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REPORT

Life Extension Is On Its Way To Becoming A Fact:
Alcor Conference

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“Growing old is a bad habit which a busy person can’t afford to develop,” was one of the many striking statements made at the Alcor conference in Monterey, California. It wasn’t meant as a joke. At long last, the hope that growing old would mean personal development rather than deterioration, a growth in wisdom and productivity rather than mental decline, “sage-ing” rather than aging, is becoming realistic. What yesterday sounded like science fiction is rapidly becoming fact.

Hardly anyone who follows the news needs to be told that we are experimenting in earnest with cloning, gene therapy, tissue regeneration, hormonal rejuvenation and freezing whole organs without damaging them. There is more reason than ever to expect that the ravages of aging can and will eventually be conquered, and that human life span can eventually be doubled—or extended even beyond that, once we understand more about the complex but ultimately modifiable mechanisms of deterioration (aging) and regeneration (“anti-aging”). Science fiction? By no means. A significant extension of human life span is a reasonable prediction that can be made on the basis of the current explosive progress in the biosciences. The Alcor conference showed just how much progress has been made in the battle for longer life, and the new paths we are beginning to explore.

by Ivy Greenwell

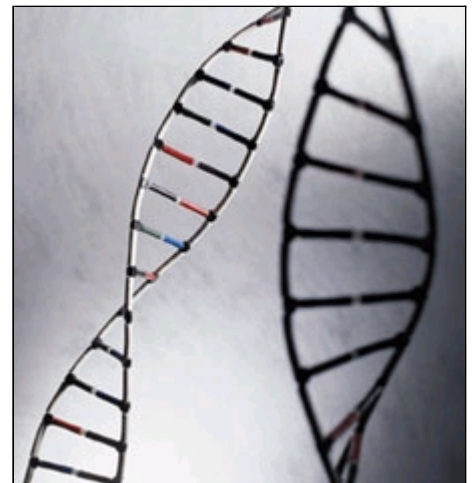
Dr. Tomas Prolla of the University of Wisconsin at Madison discussed the way certain genes change expression with aging. Dr. Prolla and Dr. Richard Weindruch are pioneers in the use of microarray-based gene profiling to study aging. A gene microarray is a small glass slide (it easily fits into a shirt pocket) that shows thousands of genes in a regular layout. The use of these microarrays represents a major breakthrough in aging research, enabling scientists to detect aging-related changes at the level of molecular genetics. Ultimately, the unraveling of the aging process at the genetic level may lead to truly significant anti-aging intervention.

There is a technique for measuring messenger RNA (mRNA) for each gene, i.e. the “expression” of that gene. Using gene microarrays, Dr. Prolla and colleagues compared gene expression in five-month-old “young adult” mice and elderly 30-month-old mice. Certain genes showed much more activation in old mice. Those were the genes that have to do with the stress response. Genes that govern DNA damage control were also upregulated, as were the genes that code for heat shock proteins (special proteins that repair other body proteins).

Neuronal injury genes were likewise upregulated with aging. “With aging, there is a marked increase in oxidative stress and inflammatory response in the brain,” Prolla said. He referred to the “gero-inflammatory manifold”—the widespread immune activation that is part of the inflammatory cascade.

This immune activation, as shown by higher levels of inflammatory prostaglandins and cytokines (IL-6, for instance), increases with age. Thus, aging means a progressive increase in chronic inflammatory status. The activation of the inflammatory response system (including the activation of the immune system) accompanies not only specific diseases such as atherosclerosis, osteoporosis and Alzheimer’s disease, but also so-called “normal aging.” In a way, our immune system increasingly becomes our enemy, destroying our own tissue.

Why this harmful over-response to stressors? Apparently evolution favored individuals showing a strong immune response to pathogens, so they could survive and reproduce. But what is optimal for survival and reproduction in youth may become harmful in post-reproductive years, as the amount of tissue damage accumulates while the energy output and capacity to repair tissue keep decreasing. At the same time, the immune function also deteriorates, with auto-immune disorders increasing, while the ability to defend against pathogens declines.



