

ALCOR LIFE EXTENSION FOUNDATION

# CRYONICS

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REVERSIBLE  
CRYONICS  
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TAX BENEFITS OF  
LIFE INSURANCE  
PAGE 20

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THE PAST, PRESENT,  
AND FUTURE OF  
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# CRYONICS



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### The Past, Present, and Future of Membership Dues

*Why do Alcor members have to pay membership dues? What are they used for? Why do I also have to pay CMS (Comprehensive Member Standby) fees? Why can't we run the organization using income from doing cryopreservations and abolish dues? Can't we just cut our costs? How will dues change in the future?* Alcor CEO Max More answers all these questions and reflects on some of the challenges of running a modern cryonics organization.

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# The James Bedford Society



Gifts have played a fundamental role in the cryonics movement since its earliest days. Dr. James Bedford, a man whose extraordinary vision led him to become the first person to be cryopreserved, and the first to make a bequest to a cryonics organization, exemplified the determination of the early pioneers of cryonics. We invite you to follow in his footsteps, and join the James Bedford Society.

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# QUOD INCEPIMUS CONFICIEMUS

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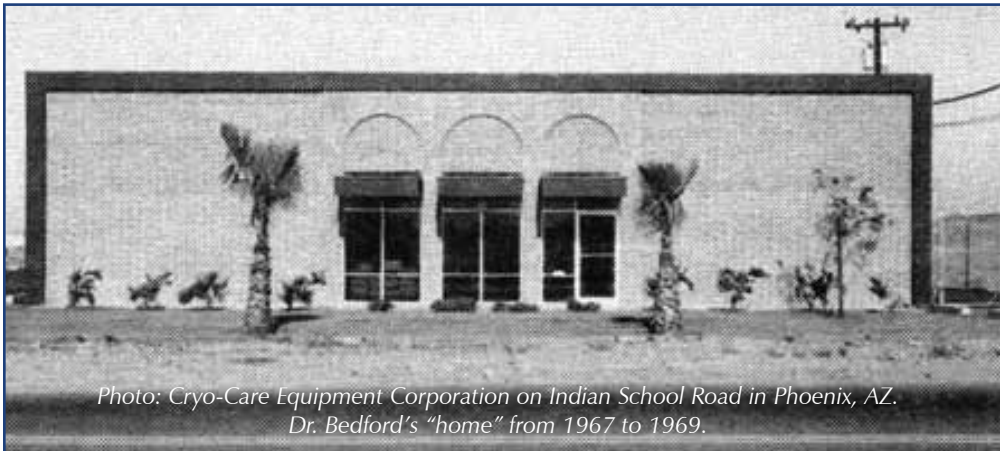


Photo: Cryo-Care Equipment Corporation on Indian School Road in Phoenix, AZ.  
Dr. Bedford's "home" from 1967 to 1969.



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## REVERSIBLE CRYONICS By Aschwin de Wolf

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In a previous column called “Iatrogenesis and Cryonics” I observed that cryonics is uniquely vulnerable to iatrogenic injury because the objectives of individual cryonics procedures (such as stabilization) are not clearly defined and due to the lack of obvious feedback that a low temperature stabilization procedure entails. This does not mean that cryonics advocates have not thought about how to look at the overall quality of a cryonics case. On the most general level we can evaluate a cryonics case by looking at the degree to which the cryonics stabilization procedure *itself* adds additional injury to the patient. This is important because critics of cryonics are usually more skeptical about the effects of stabilizing the patient at cryogenic temperatures than about the idea that a person who is considered terminally ill today may not be considered terminally ill in the future. The idea that the cryonics procedure itself does not add additional injury to the patient also ties in with the idea that one of the most important mandates of medicine is to do no harm.

What can a credible cryonics organization do to move its procedures in the direction of reversibility? At the most general level it can reflect this by formally recognizing the goal of developing human cryopreservation

technologies that are injury-free. In terms of a research objective, this means that it should aim for human suspended animation. The idea of reversible human cryopreservation is straightforward and easy to communicate. In fact, most laypeople who first hear about cryonics intuitively grasp this point. It also provides a useful benchmark to assess the degree of technological progress at a cryonics organization and evaluate the performance of a cryonics organization in cryopreserving humans.

But how can the concept of reversibility be applied to a cryonics organization that has not yet perfected reversible human cryopreservation? In this case one can still ask *how far* we can push the goal of reversibility. This raises another challenge. How can we know to what point our procedures are still reversible if we do not actually reverse them? For starters, we can look at the limits of conventional medicine (hypothermic circulatory arrest) and ensure that our procedures conform to the physiological requirements of these procedures. Another (complementary) approach is to define reversibility as maintaining *viability of the brain* and collect data that will provide us with an answer regarding how well we have achieved this objective.

