clinical study is the ‘Epigenetic Methylation Study’ (EMS), as measured by the Horvath Epigenetic Clock.

Dr. Steven Horvath is a biostatistician scientist at UCLA; he has developed a very accurate way of measuring biological age versus chronological age.

This is done by measuring methylation on various DNA sites linked to aging and specific genes. Prof. Horvath discovered hundreds of age-related DNA sites where methylation changes could be observed which in turn effect DNA and ultimately genes.

As we age, there are changes to our DNA caused by methylation. The result is methylation found at specific DNA sites turn off certain genes which are generally protective and turn on genes which promote aging. The scientific explanation is that chemical changes to cytosine—one of the four DNA bases, or ‘letters’ of the genetic code—make genes more-or-less active (on/off), and this is age-related.

The changes in a person’s DNA can determine whether the person’s body is aging unusually fast or slowly. Dr. Horvath tested this epigenetic clock on 13,000 blood samples collected decades ago, from people whose subsequent date of death was known. The results revealed that the ‘clock’ could be used to accurately predict mortality.

Dr. Horvath while acknowledging the clock accuracy has expressed his frustration because he, and other scientists, are unaware of any effective intervention to modify the ‘toxic’ methylation and reverse biological aging.
Our preliminary data suggest that Peptide Bioregulators can reverse the methylation aging process and “rewind the clock.”

A very simplistic way to think about all of this is an analogy to an automobile. Telomeres are like the tread levels on tires. The more tread the further you can travel. Longer telomeres allow for increased cell replication, about 40% more than the ‘normal’ Hayflick Limit.

More cell replication results in longer life and less disease. We know from Russian clinical studies, and three years of the American Telomerase Activation Protocol (TAP) clinical study, the Peptide Bioregulators can lengthen telomeres.

Methylation can be compared to the operational parts of an automobile (engine, transmission, fuel system, electrical, etc.) As these components wear out the operation of the vehicle is impaired—even with ample tread remaining on the tires.

We have preliminary evidence we can ‘repair’ the operational aspects with the Peptide Bioregulators which will result in less age-related methylation, which in turn will restore DNA to a ‘younger’ state.

The EMS study is now underway. We use Dr. Horvath’s technology licensed by UCLA, to test the subject’s baseline methylation levels which provide a methylation-based ‘biological’ age. We then create a Peptide Bioregulator protocol to reverse the methylation-based biological age.

Q. Another important question to ask is- have you seen any adverse side-effects with the peptides?

A. Peptide Bioregulators have been used for over thirty years in Russia, as well as the last four years in America, without any negative side-effects.

Q. In summary what do you anticipate the results of these two studies will have on human biological aging?

A. We know we can reset the telomere biological clock. We expect to do the same with the methylation clock. Our preliminary results suggest a 20-40% increase in human longevity and a significant reduction in all-cause mortality, death, and disability from heart disease, cancer, and many other diseases.

Q. In 2015, Dr. Fossel, wrote; “We are on the brink of an enormous leap forward, in which we will become capable of reversing the aging process in an obvious and striking way. We are about to not only cure and prevent age-related diseases but reset the aging process itself.” Now, it is four years later, so are we there yet?

A. Yes!

Q. I can appreciate that this is going to take a little time to sink in with a lot of people, even those who are heavily involved in the field. But right now, this does in my humble opinion represent an extraordinary possibility for a positive intervention in biological aging. Furthermore, this must be one of the most cost-effective approaches available today, for what is, after all, a cutting-edge protocol. How can folks get in touch with you if they want to enroll into your professional monitored program?

A. If people are interested in joining my program, they can get all the details via IAS by emailing: therapy@iasgroup.com

Q. Dr. Lawrence, I’d like to thank you very much for all your time today, we greatly appreciate health professionals like yourself who are pushing the boundaries, so that we may all live as-long-as possible, as healthy as possible.

A. As you would say Phil- Cheers! And, I look forward to both of us waking up tomorrow-breathing!
ORALTIDE PRO™ – THE LATEST PEPTIDE TECHNOLOGY FOR DENTAL CARE

By Leslie J. Farer

Oral health is essential to overall health, yet many overlook this important fact when devising an anti-aging protocol. Poor dental health can lead to a range of problems throughout the body including cardiovascular disease, cancer, arthritis, and other degenerative diseases that undermine the prospect of a long and healthy life. Although practicing good dental hygiene is of utmost importance, often more help is needed for teeth and gums to maintain their optimal integrity (as well as appearance).

Recent advances in dentistry have led to the development of the OraltidePRO™ line of products – concentrated mouthwash, and an intensive repair gel – to target two major issues in dental health: tooth decay due to erosion of protective enamel and gum recession due to periodontal disease. OraltidePRO™ products employ innovative peptide technology to treat these issues, allowing you to improve and maintain the health of your teeth and gums with products you can use at home.

In this article, we’ll cover enamel erosion and gum recession, their development and progression, and treatments for each, including conventional as well as cutting-edge techniques upon which Oraltide™ products are based.
The protective mineralized enamel covering teeth is the hardest substance in the body, even harder than bone. But in spite of its strength, it can't always stand up to years of wear and tear and degradation due to consumption of sugar-laden food and drinks. When enamel wears away, teeth become more susceptible to sensitivity, decay, and cavities. Unlike bone, the body doesn't have mechanisms to regenerate enamel; once it's gone it can't be replaced. However, as we'll see below, certain substances repair, or "fill in," small surface lesions that form as enamel erodes, thereby strengthening the structure of existing enamel and protecting teeth from further damage.

Enamel is primarily composed of the mineral hydroxyapatite (crystalline calcium phosphate, Ca10 (PO4)6 (OH)2). The main culprit in enamel loss is acid, specifically, the formation of organic acids from the metabolism of carbohydrates (such as refined carbohydrates, including sugar in sweets, soft drinks, and fruit juice) by bacteria residing in a substance called plaque, which is a sticky biofilm covering teeth. When exposed to low pH, hydroxyapatite crystals forming the structural backbone of enamel break down – literally dissolve – into calcium and phosphate ions in a process known as demineralization, resulting in the formation of lesions or crystal voids on the tooth’s surface. This degradation is temporarily reversible by a naturally occurring remineralization process in which calcium and phosphate ions are diffused from saliva back onto the tooth's surface and deposited into crystal voids to restore the lost structure.