This is not to say that the older body doesn't try to repair damage. On the contrary, Prolla and colleagues found that various types of "repair" or "stress response" genes are expressed much more during aging. At the genetic level, the aging process resembles a state of chronic injury. Possibly the aging organism is devoting its resources increasingly to trying to repair damage, and not to building new tissue. In fact, Prolla did find lower expression of what might be called "biosynthetic" or tissue-building genes in old animals. Hence the well-known shift from the chiefly anabolic (tissue-building) state of youth to the catabolic (tissue-wasting) state of old age. The shift toward catabolism may have a lot to do with diminished energy production by the mitochondria. Genes that govern energy production were likewise downregulated with aging, Prolla found.

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Perhaps if the medical profession and the broader public realized that on the genetic level aging presents a picture of chronic injury and chronic inflammation, we could finally get rid of the misleading term "normal aging," and address aging as a multifaceted disease for which remedies need to be found.

The remarkable discovery made by the Wisconsin team was that calorie restriction significantly dampened these aging-related changes in gene expression. "Calorie restriction partially or totally prevents the changes in gene expression due to aging," Prolla said. The "repair" genes were clearly less activated in calorie-restricted animals. The simplest explanation is that there was less damage to be repaired. On the other hand, DNA synthesis was upregulated in the brains of calorie-restricted mice.



"Even when calorie restriction is started late in life, there is a visible impact on gene expression," Prolla said. However, there is a consensus that calorie restriction extends youth, and should be started as early as possible. One comforting finding is that even slight (10%) calorie restriction produces some life extension. Calorie restriction is also an excellent means of preventing or retarding aging-related diseases such as cancer, Alzheimer's and Parkinson's disease. There are several theories trying to explain the effectiveness of calorie restriction in producing life extension. One of them emphasizes the finding that calorie restriction slows down the deterioration of the immune system.

One use of the technique developed at the University of Wisconsin is that for the first time we are going to be able to measure, at the gene level, whether certain supplements retard aging. We will be able to pinpoint the effects of drugs and dietary regimens. Likewise, once we more fully understand what happens to gene expression during calorie restriction, we should be able to find ways to produce the same benefits without needing to resort to calorie restriction. At this point, calorie restriction is the most powerful known way to retard aging, but it is also the least acceptable to the average person. Finding an equivalent anti-aging regimen that does not leave us emaciated, libido-less

and depressed is a major challenge.

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Dr. Prolla did not go into the therapeutic aspect of trying to attenuate the "gero-inflammatory manifold" of aging. However, there are inescapable implications for anti-aging medicine based on mounting evidence that inflammation does indeed play a huge role in the aging process, and in the particularly devastating diseases of aging such as Alzheimer's disease and cancer (not to mention such relatively "minor" problems as cognitive decline, osteoporosis or gum disease). Developing better anti-inflammatory drugs, as well as using potent (and safer) natural anti-inflammatories, such as green tea extract and ginger, seems a very important aspect of the struggle against aging. Before we know how to manipulate genes to achieve more regeneration, we can already use current discoveries to try to reduce deterioration.

For all the excitement about the genetics of aging, however, Dr. Prolla stressed that any single-factor theory of aging is bound to be wrong. For instance, telomere shortening is important in those cells that divide. It is non-dividing cells, however, that are regarded as most critical. Perhaps at this point we should try to answer the most basic questions, such as "how can we measure aging?" It is very exciting to speculate about conquering aging, but first we should pay more attention to investigating and measuring the aging process.

Addressing the same topic from a different angle, Glenna Burmer, MD, PhD, of LifeSpan BioSciences, Inc., gave a presentation on cutting-edge developments in decoding the genome in terms of aging-related changes in gene expression. We need to know more about which genes are expressed more when the organism is young, and which are expressed more when the organism is old. One way to do it is to use gene chips (microarrays) that compare side by side tissue from a young individual with the same type of tissue from someone over 70. Such comparative research is currently being done, mostly in order to pinpoint genes related to specific diseases of aging.