As I write this, our understanding is that, under ideal circumstances, we can keep the brain viable up to at least the early stages of cryoprotective perfusion (which is conducted around 0° Celsius). It would be desirable to have a better empirical understanding of this, and one approach would be to take a very small, microliter brain sample of a patient (an established harmless medical procedure) and subject it to a variety of viability assays (such as the K<sup>+</sup>/Na<sup>+</sup> ratio). A fruitful *research* objective would be to achieve loading and unloading of a vitrifiable concentration of cryoprotectant in the brain and recover organized electrical activity (EEG) in a suitable animal model and then modify this protocol for human cases. If we achieve this, viability of the brain may be retained during the descent to cryogenic temperatures.

Currently the “descent to cryogenic temperatures” is not a completely innocuous step because thermal stress-induced fracturing can still produce *mechanical* damage. To eliminate this form of damage and transform the challenge of reversible human cryopreservation into a biochemical problem, intermediate temperature storage appears to be a requirement. ■

# The Past, Present, and Future of Membership Dues

By Max More



*Why do Alcor members have to pay membership dues?*

*What are they used for? Why do I also have to pay CMS (Comprehensive Member Standby) fees?*

*Why can't we run the organization using income from doing cryopreservations and abolish dues?*

*Can't we just cut our costs?*

*How will dues change in the future?*

These are questions I hear frequently. Our current level of membership dues is frequently cited as a reason for members — especially those with reduced incomes — to cancel their memberships. (Many of these indicate that it's not increases in dues that are the main problem; it's the loss of a job or drop in income. Most of these individuals tell us they intend to return once their financial situation improves.) Alcor's growth rate has declined over many years, but has slowed to a distressingly low rate in the last few years. It's entirely plausible that part of this is due to the cost of membership dues.

When I took over as Alcor's president at the start of 2011, I knew I would face a difficult situation. Just one year earlier, dues were increased from \$398 to \$478. (Including annual CMS fees of \$120 this resulted in an increase from \$518 to \$598 per year.) That was the first dues increase in eight years. Just one year later, on January 1, 2011 (my first day as president) dues were raised again from \$478 to \$620 — an increase of 30%. (Including CMS fees — which increased 50% from \$120 to \$180, this resulted in an increase from \$658 to \$800 per year.)

The yearly cost of membership from 2002 to 2012 rose quite a bit, especially when you include CMS fees. No CMS fund existed in 2002. That meant that members did not get a standby unless they paid for it at the time they needed it. The universal CMS fee has enabled Alcor to provide everyone with a standby, but it has added to the total yearly cost of membership.

The recent large jumps in dues may leave members with the impression that dues have risen more rapidly than is actually the case on average. To put the longer-term increases in context, excluding CMS fees, here are the numbers:

**From 1986 to 2013, membership dues rose from \$200 to \$620, which is 4.28% per year. (This is higher than general inflation, but only marginally.)**

**Looking at the last 18 years, from 1995 to 2013, dues went from \$398 to \$620, which is 3.07% per year. (Medical inflation exceeded 10% annually in the 1990s, and 6% over the last few years.)**

**Why so high and why the increases?**

Clearly, dues have risen significantly. But let's put the rise in the context of overall inflation. When dues were increased by 10% back in 2002, that increase lagged the CPI increase of 16% over the same period. In other words, dues were not raised enough to keep up with the general rise in prices. When that lag is allowed to build up over years, the adjustment becomes more painful. Almost a decade went by before dues were again adjusted. Even as recently as 2010, Alcor's dues were only \$30 a year greater than they were in 1992 after adjusting for inflation. Only at the start of 2011 did the rise in dues finally move significantly above the rate of inflation.

Apart from catching up with inflation, the one-two punch of 2010 and 2011 were necessary to enable Alcor to tackle its structural deficit. (By "structural deficit" I mean a deficit when irregular and unpredictable revenue from bequests, donations, grants, and cases is excluded. Alcor has shown an apparent budget surplus over the last two years, but still has a structural deficit.) Since a substantial grant was expiring after three years, a deficit of

\$400,000 was projected if no action were taken.

Over years and decades, some of Alcor's expenses rose substantially. Employee expenses account for a large part of overall expenses. Those expenses rose significantly over the years as Alcor transitioned away from a volunteer-based to a professional organization (starting very modestly in the 1980s) and found itself paying more market-based salaries. I do not expect this increase to continue. On the contrary, *if* we can grow our organization, I believe that we are at a point where economies of scale can be reaped: we could double or triple our membership without coming even close to doubling or tripling staff costs. But the painful dues increases of recent years have reflected the reality of catching up with inflation and with realistic employee costs rather than unsustainable volunteerism.