Enamel remineralization and demineralization is an on-going dynamic process. Problems occur when the dynamic is shifted toward demineralization, which leads to permanent enamel loss and its consequences: tooth decay due to bacterial invasion deeper into the tooth, cavities, sensitivity resulting from the exposure of dentin (the layer below enamel), and further structural decline including cracks, chips, and fractures (not to mention unsightly tooth discoloration and chipped, cracked teeth). The good news is, there are steps you can take to favor remineralization, including dietary modifications limiting sugar and refined carbohydrates, regular dental checkups and biannual cleanings, and daily brushing and flossing, in addition to specialized treatments and products.
ENAMEL REMINERALIZATION TREATMENTS

Fluoride treatments and fluoride toothpastes and mouthwashes have been widely touted as effective in promoting the diffusion of calcium and phosphate ions back onto the tooth surface to fortify the enamel crystalline structure, resulting in the formation of fluoridated hydroxyapatite and fluorapatite, instead of natural hydroxyapatite. Although the resulting remineralized tooth surfaces are stronger and more acid resistant than the original teeth, critics believe Fluoride to be potentially toxic. According to the International Academy of Oral Medicine and Toxicology, Fluoride exposure at levels currently thought to be safe can cause damage to the cardiovascular, nervous, endocrine, immune, and skeletal systems, and has been linked to diseases such as Alzheimer’s, cancer, diabetes, and heart disease.¹

Recent advancements in dentistry for enamel strengthening (and alternatives to fluoride) encourage natural hydroxyapatite mineralization, include the compound casein phosphopeptide-amorphous calcium phosphate (CPP-ACP), a key ingredient in OraltidePRO™.

Casein phosphopeptides (CPP’s) are a group of peptides derived from milk protein casein with the ability to markedly increase the solubility and bioavailability of calcium phosphate to produce a form known as ‘amorphous calcium phosphate’ (ACP).² ACP is a precursor in the formation of hydroxyapatite, making it a valuable remineralizing agent. In the past, the use of un-complexed calcium phosphate for remineralization was ineffective due to its low water solubility resulting in low concentrations delivered to the tooth surface. But now, this new technology using CPP to facilitate high, even supersaturated concentrations of calcium and phosphate ions, has shown much success.

CPP-ACP binds readily to the surface of the tooth where it deposits a high concentration of ACP, (2-4) repairing lesions in enamel and protecting against acid erosion. Several studies show that ACP fights tooth decay in animals and humans (5,6) by increasing the resistance of enamel.

Mineralization of crystal voids to fortify existing enamel and prevent further damage is far preferable to more aggressive techniques that become necessary once enamel is past the point of lesion repair, such as bonding (in which resin is applied to the tooth with eroded enamel, then trimmed and polished) or crowns or veneers (which involve capping the damaged tooth).

When it comes to strengthening enamel and protecting against tooth decay and sensitivity, fluoride-free Oraltide-Pro™ is a great choice – they incorporate the proven mineralizing treatment CPP-ACP into dental care products you can use at home – safely.
GINGIVAL RECESSION - NOT JUST AN AESTHETIC CONCERN

Besides restoring and maintaining the structural integrity of enamel to prevent tooth decay, OraltidePRO™ also targets another major issue in dental health – gingival (gum) recession, a condition in which the immediate gum tissue surrounding teeth recedes or pulls back, exposing the lower part of the tooth and root. Gum recession is common, especially with age – it’s estimated that over 60 percent of the human population has it. Besides its unattractive appearance, gingival recession may be an indicator of deeper problems.

Gingivitis, the first step in the development of periodontal disease, is an inflammation of gum tissue caused by bacterial build-up in plaque. If caught in time, gingivitis can be treated and reversed with proper plaque control, (i.e., dental cleanings at least twice a year, daily brushing and flossing, and antibacterial mouthwashes); otherwise it may progress to the more serious periodontitis.

In periodontitis, the inner layer of gum and bone pull away from teeth forming deep pockets that harbor tissue-destroying bacteria as plaque spreads below the gum line (see figure 1). As the disease progresses, the alveolar bone, (a thin layer of bone that forms the sockets around the roots of the teeth) and connective tissue, including the periodontal ligaments that anchor teeth in place degenerate, resulting in loose teeth, and potentially permanent loss of teeth. Periodontal disease should be treated as soon as detected (preferably prevented in the first place), since besides causing gum, bone, and tooth loss, it may lead to systemic inflammation and a range of illnesses throughout the body, including cancer, heart disease, diabetes, arthritis, and others.

Besides periodontal disease, other factors leading to the development of receding gums include genetic predisposition, overly aggressive tooth brushing, (which causes damage to both enamel and gums), poor dental hygiene (i.e., inadequate brushing and flossing, and infrequent dental checkups and cleanings), teeth grinding and smoking.

Figure 1: Loss of gum and bone tissue in periodontitis: Ref. openi.nlm.nih.gov
TREATMENTS FOR GINGIVAL RECESSION

There are a variety of conventional and recently developed regenerative methods to alleviate gum recession.

First let’s look at the conventional treatments. In mild cases, dentists often perform a ‘deep cleaning’ procedure called ‘tooth scaling and root planing’ to remove harmful bacteria-containing plaque on teeth and below the gum line and smooth irregular root surfaces. For more advanced recession, surgery is often necessary to cover the exposed root. ‘Open flap scaling and root planing’ involves folding back gum tissue, removing bacteria and plaque from pockets, and then tightening and securing gum tissue over teeth to remove or shrink pockets. Soft tissue grafts are another commonly used procedure in which tissue taken from underneath the palate (roof) of the mouth, or from the palate itself is grafted to gum tissue surrounding a tooth’s exposed root. Although these conventional methods may slow the progression of periodontal disease by removing bacteria, reducing pockets, or covering the exposed areas with soft tissue grafts, they do not improve the overall health of the supportive tissue structures.

Now we’ll examine state-of-the-art methods that treat and even reverse gingival recession by actively promoting the regeneration of damaged or lost tissues.

In recent years, great strides have been made in a fascinating area known as regenerative medicine that uses techniques such as implanting a membrane or scaffold seeded with a patient’s own cells or applying tissue-stimulating proteins or other biologically active molecules to the affected area to jumpstart the body’s innate healing mechanisms for the purpose of rebuilding ‘from scratch’ functional tissue to replace damaged or lost bone or tissue. Regenerative medicine has multiple applications in rebuilding organs and tissues system-wide, such as skin, cartilage, kidneys, liver, etc., as well as periodontal supportive structures including bone and gingival tissue.

Several studies show that grafts engineered from various types of scaffolds and cell types are a viable, if not superior alternative to the soft tissue grafts discussed above. For example, in one such study, membranes seeded with patients’ own gingival-derived cells initiated the regeneration of receding gums. Tissue-engineered grafts were created by culturing a small portion of a patient’s gum tissue. Fibroblasts (cells involved in wound healing and tissue repair – we’ll see more on fibroblasts below) derived from the cell culture were then infused onto a membrane, which was trimmed to fit the affected root area and sutured to adjacent gum tissue underneath a ‘flap’ that completely covered the graft. Within weeks, the fibroblast-seeded membrane triggered the growth of new, disease-free gingival tissue in the area previously marked by recession.