Burmer would agree, however, that the underlying disease is the aging process itself. "Aging is a universal genetic disease," Burmer said. Current medical thinking, however, separates aging into different diseases: cardiovascular disease, brain aging, kidney aging and so forth, rather than investigating the underlying pathology of aging. Hence the emphasis on trying to find genes that are upregulated or downregulated in particular diseases, and can be targeted with drugs for that particular disease. But it is now obvious that many genes change their expression simply as a function of aging rather than a particular disease.

"Hundreds of genes go up or down with aging," Burmer said. When we compare young skin with old skin, for instance, we see that some genes are expressed much more in young skin, and other genes in old skin. The gene for apolipoprotein A2 is more expressed with age, as is one for the prostacyclin receptor. Genes that govern energy generation or utilization change their expression with aging, as do those that govern the response to oxidative stress. Also upregulated are various pro-inflammatory genes, such as the 5-lipoxygenase gene, which controls leukotriene production.

We are just beginning to decipher genes and make sense of certain gene clusters. The goal is to analyze 500 genes a day. Another goal is to use multi-tissue arrays to compare gene expression in a tissue from a 20-year-old with that in a tissue from a 75-year-old. Some of the most interesting genes have very few copies per cell, Burmer stated. For these, special sensitive arrays must be used.

Thanks to gene chips, we finally have a feasible way to hunt for "longevity genes." It turns out that such genes not only extend life span, but also delay senescence. They keep an individual young and healthy for a longer time. Theoretically at least, longevity genes would make people in their 60's or even 70's enjoy the kind of vigor that is now associated with one's 30's and 40's. They would not be plagued with arthritis, bone and muscle atrophy, creeping obesity, diminishing eyesight and hearing, forgetfulness, sleep disorders, thinning, graying hair and all the other dreary signs that physical and mental decline have begun in earnest. Does this sound like science fiction? The recent studies on centenarians have confirmed that these exceptional individuals enjoy a slower rate of aging, and typically remain in good health until the very end.

So far, 20,000 genes have been analyzed, with 500 identified as "aging genes." Does the progress in mapping the genome and identifying genes mean that gene therapy will soon become commonplace? Not really. Burmer pointed out that the delivery mechanism for gene therapy has to be very precise. We must improve the vectors (such as viruses, which can carry a gene inside the target cells). The new-generation vectors are already more promising, but much work remains to be done in order to resolve the issues of safety, precision and long-lasting results. Thus, somewhat surprisingly, the more likely result of genetic research development in the near future is going to be not gene therapy per se, but the creation of better, more precise drugs that can modulate the expression of certain critical genes. Some of these drugs could be "smart drugs" that will boost intelligence. Eventually the developments in genomics are likely to lead to a "healthy doubling of the human life span," Burmer predicts.



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Dr. Burmer also observed that if all genomic research were collaborative, with shared data, progress would be faster. Pharmaceutical companies, however, want to "own" a gene for which they try to target a drug. Genomic research is expected to speed up the creation of new and better drugs—hence the great interest of drug companies in molecular genetics. At present it is difficult to find a solution to this conflict between the ideal of open scientific communication and the economic imperatives of drug research.

One participant wished to know if genes characterizing various ethnic groups are going to be compared. Burmer replied that it is too early in the game for that. First we need to answer basic questions about the "meaning" of individual genes. In addition, human genetic variation is huge, and millions of tissue samples will need to be analyzed. At this point, Burmer said, we have not yet deciphered 90% of the human genome. In fact, there is not even a consensus as to how many genes there are. All agree that most genes have not yet been identified, and we don't know what huge portions of our DNA "mean." Nevertheless, this is the birth of a scientific revolution. Burmer compared the gene-decoding work currently being done with Leeuwenhoek's first beginning to look at cells through a microscope. It is a start of a new era.