Quite a few members do not pay the full \$800 per year. They pay significantly less if they are family members, students, or members of 20 years or more. This will not be much comfort if you pay the full amount. You may be grumbling at the cost even if you understand the need to catch up with inflation and to tackle budget deficits. Even so, consider that this amount is a less-daunting \$67 per month. On a still shorter-term basis, your last, best chance to beat death is setting you back \$2.19 per day in dues. That's a little more than a tall cup of coffee at Starbucks (and quite a bit less than a short latte).

This is significant for some members; for others, it may be more the rapid recent increases rather than the actual cost which are problematic. Considered as a daily or weekly amount, the cost of membership may seem quite modest. But the success of cryonics for any particular individual is highly uncertain, while the current cost is real and obvious. Even if — for most members and many potential members — the cost is quite manageable when considered objectively, willingness to pay is based on perceptions of costs and benefits, not necessarily on objective facts.

At current levels, it's seems clear to me that dues cannot increase (beyond pacing inflation) without reducing membership retention and growth. So, my remarks

about the (objective) modest-for-many costs of cryonics should not be taken to suggest that I am in the least dismissing concerns about rising costs, or that I am not doing all in my power to address those concerns. I have no doubt that the current level of dues is resulting in lost members and is making it harder to attract new ones. Growth is important to deepen our resources — so we can defend ourselves against attacks, do more research, and ensure the continued existence and resilience of the organization.

### **Why not pay for operating expenses from Cryopreservation Fund income?**

Doesn't it make sense to pay operations out of the income from the service of providing a cryopreservation and long-term care? After all, Alcor isn't like a club where you pay monthly or yearly dues in exchange for specific services.

Membership dues have never been sufficient to cover all of Alcor's recurring costs, so we have relied on other sources of income, including donations and bequests. But let's restrict ourselves solely to considering what it would take to replace membership dues. Membership dues will bring in an estimated \$453,000 in 2012 (that's after allowing for bad debt). So that's the amount that would have to be generated from case income.

Given Alcor's existing membership base and the distribution of ages and life expectancies, we expect about eight cryopreservation cases per year. The actual number varies wildly: Not long ago, we had 12; in 2012 we had only three. Suppose all cases were not only fully funded but each cryopreservation fund averaged \$40,000 above current minimums. If we continue the practice of distributing half of these funds to the PCT and half to operations, those eight cases would generate  $8 \times \$20,000 = \$160,000$  for operations. We would need to have almost 23 cases per year, funded \$40,000 above minimums, to replace membership dues.

This is very much an approximation. On one hand, we might reduce the estimated number of annual cases needed to eliminate dues because most cases funded exactly at the minimum should produce a modest

gain for operations. However, this varies by case and is not always true. On the other hand, the 50/50 split between PCT and operations is only a default. We use that rule when a member does not specify the distribution of funds above the minimum. Most of those who do specify a distribution give it all to the PCT, or some to research, with very little to going to operations. This would have to change in order for over-minimum funding to have a major impact on dues. The actual number of cases per year required to reach the "dues-replacement level" might therefore be 23, or 40, or any other number, depending on assumptions about over-minimum funding and distribution of that funding.

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*"The universal CMS fee has enabled Alcor to provide everyone with a standby, but it has added to the total yearly cost of membership."*

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By the time our membership has grown enough to reach the dues-replacement level, expenses would probably have risen (although I expect costs to rise considerably less than proportional to membership size from this point on). So the dues-replacement level would then be higher than on the assumption of fixed operating costs.

The problem should be obvious. *Even if* all members provided cryopreservation funds \$40,000 over minimums, we would need at least three times as many cases as we can reasonably expect today in order to generate sufficient revenue to replace membership dues. Compounding the challenge, *most* Alcor members today are funded at levels *below* current minimums. These cases are likely to drain operating funds.

But this is not an all-or-nothing matter. I would like to see us move gradually in the direction of funding operations more from cryopreservations and less from membership dues. This will take time and growth and major improvements in

funding of cryopreservations. We could shift funding much faster in that direction if we received major bequests, especially if some went to the Endowment Fund and some to the Operational Reserve Fund. The uncertainty of bequests and of operational income from cases is why these are excluded from calculations of the structural deficit. As membership grows, we should be able to gradually make allowance for these more irregular forms of income, recognizing their contribution to the structural budget and thereby having a downward effect on membership dues.

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*“To those members who have been with us for many years, let me point out that we have recently introduced a new discount (\$186 reduction per year) for individuals who have been members for 20 years or longer.”*

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### **How will dues change in the future?**

Let us separate out the likely trajectory of (a) basic membership dues from (b) membership dues as they may be for any particular individual if we adopt an Underfunding Plan in which members with below-minimum cryopreservation funds pay additional dues to partly compensate. (That would only happen if they did not raise their funding to current minimums or, in the case of whole body members, refused to be switched to neuro member status, and who do not qualify for the Hardship Fund.) I will consider here only the plausible direction of membership dues for members funded at current minimums or higher.