In addition to tissue-engineered grafts, periodontal tissues can be reconstructed using growth factors, specialized signaling molecules that mediate wound healing and tissue repair. There are a variety of growth factor types such as vascular endothelial growth factor (VEGF), fibroblast growth factor (FGF), and transforming growth factor (TGF), that interact with cells and other growth factors to ‘orchestrate’ growth and repair processes such as cell proliferation, extracellular matrix production, and collagen synthesis. The extracellular matrix is a network of carbohydrates and fibrous proteins including collagen that surrounds cells to form a mesh or network providing structural support to tissues. During wound healing, fibroblasts (the cell type seeded onto the tissue-engineered grafts described above) migrate to the wound site, proliferate, and are stimulated by certain growth factors to generate the collagen-containing extracellular matrix necessary for new tissues.
These key processes – extracellular matrix and collagen production by fibroblasts in response to cell signaling by growth factors – are among the most important ones that researchers experimentally manipulate in order to repair damaged or create new tissues. Several studies have examined the use of growth factors to initiate tissue generation, including periodontal tissues. In vitro studies show that TGF-β (a subtype of TGF) stimulates collagen production and cell proliferation (necessary for tissue generation) in human and animal gingival fibroblasts and periodontal ligament cells. (9) In humans, a placebo-controlled clinical trial on 253 patients with periodontitis demonstrated that topical application of another growth factor, FGF-2 (an FGF subtype) to alveolar bone regenerated periodontal tissue that had been destroyed by periodontitis.10
ORALTIDE PRO™ PRODUCTS ARE BASED ON REGENERATIVE MEDICINE TECHNIQUES

These studies are among a multitude that demonstrate the power of regenerative medicine to rebuild tissues, including periodontal supportive structures. Now this potential can be harnessed in products that you can use at home as part of your routine daily dental care regimen.

Instead of a fibroblast-seeded membrane implant, or the direct application of growth factors to periodontal tissue to initiate regeneration, OraltidePRO™ works by an alternate, though similar mechanism. The products contain two peptides, palmitoyl pentapeptide-4 and palmitoyl hexapeptide-12, which stimulate gene expression of TGF-β and the resulting biosynthesis of the extracellular matrix and collagen by fibroblasts.¹¹ Rather than applying growth factors directly to degenerating periodontal tissues, as we saw in the clinical study above, OraltidePRO™ works by orally delivering peptide ‘stimulators’ of growth factors which spur the body’s own production of TGF-β, activating fibroblasts to proliferate and do their job of tissue reconstruction (in this case, gingival tissue) – a novel, as well as cost-effective and convenient approach to treating receding gums.

ABOUT ORALTIDE PRO™ PRODUCTS

The OraltidePRO™ product line consists of a concentrated mouthwash and an intensive repair gel. Each product contains the three peptides we’ve discussed: CPP-ACP (for enamel remineralization), palmitoyl pentapeptide-4 and palmitoyl hexapeptide-12 (for gingival regeneration) to simultaneously repair damaged enamel, relieve tooth sensitivity, prevent tooth decay, and rebuild receding gums.

The intensive repair gel also contains xylitol, a natural anti-bacterial and anti-inflammatory sweetener, as well as bicarbonate of soda and coconut oil.

The peptide active ingredients are produced using a patented hydrolysis technology and purified using membrane ultrafiltration. OraltidePRO™ products are manufactured from whole food grade raw materials without any harmful ingredients such as fluoride, alcohol, titanium dioxide or chemical bactericides (chlorhexidine, gluconic acid, etc.)
HOW TO USE ORALTIDE PRO™

OraltidePRO™ repair gel
Using it is like a tooth whitening kit, in that the gel is squeezed into a supplied gum shield and then applied onto the teeth. The shield should remain in place for 10-15 minutes. Afterward, the teeth can be brushed and rinsed as usual. This procedure only has to be undertaken once a week.

OraltidePRO™ concentrated mouthwash
It can be used once daily, diluted with just one ml of the mouthwash combined with 10 ml water and held in the mouth and circulated for 5 to 10 minutes.

Incorporating both products into your dental care regimen will have a synergistic effect. Also, practicing other critical aspects of good dental hygiene: use a soft-bristled toothbrush, floss daily, make regular appointments for dental exams and cleanings, don’t smoke, and consume a healthy diet (limiting or eliminating refined carbohydrates and sugars).

The OraltidePRO™ range is at the leading edge of dental care products employing the latest peptide technologies to restore and promote the integrity of the teeth and gums, which will positively impact not only your dental health, but also your overall health.

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By Will Block

Peter Attia, M.D., is a Canadian-American physician of Egyptian descent who works exclusively on longevity. He is currently writing a book on rapamycin. A Stanford/Johns Hopkins/NIH-trained physician, Dr. Attia is assisted by a team of researchers reading 1,000 papers per month out of the 100,000 new papers the U.S. National Library of Medicine receives each month. Peter feels that very little longevity news gets away from him and he plans to hire more researchers soon.

Peter Attia, M.D.

A PHYSICIAN FOCUSING ON THE APPLIED SCIENCE OF LONGEVITY

Starting out sending a small weekly email to a handful of friends in 2010, he quickly grew into a far larger mailing list including friends of friends, and eventually complete strangers. In 2011, Peter started sharing what he was learning on his old blog at EatingAcademy.com. While still of some concern, the subject of that blog has been principally superseded.

Today, what began as a personal blog about nutrition has grown into a sizeable site (peterattiamd.com): an ever-expanding core of content on exercise, nutrition, metabolic disease, and other topics related to the science of longevity.

His goal with this site is to translate the cutting edge of longevity science, (something roughly 0.01% of people are able to truly fathom) into something that is more genuinely understandable, and something that propels individuals to act, as a consequence of data garnered by his research team and through extensive interviews with leaders in longevity.

Peter’s approach focuses on delaying the onset of chronic disease (aging), while simultaneously improving quality of life (healthspan). To accomplish this, Peter’s practice applies nutritional biochemistry, exercise physiology, sleep physiology, the study of lipids, pharmacology, techniques to increase distress tolerance, and the study of hormones. His favorite subject these days is a drug, which he consumes weekly, specifically rapamycin. Peter’s dosage sweet spot is 2 mg to 5 mg of rapamycin, every 5 to 7 days.

“... across about a billion years-worth of evolutionary animal models, everything from yeast to worms, fruit flies to mammals, rapamycin seems to universally increase the length of life.”