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Therapeutic cloning

“Cloning—nobody thought it would happen,” Dr. Michael West mused, in his typical understated way. The founder of Geron and now the President of Advanced Cell Technology in Worcester, Massachusetts, Dr. West was speaking both of reproductive and therapeutic cloning. The goal of therapeutic cloning is to create replacement organs and reset the cellular clock. The most amazing and unexpected finding of Dr. West’s team is that cloning can be used to reverse cellular aging and not just restore the original telomere length, but actually extend telomere length beyond normal.

The term “cloning” is still most often used to mean reproductive cloning, the kind that produced Dolly, the famous cloned sheep. (Have you ever wondered about the origin of her name? She was named after Dolly Parton, because a mammary cell was used as the origin of Dolly’s DNA). Reproductive cloning is probably going to be important chiefly in animal breeding, so that a superior cow or bull or horse, i.e. a specimen showing especially desirable characteristics, can be replicated—a practice that is already widespread among orchid growers.

Human reproductive cloning remains highly controversial, although in the future it may become as accepted as in-vitro fertilization is today. What most people do not realize is that the cloning technique can be used not just to reproduce a genetic copy of the adult, but also to combine DNA from two or more parents. In fact, with each chromosome coming from a different individual, the cloned organism could have as many as 46 “parents.” More realistically, this kind of cloning might enable a lesbian couple, for example, to have their own biological offspring—a child that is genetically a combination of DNA from both women. As for the ethics of such cloning, Dr. West wisely suggested that we should be guided by love and compassion.

But therapeutic cloning is not designed to bring to life new human beings. Rather, its ultimate goal is to help those already alive. The problem with organ transplants is that the recipient’s immune system has to be suppressed in order to prevent rejection. Therapeutic cloning offers the advantage of creating new organs that are genetically identical with the individual.

But if the individual is elderly, aren’t the organs cloned from his tissue going to be senescent also? Won’t the cells of those cloned organs have short telomeres, as short as the telomeres in the parent cell, or maybe even shorter, indicating a very limited capacity for replication?

Before we answer this question, let us take a look at how cloning is done. In broad outline, the technique Dr. West has developed is as follows. He takes a senescent fibroblast (a skin cell) that is at the end of its ability to replicate. He then removes the nucleus from a bovine ovum (these can be cheaply obtained from a slaughterhouse). Next, he puts the fibroblast into the enucleated ovum. An electric shock then starts the embryonic development. And here comes the surprise: the resulting cells have been found to have longer than normal telomeres. It is as if something overcompensated for the senescence of the DNA-donating fibroblast. The aging clock, as measured by telomere length, has been reset not just to normal, but beyond. We do not understand how this takes place. It may have something to do with the fact that germ cells (in this case the bovine egg cells) possess telomerase, the “immortalizing enzyme” that rebuilds telomeres. But the details remain to be elucidated. Dr. West pointed out that we still know very little about embryonic development.

You may remember the concern when it was discovered that Dolly had shorter than normal telomeres. West explains this as most likely due to the different cloning technique used to produce Dolly. The fact that quiescent (non-dividing) mammary tissue was used may be one of the main factors. Using skin cells (fibroblasts), West and his colleagues discovered a way to regenerate an animal with longer than normal telomeres, indicating a longer cellular life span. It is yet too early to know if this will result in a longer life span of the cloned animals.

But how does this relate to therapeutic cloning, where we are concerned with generating a new organ, not a new individual? In the future, will an 80-year-old be able to receive a “young” liver grown in the lab, using his own genetic material? Apparently so. When



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a nuclear transfer is successful, the second stage (up to 14 cell divisions) is a cellular mass called a "pre-embryo." These cells are not yet differentiated. Using special techniques, scientists are beginning to learn how to direct the development of these cells into specific tissue and, ultimately, into whole organs. So far, much of this work has been accomplished using stem cells. But stem cells are very difficult to harvest from adult individuals. Fibroblasts, by contrast, are plentiful.