Rapamycin binds to a complex called mTOR (mechanistic target of rapamycin) found in our cells and inhibits its activity. Among the many things that mTOR does (when inhibited), is to regulate autophagy causing cells to eat themselves. In this process, dysfunctional cells (like cancer cells) tend to be “eaten” first. In a sense, turning down the negative of mTOR mimics what happens to the body in a nutrient sparse environment. It is quite like what an individual can achieve by limiting the amount of food one eats, while avoiding malnutrition.
AUTOPHAGY NOBEL PRIZE

The Nobel Prize in Physiology or Medicine 2016 was awarded to Yoshinori Ohsumi. The Nobel Laureate discovered and explicated the mechanisms underlying autophagy, a fundamental process for degrading and recycling cellular components. *Rapamycin induces autophagy.*

The word autophagy originates from the Greek words auto-, meaning “self,” and phagein, meaning “to eat.” Thus, autophagy denotes “self-eating.” This concept emerged during the 1960s, when researchers first observed that the cell could destroy its own contents by enclosing it in membranes, forming sack-like vesicles that were transported to a recycling compartment, called the lysosome, for degradation.

Difficulties in studying the phenomenon meant that little was known until, in a series of brilliant experiments in the early 1990s, Yoshinori Ohsumi used baker’s yeast to identify genes essential for autophagy. He went on to clarify the underlying mechanisms for autophagy in yeast and showed that similar sophisticated machinery is used in our cells.

OHSUMI’S DISCOVERIES LED TO A NEW PARADIGM

His discoveries opened the path to understanding the fundamental importance of autophagy in many physiological processes, such as in the adaptation to starvation or response to infection. Mutations in autophagy genes can cause disease, and the autophagic process is involved in several conditions including cancer and neurological disease. Rapamycin is an autophagy inducer.

Dr. Attia doesn't foresee endless horizons. As is true for another group of scientists highly interested in rapamycin, for “research” purposes, Attia does not favor “forever,” nor does he believe that life favors forever. He is not an immortalist. Too bad!
In a recent paper (2014), it was reported that rapamycin extended the median lifespan 23% in male mice and 26% in female mice. Rapamycin, a drug used as an immune-suppressant in the treatment of organ transplant patients, may be the most potent life-extension drug currently available. Adding to that, the practice of rapamycin in anti-aging medicine is just getting started.

Mikhail Blagosklonny, an M.D. doctor and a Ph.D. scientist at Roswell Park Cancer Institute in New York, has been the most notable and vocal advocate of the use of rapamycin to extend human lifespan. While rapamycin has adverse side effects in humans who take it daily for immuno-suppression, recent research has found that pulse dosing—perhaps once a week—may confer most of the anti-aging benefits without any significant adverse side effects.
WILL RAPAMYCIN FIGHT AGING IN HUMANS AND EXTEND LIFESPAN?

Unfortunately, clinical trials of rapamycin for life extension purpose are unlikely to happen any time soon, but some people would like to find out. Alan S. Green, M.D., who practices medicine in New York, is among them. Beginning in early 2016, he began to take rapamycin himself, along with metformin, an angiotensin blocker, and aspirin.

Based upon empirical medicine principles, Dr. Green decided that rapamycin at 6 mg once a week would be an aggressive treatment and 3 mg once every 10 days would be a conservative treatment. His choice was the aggressive treatment. In January of 2016, he began the rapamycin-based Koschei ["Deathless," A Slavic Legend of Immortality," from mythic Russia] formula with intent to take it for one year; in what could inoffensively be called a "proof-of-concept" experiment. He didn't have to wait one year, for by 4 months—the results were "miraculous"—so he kept on going.

Dr. Green lost 20 pounds, his waist-line went from 38 inches to 33. He bought a pair of size 32 jeans and didn't have to wear joggers any more. Dr. Green could walk 5 miles a day and ride a bike up hills without any hint of angina. Creatinine went from elevated to normal and fasting blood sugar went down. At the time of this interview, it’s now over one year and Green still feels great. He’s also had no mouth sores, the most common clinical side-effect. For the good doctor, rapamycin is the world’s greatest medicine!

Moreover, Dr. Green is the author of another study using both an AGE (advanced glycation end-products) diet and intermittent rapamycin, just as above (pulse dosing), for three years following Blagosklonny’s most recent paper, and for himself, Green’s most recent paper described above. It has been known since the 1930s that caloric restriction extends lifespan and slows aging.

RAPAMYCIN OFFERS CALORIC RESTRICTION

*Rapamycin could be characterized as caloric restriction in a pill.* In a mouse study, rapamycin had similar effect in aged mice as 40% caloric restriction in restoring the dysfunction of old hearts to normal function of young hearts. The effect of caloric restriction is to decrease mTOR indirectly; the effect of rapamycin is to decrease mTOR directly.

On one hand, aging may be defined as an increase in the probability of death. If a drug extends lifespan, that drug is slowing aging. Aging is the main risk factor for age-related diseases such as heart disease, cancer, Alzheimer’s disease, or blindness. Slowing down aging and staying young is the goal of Rapamycin Medicine.

Rapamycin has only one action; it targets or binds to mTOR and thereby lowers mTOR activity. mTOR and rapamycin are related as a key to a lock. As previously stated, mTOR has been conserved through 2 billion years of evolution. mTOR functions as the central hub of the cell signaling system, the command and control center of the cell.

Rapamycin was approved in 1999 as a prescription medicine to be used in high daily dose as an immunosuppressant in transplant medicine. Used in this manner, rapamycin has very limited benefits aside from use in transplants as it has too many side effects to be of general interest. Miraculously, when used as a non-immunosuppressant, low dose, weekly medication, rapamycin is transformed into a silver bullet, with minimal side effects and extraordinary benefits. For rapamycin, less is more.
RAPAMYCIN IS TO PENICILLIN AS...

Rapamycin, like penicillin, was the product of biologic warfare between bacteria and yeast. Like a mirror image, penicillin—discovered in 1928—was made by yeast to target bacteria, while rapamycin, found in the soil of Easter Island (Rapa Nui, the local name) in 1965, was made by bacteria to target yeast (the other way around). The bacteria of Easter Island targeted mTOR, the command and control center of the yeast cell. However, mTOR, the substance targeted in this never-ending war between yeast and bacteria, is the command and control center of not just yeast. It takes that role for every living thing on planet earth. mTOR is, (in essence) the “secret” of how life is organized within the cell.

Rapamycin has another similarity to the discovery of penicillin. In the cancer sections of his papers, Mikhail Blagosklonny states; “Rapamycin could be used for extension of healthy lifespan and prevention of age-related diseases by slowing down the aging process. This may become one of the major breakthroughs in medicine since the discovery of antibiotics.”
MTOR AND AGE-RELATED DISEASE

mTOR is a complex large protein located in the cytoplasm of the nucleus. It operates in close communication with the nucleus of the cell. It is always there. The nucleus contains the DNA that comprises the blueprints for making each individual protein. mTOR signals the nucleus: make this protein! mTOR senses what is happening within the cell and then signals the nucleus how to respond. The nucleus then responds by providing the blueprints to make the specific different proteins.

THE WATERSHED YEAR

In the developing and growing animal, mTOR functions to near perfection, mTOR’s primary purpose is growth and development.

mTOR has been conserved for two billion years for good reason. However, in the older animal, mTOR has a dark side. This dark side was first recognized by Mikhail Blagosklonny.

The year 2006 was a watershed year in biology, hosting one of the most important papers since 1859, the year Charles Darwin published, “On the Origin of Species.” The great biologist’s paper is at the foundation of evolutionary biology.

The year 2006 paper was “Aging and Immortality: Quasi-Programmed Senescence and its Pharmacologic Inhibition.” In his research, Blagosklonny postulated that aging was a disease process caused by overactive mTOR.

Moreover, overcharged mTOR was also responsible for most age-related disease. Aging was merely the accidental result of another program that was the continuation of the essential growth program.

Aging and age-related disease are two sides of the same coin, the same disease process. Furthermore, mTOR could be controlled with rapamycin. Mankind has in hand the ability to ameliorate aging and age-related disease.

In summary, the problem for the old animal is that mTOR is at too high a level after growth and development have stopped. Elevated activity of mTOR is at the very center of aging and age-related disease. What the old animal needs is a kind of software upgrade (mTOR 2.0) for the older animal. Nature and evolution never provided this fix; however, rapamycin is the pharmacologic fix for the older animal.
Nonetheless, all drugs have some side effects.

If you think that aging is not a disease and rapamycin is only for prevention, then you might say rapamycin should not be used.

On the other hand, if you have felt the sting of aging and believe rapamycin is treating a disease which you very much already have, then you are far more inclined to think the risk of side effects is justified.

For the past 2000 years, aging has been considered a natural process—not a disease. The reasoning was, everybody gets aging and if everybody gets it, then it is natural. This is the accepted view of orthodox medicine and the position officially endorsed by the FDA.

Disease is defined as: “A disorder of structure or function in a human, animal, or plant, especially one that produces specific signs or symptoms or that affects a specific location and is not simply a direct result of physical injury.” Disease causes pathology.

Dr. Green considers aging to be not just a disease, but “the mother of all disease,” a chronic progressive disease with 100% incidence and 100% fatality. As does Mikhail Blagosklonny!

The general rule in medical treatment is that medicines used to treat a disease that somebody has, i.e. cancer, can have side effects; but a medicine used for prevention should have no side effects.

I consider intermittent rapamycin to have few side effects, a safe drug in the general world of dark drugs.

AGING CAN BE DIVIDED INTO EARLY AGING AND LATE AGING

Early aging includes the pathology in the 50-99 age group and the increasing susceptibility to age-related disease. Late aging includes the pathology in the 100-110 age group. Late aging inexorably leads to death. There is no current prevention or treatment for late aging and late aging is outside the scope of Dr. Green’s practice.

The use of the term “aging” in an earlier paper only refers to “Early Aging.” Blagosklonny uses the term, “post aging syndrome” to refer to late aging. While Green believes that “early aging” is multi-factorial, he also believes that increased activity of mTOR is postulated as a very significant cause of early aging and a significant and treatable factor in the cause of the common age-related diseases.

Green’s office treats the disease of increased activity of mTOR. The objective of such treatment is to ameliorate and prevent the vast amount of diseases associated with elevated mTOR. Treatment is directed against the disease of aging, and age-related diseases are connected to aging as smoke is to fire.
EXTREME RAPAMYCIN ENTHUSIAST

Blagosklonny first entered the cancer treatment field in the early 2000s, when he realized the same qualities that made rapamycin effective at slowing tumor growth might also help it slow the aging process.

He became so convinced of rapamycin's potential, and its safety, that he tried it himself. Some people asked him whether it is dangerous to take rapamycin. Blagosklonny says, “It's more dangerous to not take rapamycin than to overeat, smoke, and drive without a [safety] belt, taken together.”

Many colleagues have regarded his advocacy as a bit over-the-top. When Blagosklonny submitted papers to major journals making these arguments, they were brutally rejected. “Sometimes, the reviewers would call me names,” he says.

That started to change in 2009, when a large National Institutes of Health-funded study established that rapamycin and its derivatives helped mice live longer. The NIH scientists started mice on the drug at 20 months, or late-middle age in mouse terms, (mice typically live two to three years). Male mice on rapamycin lived 9 percent longer. The females' life span was extended by 14 percent. This is roughly the equivalent of giving 60-year-old women a pill that would enable them to see their 95th birthday.

IS AGING A DISEASE?

It really doesn't matter, because aging is already treated using a combination of several clinically-available drugs, including rapamycin. Whether aging is a disease depends on arbitrary definitions of both disease and aging. If you consider treatment, aging is a deadly pre-disease, despite being a normal continuation of normal organismal growth. It can be successfully treated, thereby delaying classic age-related diseases such as cancer, cardiovascular and metabolic diseases, and neurodegeneration.

While Mikhail Blagosklonny has published on a broad range of topics on cancer, his interest lately seems to focus on the link between cancer and aging and finding links and similarities in their pathways. Having dedicated many years to studying the molecular and cellular biology of oncogenes, genes capable of transforming a cell into a tumor cell, and tumor suppressors, Blagosklonny has helped reveal that rapamycin, a cancer drug, can help extend the longevity of life.

References

5. Blagosklonny MV. Disease or not, aging is easily treatable. Aging (Albany NY). 2018; 10:3067–78. 10.18632/aging.101647
A BREAKTHROUGH FOR CATARACT

Can-C™ eye-drops are the original™ brand-developed by Innovative Vision Products (IVP). This group were the first to research, publish and prove how eye-drops can reduce and even eradicate cataract. Accordingly there are active US and EU patents (and others pending) on this unique and special product.

Unique formula:
Can-C™ eye-drops are the formula from the original published human trials. They contain a purified and racemized form of n-acetylcarnosine (made in Japan); this natural dipeptide has potent anti-glycating and anti-oxidant properties that prevents lipid peroxidation. Note that the formula is important— it’s not all about the n-acetylcarnosine; the specific carrier agents and their purity are also important. If you look at the Can-C™ formula you will see differences to the copycats, (remember it is only Can-C™ that is patented in recognition of the original work). If you want the best possible results in the fastest possible time, then choose Can-C™ to deliver them according to the clinical trials.

Clinical trial:
Patients placed two-drops of Can-C™ into their eyes twice daily for a 6-month period, the outcome was:
- 90% saw an improvement in their visual acuity.
- 88.9% of patients showed improvement in the clarity of their lens.