Needless to say, this technology is in its infancy. It can take a long time between pioneering experiments and medical application. Questions of safety need to be answered. Human egg cells are more likely to be used for human therapeutic cloning, and those are difficult and expensive to harvest. Many details remain to be worked out. But we are witnessing the birth of what Dr. West calls "regenerative medicine." "Life is immortal in the sense of regeneration," Dr. West stated. It is not enough to reduce degeneration—the true way to combat aging is to increase regeneration. Thanks to pioneers such as West, we can hope that science will make further advances in learning how to harness and direct the awesome potential of regeneration.

Chronobiology and life extension

Richard Morales, MD, specializes in anti-aging medicine and runs an anti-aging clinic in Mexico. He explained that his approach to rejuvenation emphasizes trying to regain a youthful synchronization of the circadian rhythms. This idea stems from the theory that aging is basically controlled by the brain. Our primary anti-aging task should be to concentrate on restoring optimal function of the central nervous system.

Perhaps the most fascinating idea was that of a chronobiological diet. The main principle is eating every three hours, with different nutrients emphasized at different times of the day. The morning starts with fruit; protein and complex carbohydrates are consumed later. While many nutritionists would agree that small frequent meals are best, it would take a lot of research to establish whether it is indeed best to start the morning with fruit, and so forth. While researchers have designed a diet that keeps expensive laboratory primates healthy and very resistant to cancer, our lack of knowledge about what kind of diet is optimal for most humans remains an embarrassing problem.

Another component of the resynchronization program is light therapy. We know that exposure to intense light strongly affects the production of melatonin. Other important hormones, growth factors and various neurochemicals are also affected by light. Some experts have suggested that light is absolutely necessary for optimal function, and many people in the industrialized nations do not get sufficient exposure to daylight. If intense light is provided later in the day, it resets sleep until a later time. Exercise too can have this effect.

A very effective part of this presentation was a graph showing the decline of catecholamines (dopamine and norepinephrine) and serotonin with aging. Dopamine declines much more dramatically, so that the youthful predominance of catecholamines disappears, and the ratio of dopamine to serotonin shifts considerably. In order to restore neurotransmitters to more youthful values, Morales uses agents that serve that neurotransmitter precursors. It could be pointed out that another way to increase dopamine is through the use of the drug Deprenyl (selegiline); based on the evidence from animal studies, there is also reason to think that powerful flavonoids such as those found in blueberries and strawberries, and even vitamin E, increase dopamine.

Another interesting part of the Multi-Modal Anti-Aging Program as practiced by Dr. Morales is an attempt to restore a more youthful sleep pattern, particularly the slow-wave sleep, which becomes increasingly deficient in the elderly. It is during slow-wave sleep that most growth hormone is released. One way to increase the depth and amount of slow-wave sleep is by using a compound called hydroxygammabutyrate (HGB). Unfortunately, though many experts claim that HGB is safe and beneficial when used correctly, HGB has been classified as an illegal drug. The closest we can come to it is a legal precursor called gammabutyrolactate (GBL). Morales uses both GBL and melatonin to regulate sleep.

Exercise is also a crucial part of the rejuvenation program. It's not aerobic exercise, however. Morales believes in the special efficacy of weight lifting as anabolic anti-aging exercise, particularly if the leg muscles are involved. The purpose here is to stimulate the release of growth hormone. Exercise also lowers blood sugar and insulin levels, both of which tend to increase with aging. Also, as everyone knows, weight lifting builds muscle, and can thus at least partly reverse aging-related muscle atrophy (after age 40, women begin to lose half a pound of muscle a year; menopause may accelerate that loss to a pound a year). If Morales is right about the special importance of leg exercise, then the advice to choose living on the second or third floor so that you have to take the stairs may indeed be valuable anti-aging wisdom.