There have been numerous reports of cataract shrinkage and even disappearance with documented evidence that Can-C™ eye-drops remain effective (and safe) more than 24-months later. The most commonly expressed initial reports are that glare is significantly improved, (for example night driving is much safer) and color perception is enhanced.

Improving eye-sight:
More evidence is mounting that Can-C™ is efficacious for many conditions including:
- Cataracts (particularly the senile version)
- Glaucoma
- Presbyopia
- Corneal disorders
- Eye strain
- Ocular inflammation
- Blurred vision
- Vitreous opacities and lesions
- Diabetes mellitus complications
- Contact lens users
- Dry eye syndrome

Of special interest may be to persons who wear contact lenses. This is because Can-C™ inhibits the accumulation of lactic acid and therefore contacts can be worn for longer periods without pain.

We have also received reports that Can-C™ not only aids dry-eye syndrome with its lubricants, but that Can-C™ helps to unclog proteins from the lacrimal ducts, thus releasing more natural tears onto the eye.

In a similar way, it is also believed that the unclogging of proteins in the eye’s drain, (the Schlemm valve), helps to reduce intraocular pressure and thus aids glaucoma.
Deprenyl is also known as selegiline, it was created in the 1960s by Professor Joseph Knoll, principally as an aid to Parkinson’s patients—because deprenyl has a significant benefit to improve dopamine levels in the brain.

Significant longevity studies
Professor Knoll’s experiments with rats produced some of the most incredible longevity benefits. When fed deprenyl in their food, they lived longer than those that were not. After the last non-treated rat died, the first of the deprenyl rats hadn’t! These results were in another study conducted from research by, Dean, Fowkes and Morgenthaler—published in the book, ‘Smart drugs & nutrients’. It highlights that the loss of dopamine in humans with age, can be mapped against the development of Parkinson’s and even death.

Deprenyl has been expressed as a MAO-b inhibitor. Preventing the enzyme monoamine-oxidise type-b from destroying dopamine, ergo leading to its greater availability in the brain.

The inhibition of the more common MAO-a can be problematic, leading to something called ‘the cheese effect,’ not a side effect of deprenyl, although it should be noted that dopamine can inhibit type-a, usually at very high doses of 20mg. Professor Knoll has noted that there is another significant action of deprenyl and this is the raising of PEA levels. PEA is a catecholamine activity enhancer that raises norepinephrine levels, it’s a significant attention agent that is behind the primary mechanism of the famous Eugeroic drug—modafinil (Provigil). Read professor Knoll’s books- ‘The brain and itself’, or ‘How selegiline/deprenyl slows brain aging.’

Avoid overuse since it can lead to what may appear to be an oppressive behaviour, as others around you are not focused and ‘on the ball’ as you! We recommend breaks from deprenyl use.

Some advocate one week off in the month and other use it during the weekdays but not at the weekends.

Doses are based on need and age. Parkinson’s patients will require large doses. A person wanting to improve their cognitive performance may want to consider 1mg to 3mg per day, with occasional breaks. These doses do not take into account synergy with other dopamine enhancing agents and persons using anti-depressants should consult with their physician. Deprenyl tablets are provided in 5mg form (Jumex), some like to take ½ to 1 of these tablets 3 times a week. The use of the deprenyl liquid (Dep-Pro) is particularly attractive for those using deprenyl to generally support, portect and improve neurological function, since 1 drop = 1mg, the liquid can be dosed very precisely by placing those drops into a cold drink. Avoid use in the late evening to prevent any sleep disruption.
BECAUSE NOT ALL MELATONINS’ ARE CREATED EQUAL

Melatonin is produced by the pineal gland at night to regulate our circadian rhythm, (sometimes called the sleep wake cycle). As we age the amount of melatonin we produce reduces resulting in many older people sleeping less and having a lower quality of sleep. Our melatonin has been formulated by the world’s foremost melatonin expert Dr. Walter Pierpaoli, his Melatonin Zn Se, or MZS™, is totally unique since it is designed to mimic the natural night peak of melatonin- to leave you feeling refreshed and alert the following day.

What does Melatonin do?

Melatonin is vital to protect our hormonal system, regulate immunity and repair our body’s cells. It is commonly used by shift workers and also to treat jet lag and age related sleep disorders. Melatonin is an extremely effective antioxidant; in fact on a molecule to molecule basis; melatonin has proved to be significantly more efficient in neutralizing toxic hydroxyl-radicals than the two well-known free radical scavengers, glutathione and mannitol. Its effect on longevity is well documented. Experts believe melatonin has a positive effect on aging.

Age related macular degeneration (ARMD) comes in two forms, wet and dry and is a notoriously difficult disorder to treat and is linked to blindness. A 24-month study, (published in NY Academy of Science, 2005, 1057:384-392) on 100 patients showed that after 3 months, the majority of patients taking 3 mg of Melatonin Zn Se nightly had halted the progression of their age related macular degeneration and at 6 months many showed reversal of their ARMD. Remarkably this was true for both the wet and dry forms!

Why is Dr. Pierpaoli’s MZS™ more effective than other melatonin supplements?

Firstly it is of pharmaceutical quality at a dose of 3mg. Secondly, it contains the synergistic ingredients of selenium and zinc. Thirdly and most importantly- it is designed to release at a very specific time. Dr. Pierpaoli’s research led him to perfect a formula that exactly mimics the pineal gland’s release of melatonin. MZS is the only melatonin supplement to follow nature’s own night peak. Take half to one 3mg tablet at bedtime only; do not take more than two tablets. By taking MZS™ between 9pm and 11pm you will create a night peak between 1am and 3am, this is the most natural and normal time to have the highest melatonin levels.

MZS is much more than a sleep aid and melatonin has many published benefits. MZS comes with the endorsement of Dr. Pierpaoli. If you’ve tried other melatonin and didn’t notice a significant effect, then we highly recommend you try Dr.Pierpaoli MZS for a superior experience.
NATURAL ESTROGENS AND PROGESTERONE FOR WOMEN

In this featured section we are focusing on the use of natural estrogens and progesterone for women, normally utilised to aid the menopause. IAS carries a wide range of bioidentical hormones - a term that means ‘natural to and in the body.’

When hormone replacement therapy (HRT) was developed in the 1920s, estrogens had to be derived from horse urine because a laboratory solution was too difficult or expensive to synthesize. Facts pointed out by Dr. Wright in his best-selling book ‘Stay Young & Sexy’. Estrogens can be easily produced now. Some people believe that the known side-effects from ‘traditional HRT’ are due to the fact that the hormones given are not correct.

Introducing Esnatri

Esnatri™ is our bioidentical triple estrogen cream. One of the best biodentical estrogen creams available. It comes directly from the work of Dr. Wright who has shown that the majority of women produce estrogens in the ratios of 90% estriol, 7% estradiol and 3% estrone.

Most tri-estrogen preparations attempt to replicate the human hormones estriol, estradiol and estrone, apply them in the ratio of 80:10:10, while some even entirely over-look estriol, claiming it is a weak estrogen. But, women naturally produce high levels of estriol and it is considered to have anticarcinogenic effects.

The Esnatri cream can be applied by daily rotation to your neck, upper chest, breasts and behind your knees, or inner thighs. A typical starting dose is 2mg. Start from day one (of what would have been the start of your menstrual cycle) and continue until day 25. You should stop for five days, before repeating the application at the start of the next menstrual cycle. During these last few days, the estrogen receptors are being allowed to ‘rest’ as they have been accustomed.

Combining Estrogen with Progesterone

Progesterone is the counterbalance to estrogens. Women can significantly decline in estrogen levels during menopause – they rarely reach zero production levels, whereas progesterone can sometimes not be measured at all in elderly women. It is also the low progesterone that most significantly impacts bone strength, leading onto osteoporosis. There are numerous reasons to ensure that progesterone is also taken alongside an estrogen therapy. IAS provides a 5% strength natural progesterone cream. Typical doses are 25mg to 30mg of progesterone applied on day 10 and continuing to 25. The start date varies according to the usual timing of your ovulation. As with the Esnatri cream, sop for the last 5 days of your cycle so that the estrogen receptors have their accustomed ‘rest’ period. Remember, your hormone replacement therapy should be overseen by a physician and should not be undertaken if you have undergone cancer treatment.
OXYTOCIN FOR PASSION AND SEX

Oxytocin is a hormone produced by the hypothalamus, excreted via the pituitary gland. Its orthodox medicine role is to help women give birth, since the large dose that’s injected helps to relax the uterus and alleviates the passage of the child. Dr. Thierry Hertoghe’s book, ‘Passion, sex and longevity, the oxytocin adventure’ highlights that it has many other roles too.

The Love Hormone

Oxytocin has been dubbed ‘the love hormone’. It can induce feelings of bonding and care. Its measurements have been taken between lovers, friends, relatives, parents and their children etc. It has been noted that oxytocin levels are higher when they are in their presence. Mothers naturally bond with their children, but even men, (especially those who experience the live birth), express their emotions as wanting to care and protect their offspring. These effects may be attributable to the release of oxytocin hence triggering the bond. However, psychopaths are notoriously low in their oxytocin levels, which may be a cause of their uncaring feelings towards other humans.

The pain and orgasm connection—Fibromyalgia can be a very debilitating disorder with a lot of pain, sometimes constant for those who suffer with it. In women, it was noted that when they were experiencing an orgasm they felt no pain at all. Women undergo a burst of oxytocin during orgasm. Trails were undertaken to see if oxytocin supplementation could alleviate the pain of Fibromyalgia, there was some success, but the side-effect noted was that those women now enjoyed multiple orgasms!

The effects of Oxytocin

Dr. Hertoghe explained that some will not feel the effects of oxytocin. For two reasons, (if we consider that the dose is correct for that individual). Firstly, some people are ‘low’ in their principal sex hormone, so if a man is low testosterone, or if a woman is low estrogen, it is possible that oxytocin will not elicit its full potential in those persons. The other issue could be low vasopressin, vasopressin is a counterpart to oxytocin, produced and released via the same glands. In cases of vasopressin deficiency, the patient may enhance the oxytocin experience by adding one or two sprays (10IU) each of vasopressin via the Vaso-Pro nasal spray.

Doses are very dependent upon its use. For social or sexual enhancement, 5 IU to 10 IU is a ‘typical’ dose. Dr. Hertoghe reduced the doses that he recommends in his books, (Transmitted via personal conversation to me). Currently IAS is providing Oxy-sub in 20 IU trouches (a soft sublingual tablet). These can be cut into half or a quarter for a dose of 5 or 10 IU and should be placed under the tongue and allowed to melt. The other option is Oxy-Pro which is applied intranasally delivering 10 IU per spray.
SPOTLIGHT

THE DISCOVERY OF GENE SWITCHES IN FOOD

Today Professor Vladimir Khavinson is the President of the European Academy of Gerontology and Geriatrics, but in the 1980’s he was a Colonel in the Soviet Union military medical corps. At the time, he and his team were approached by Kremlin officials, they wanted them to find a way to protect their troops from a myriad of problems; issues such as radiation for submariners in nuclear submarines to troops that may be blinded from known, (but thankfully unused) new weapons such as battlefield lasers.

A former Soviet military secret!

What their research uncovered - that was used for two decades on many thousands of men and women - was a remarkable link between short chain peptides and DNA. This former military secret is now available to the public as peptide bioregulators. Their published research has identified that each organ / gland / tissue uses a highly specific short chain peptide, obtained from food, to act as a ‘short-cut’ to initiate protein synthesis. These peptides, unlike proteins, can enter the blood through the stomach. Through a comprehensive list of patents and even copyrighted PowerPoint slides, the Russian research group have shown that each of the concentrated peptide bioregulators so far examined, interact with particular strands of DNA - effectively and very specifically activating repair and regenerative processes.

This is a remarkable story since what we are describing here are peptides that act as individualised gene switches. To date, they have been tested for many years on thousands of individuals, without report of any serious side effects or contraindications. We believe that they could be set to ‘out do’ stem cells. Why? Because this peptide therapy is relatively cheap, highly specific, can be taken orally and doesn't require any suppression of the immune system to operate fully (as stem cells do).

Oral material from the trials

The peptide bioregulators available via IAS are the bovine originals; sourced from carefully chosen Danish calves and processed through pharmaceutical processes and filters. They are not the synthetic versions which have not been studied/ proven. Peptide bioregulators act as they sound-to regulate; for example, Thyreogen® the thyroid peptide would increase thyroid activity if it were too low, but decrease if it were too high.

Dosing

Doses are very dependent upon the need and unlike hormones these peptides do not have to be taken every day, hence making them a cost effective regime. A typical/ average use could be considered as follows:

- Start with an intensive course: 2 capsules once a day for 30-days.
- Thereafter use 2 capsules once a day for 10-days, repeat every 2, 3, 4 or even as little as 6-months.

The story of the peptide bioregulators is a remarkable one and we recommend that you to read the articles and interviews and see the video on the IAS website.
PIRACETAM, THE ORIGINAL NOOTROPIC

Smart drugs and nutrients, or to give them their correct medical terminology- nootropics, are agents that can not only improve conditions of senile dementias, but in recent times have become popular for older individuals to improve their mental and cognitive processes.

It was Ward Dean, M.D. who highlighted these facts through his very popular ‘Smart Drug’ series of books in the 1980s, since then the term ‘smart drugs’ has become mainstream.