Another way to stimulate growth hormone release is by increasing thyroid activity. Morales referred to a "thyroid promoter" as one of his therapeutic tools. Exercise also increases the levels of T3, the most active of the thyroid hormones. As for steroid hormone replacement, Morales favors transdermal delivery (creams, gels, patch). This includes DHEA.

The speaker also mentioned the need to modulate FSH and LH, the gonadotropic hormones that dramatically increase in women after menopause. It is not yet clear, however, what the involvement of these hormones may be in causing accelerated aging, or how early in life hormone supplementation (or another intervention) would have to start in order to significantly suppress FSH secretion.

We were somewhat surprised that Dr. Morales didn't specifically address the daily cortisol cycle, the derangement of which appears to play an important part in the deterioration of sleep and accelerated aging. If cortisol is too high, especially at night, immune function, muscle, bone and skin regeneration all suffer. The production of growth hormone and thyroid hormones also suffers. It is possible, however, that Dr. Morales' clinic attempts to deal with normalizing the cortisol rhythm through indirect means, such as exercise and providing precursors for the thyroid hormones, and for the various neurotransmitters.



Developing better anti-inflammatory drugs, as well as using potent (and safer) natural anti-inflammatories, such as green tea extract and ginger, seems a very important aspect of the struggle against aging.

Dr. Morales caused some controversy over his use of Winstrol, a synthetic anabolic steroid, for both men and women. He claims that Winstrol, a testosterone analogue, gives women "nice, smooth skin" without side effects such as acne or facial hair; given to men, injectable Winstrol builds muscle. Some members of the audience appeared skeptical over the wisdom of using Winstrol rather than natural testosterone.

Light therapy, sleep improvement, weight lifting, small, frequent meals, and providing nutrients known to improve brain function all appear to make sense in terms of what we already know about anti-aging medicine. Much research obviously remains to be done, both in terms of basic research and controlled clinical trials. Nevertheless, it is obvious that chronobiology has the potential for making an important contribution to anti-aging medicine.

Various theories of aging

Dr. Gregory Fahy presented the theory of genetically programmed aging. In the view of molecular geneticists, the increase in the levels of free radicals is the consequence of aging rather than a cause of aging. Up to a certain age, every organism has the ability for adequate repair. At some point in the organism's life, however, the rate of protein synthesis drops off suddenly and dramatically, and aging starts to proceed at an accelerated pace. This suggests the existence of a genetic mechanism that initiates an aging cascade.

Menopause is certainly one example of an "aging clock." There is also some fascinating research on fruit flies and life extension through manipulating the gene that codes for Elongation Factor 1-alpha, a protein crucial for regulating protein synthesis. Calorie restriction likewise appears to involve an increase in growth hormone and protein synthesis, as well as a slower rate of brain aging.

If there is a main "aging switch," then it is most likely located in the central nervous system, Dr. Fahy concluded. A conference participant confirmed this, saying, "Think neuro; forget everything else." Keeping the brain youthful does indeed appear to be a central task of anti-aging medicine. It may take gene therapy, however, to extend human life span in a truly significant manner. Proponents of nanomedicine might also argue that until nanotechnology, with its potential to repair the body at the molecular level, is taken seriously and developed, we can't make a leap into truly extended, possibly even "open-ended" life span.

The theoretical shift toward a more genetic perception of aging has broad implications. It is not enough to concentrate on the damage done by free radicals and glycation; we need to expand research on genetic clocks and genetically determined capacity for repair and regeneration. Our dependence on oxygen and glucose means that there is no escape from free radicals and glycation. However, it is likely that we can find at least partial means to enhance our ability to reverse and repair the damage. After all, a young organism has an impressive capacity for repair, for tissue building and regeneration; the true hope for conquering aging appears to lie in learning more about how to induce regeneration.