Piracetam, the original nootropic

The smart-drug we focus on here was in fact the first, developed as it was by Dr. Giurgea for UCB laboratories in Belgium in the 1960s. Originally it was designed to assist with travel and altitude sickness, but shortly afterward individuals realised that piracetam had positive cognitive enhancement effects.

Piracetam is a cognition agent that has been used successfully to treat a wide range of conditions, for example it has been shown to increase a person’s attention levels and improve memory and intelligence. Piracetam can help to slow down ‘senile involution’, dementia and Alzheimer’s disease. In tests and trials, piracetam induces significant improvement to memory consolidation and recall in those suffering from ‘age-associated memory impairment’.

Piracetam has also been used to improve patient’s recovery from strokes, particularly improving post stroke speech impairment (aphasia). Another use has been in cases of acute and chronic cerebral ischaemia, (decreased blood flow to the brain). Using piracetam has restored speech and the use of limbs in these patients; it has also increased neuronal activity in the brain when measured with EEG.

For regular individuals, piracetam has been shown to enhance idea creation and the ability to ‘see things through,’ in other words to have ideas and being them to fruition. The level of clarity piracetam creates is often described/ perceived as; “the fog has lifted.”

How does piracetam work?

Piracetam’s key and unique method of action is upon the Corpus Callosum, the region of the brain that links the two hemispheres. Most experts believe it is the key that gives piracetam users the ability to channel greater brain potential by connecting the logical side of the brain with the creative side more effectively.

What are the doses of piracetam?

A common dose is 800mg tablets three times a day, then lowering to 800mg twice a day after the first month. The effects of piracetam can be enhanced if taken concurrently with centrophenoxine or hydergine. Side effects are minimal and seldom experienced, but should you experience nausea or a headache then it is usually caused by an overdose. In which case reduce the dose and build up more slowly.
FOR THE HYPOTHYROID EPIDEMIC

Dr. Broda Barnes in the 1970s estimated that 40% of the adult population was deficient in thyroid hormones; he published this statement in his excellent book ‘hypothyroidism, the unsuspected epidemic.’ Since then, pupils of Dr. Barnes, such as Dr. Richard Wilkinson, have suggested that this figure could be even greater now!

This is important because the thyroid gland is of pivotal importance to our overall health, but like the majority of hormones, as we age the production of thyroid hormones decline. This lack of thyroid function is the root cause of a wide variety of age-related health disorders. Ergo, supplementation with a synthetic, or a natural thyroid can have a significant positive effect on a wide range of age-related problems.

The importance of the thyroid gland

The hormones produced by the thyroid control the body's metabolism - the rate at which it burns calories for energy. It also controls the body's utilization of fat, so a decline in the secretion of hormones from the thyroid gland, (known as hypothyroidism) can result in wide range of symptoms such as poor concentration, confusion, memory problems, cold hands and feet and weight gain.

Another serious condition which can be caused by and result from an underactive thyroid are painful musculoskeletal issues that affect tendons, muscles and ligaments.

Choosing between synthetic and natural thyroid supplements

IAS stocks a comprehensive range of both synthetic and natural thyroids, although we advocate the use of a natural supplement over a synthetic, this is because products such as Armour® are of a porcine origin, so they naturally contain the full spectrum of T1, T2, T3 and T4 thyroid hormones, (note the bottles only list the amounts of T3 and T4 because very few physicians are familiar with T1 and T2).

Natural desiccated thyroids are measured in grains; with one grain being equivalent to approximately 60 mg. IAS carries doses from ¼ grain to 2 grains, with brands including Armour®, ERFA® and Nature®. IAS also provides synthetic T3 in 20 mcg and T4 in 100 mcg tablets.

Thyroid supplements provide potent antiaging protection. Many aging individuals can benefit from taking a thyroid supplement because this remarkable hormone has such a profound affect across so many different conditions. Many antiaging physicians consider thyroid support an essential part of any serious attempt to improve a person's health-span and longevity.
www.antiaging-systems.com is your comprehensive resource for information about all the leading commercially available antiaging, preventative and regenerative products and therapies available today. Visit www.antiaging-systems.com and find articles, videos, audio-files, all referenced with a guide of where to obtain your needs.

Currently the site covers topics related to all the following products.

**BOOKS**

- Atlas of Endocrinology
- Great Teeth for Life
- Passion, Sex & Oxytocin
- Physician Hormone Handbook V2
- Cataract Cure
- Melatonin, the Key of Life
- Peptides in the Control of Ageing
- Reversing Physical Aging V1
- Eyesight Saviors
- Natural Skin Cancer Treatments
- Peptide Biomarker Revolution

**DIAGNOSTICS**

- Bio-Clip™ CUFF
- Foodsafe®

**GHRPS**

- GHRP2 (GHRP2-Pro™)
- Sermorelin (Serm-Pro™)

**HORMONES**

- Aldosterone (Aldo-Pro™)
- HCG (HCG-Pro™)
- MSH2 (MSH2-Pro™)
- Progesterone (Progest-Pro™)
- TRH (Abaris™)
- DHEA (DHEA-Pro™)
- Hydrocortisone (Hydrocort-Pro™)
- Oxytocin (Oxy-Pro™)
- Vasopressin (Vaso-Pro™)
- Estrogens (Esnatri™)
- Melatonin (MZS™)
- Pregnenolone (Preg-Pro™)
- Thyroid (Armour™ etc.)

**NUTRITION**

- 1st Line™ (OSCN)
- Benfotiamine (Milgamma™)
- Boluoke® (Lumbrokinase)
- Beta-Glucans (BG-Pro™)
- Boost-Pro™
- ACF-228®
- Boluoke® (Lumbrokinase)
- Can-C™ + capsules
**PEPTIDE BIOREGULATORS**

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**TOPICALS**

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SAVE ON MANY ANTIAGING PRODUCTS.
Simply use the voucher codes below within the stated timeframe and on the websites mentioned, (products may be restricted in some countries and prices may be subject to taxes and S&H where appropriate).

SPECIALIST

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<tr>
<td>RAPA-PRO™ (Rapamycin) 12x 5mg scored tablets</td>
<td>$89.99</td>
<td>$79.99</td>
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<tr>
<td>TRH (Abaris™) 30x 5mg sublingual tablets</td>
<td>$119.99</td>
<td>$82.99</td>
<td>$36 (expires end December 2019)</td>
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NUTRITION

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<td>ACF228</td>
<td>30 capsules</td>
<td>$39.99</td>
<td>$33.33</td>
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<td>CAN-C™</td>
<td>2x 5ml vials eye-drops</td>
<td>$39.99</td>
<td>$35.99</td>
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<td>ORALTIDE-PRO™</td>
<td>60ml mouthwash concentrate</td>
<td>$39.99</td>
<td>$34.99</td>
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Professor Khavinsons Peptide Bioregulators

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