Some conference participants reacted to Dr. Fahy's presentation of the genetic-clock mechanism of aging with a degree of dismay. Actually, any gain in knowledge about the mechanisms of aging is a reason for rejoicing, Fahy pointed out, since it makes the possibility of intervention more likely. Knowledge really is power. Once we understand something, we can at least hope that eventually we will figure out a way to deal with it. Beginning to understand aging from a genetic point of view represents tremendous progress. Right now we still need to study the aging process at the molecular level to learn more about the aging-related degeneration. In the future, the emphasis may shift to finding ways to increase regeneration.

If the speed of aging is controlled chiefly by a genetic "switch," does this mean that taking antioxidants is pointless? Not really. While free radicals do not single-handedly cause aging, they definitely contribute to the aging process. Aging can't be attributed to any single agent, even if a genetic program is the main mechanism. Free radicals may be a "side show," but it is an important side show. Note the faster aging rate in heavy smokers and diabetics. It's not just the wrinkles; the prematurely aged skin of smokers and diabetics reflects the condition of all their connective tissues. Most important, their life expectancy is lower. Our current attempts at damage control may be crude, but they still appear to be better than doing nothing at all. No matter how advanced

regenerative medicine may eventually become, minimizing the damage will always make sense.

Antioxidants alone may not help you live considerably longer (let's say to 150), but they have been shown to help prevent life-shortening diseases. Thus, we are much more likely to live a healthy and productive life if we take good care of our health through all means available. First, we need to modify our diet—even 10% calorie restriction produces significant health benefits, and possibly even some life extension. We need to make sure we get regular exercise, enough sleep and rest, engage in enjoyable outdoor activities, reduce stress, cultivate supportive friendships and yes, take the right supplements such as acetyl-carnitine, CoQ10, lipoic acid and flavonoids. You will then be maximizing your chances of still being around for the next round of anti-aging breakthroughs.

Conclusion

The ALCOR conference showed that we are at the threshold of a revolution in medicine. The idea of extending human life span can no longer be regarded as science fiction. Developments such as cloning, gene therapy, chronobiology and nanomedicine carry an enormous promise. If the progress in these fields still seems too slow for our needs, it is up to us to support this kind of research as best we can.

Looking back over the most outstanding lectures, one can't help but notice the emergence of genetic research into aging as a revolutionary new development. We can at last investigate the aging process in terms of different expression of various genes. We can see aging at the molecular level: the prevalence of high energy production and high capacity for regeneration in a young organism, with a shift toward a state of low energy production and chronic injury and inflammation in an old organism, without enough resources for adequate regeneration. With further developments in cloning, we may indeed be able to grow new replacement organs from our own cells. Genetics may also furnish techniques to “edit” our DNA. Controversy is bound to rage on for decades, but everyone agrees that something monumental is in the making. We now have much better tools to work on finding better ways to decrease deterioration and increase regeneration. The medicine of the future is probably going to be mainly “regenerative medicine,” to use Dr. West's excellent phrase.

We are witnessing a shift toward spending research money on finding ways to make people live longer in a disease-free, productive state. Positive visions of human life are at last beginning to prevail. But for those who are in dire need of the new biotechnology, the progress is still agonizingly slow. Too few scientists are involved in anti-aging research. Again, we can help insure that such research receives adequate financing.

Ultimately, the message of the ALCOR conference was that the anti-aging revolution is gathering momentum, and those of us who want to live long and happy lives are no longer wishful thinkers, but rather informed realists. Let us make sure that we stay as healthy as possible through the means available today so that we may reap the benefits of tomorrow's further discoveries. As the nanotechnology pioneer, Eric Drexler, PhD, said, “The prospects for long life and health are genuinely excellent.” An even greater sense of hope can be derived from Dr. West's memorable pronouncement: “Life is immortal in the sense of regeneration.” The Alcor conference showed that we have begun to make revolutionary progress toward regenerative medicine.

By the way, some readers may wonder about the meaning of the word “Alcor.” It's not an acronym. It happens to be the name of the faintest star in the handle of the Big Dipper. In antiquity, being able to see Alcor was used as a test of visual acuity. Thus, Alcor stands for clear vision.

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