All other factors being equal, dues will depend mainly on costs and growth rates. As I mentioned earlier, I believe we have reached a point where further membership growth will allow us to reap economies of scale: We can increase membership with a less than proportional increase in operating

expenses. Posited economies of scale are not mere wishful thinking: Consider that, say we grow our membership by 100%: while we might need some additional help in some areas, we would not need to hire a second president, or a second finance director, and so on. Only in 2012 did we introduce Associate Membership, an option that is essentially costless and yet which could generate significant income if we can grow this category into the hundreds or higher. At the same time, future increases in employee costs should closely track overall increases in compensation throughout the economy (tracking a combination of inflation and productivity gains), with little or no need for catch-up increases.

In addition, we have been and will continue to take measures to control and reduce costs where possible. For instance, I eliminated one full time position; it may be possible to reduce employee costs further; we are renegotiating contracts and licensing arrangements; requiring that all new ongoing expenses be compensated by cuts in other areas (until structural budget balance is achieved); and have insulated the building to save several thousand dollars annually. We are also benefiting from Life Extension’s generous support in covering the production and distribution of *Cryonics* magazine.

These core factors strongly suggest that if we can grow membership, we should be able to hold down membership dues. If growth is slow, while inflation continues to average around 3% annually, we may only be able to prevent further rises. If growth is stronger, reduction in dues becomes feasible.

Currently, the 2% draw from the Endowment Fund adds about \$70,000 to operating funds. If we can grow the Endowment Fund, that 2% draw will grow. Suppose we were fortunate enough to receive a \$10 million infusion. That would generate an additional \$200,000 annually. That would be equivalent to 44% of the income expected from membership dues for 2012. Even after eliminating the structural deficit, that would allow a significant reduction in dues. We cannot know the amounts involved, but we should work toward building the Endowment Fund

over time. This will help keep a lid on — and perhaps reduce — membership dues.

Here’s another possibility: Suppose our wealthier members recognize the benefits of improving retention and growth in number of members, and contribute to a fund specifically intended to subsidize dues over a period of time (perhaps ten years). The idea would be to use the fund provided to cut dues so as to accelerate membership growth, to reach a point where the additional members would more than replace that fund income (and allow us to reap economies of scale).

We have already noted another factor that could allow us to progressively reduce membership dues — and possibly one day eliminate them altogether: An increase in bequests and over-minimum funding of cryopreservations, some part of which is directed to operations. We are currently thinking about one step in this direction: The possibility of reducing or waiving CMS fees for sufficiently well-funded members. This may turn out not to be feasible, but it’s a possibility we are pondering.

To those members who have been with us for many years, let me point out that we have recently introduced a new discount (\$186 reduction per year) for individuals who have been members for 20 years or longer. This is intended to reward members who support Alcor over the long haul, and to show that they will not face ever-rising dues. I will be proposing further discounts for members of 25 years’ and 30 years’ standing.

Finally, I understand that my grounded optimism about future dues may not greatly comfort those of you who are struggling *today*. You may have lost your job, or retired, and been forced to take a lower-paying job in this difficult economy. Whatever you do, if you are struggling, *talk to us*. We work with members as best we can. If you’ve fallen far behind in paying dues and you never respond to our communications, eventually your membership will be cancelled. Talk to us, show willingness to work with us, and we can figure something out.

*My thanks to Bonnie Magee for suggestions that improved this article. ■*



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## ANCIENT BRAINS

By Chana de Wolf



Cryonics seeks to preserve terminally ill humans in anticipation of future medical advances that may restore these patients to youthful vigor, cure their devastating diseases, and resuscitate them from cryopreservation itself. At the core of this mission lies the goal of preserving that which we know to be most important to continuity of the person him/herself: the brain.

Absent reversible cryopreservation of the brain (i.e., maintenance of viability), a cryonicist's best hope for eventual resuscitation lies in preserving brain ultrastructure with as much fidelity as possible. Improvements in cryopreservation solutions, methodologies, and protocols from the field to the operating room have greatly enhanced our ability to meet this objective, as evidenced by microscopic evaluations of tissues vitrified in the lab. More recently, CT scans of patients after neuropreservation have provided valuable feedback as to the efficacy of cryoprotective perfusion in actual Alcor cases. Such progress bodes well for good patient outcomes.

But even our greatest attempts at optimal preservation are thwarted by issues such as long ischemic periods resulting in significant perfusion impairment or even the inability to perfuse at all. So how do we evaluate these patients in light of our objective?

Perhaps the best place to start is the extreme. Let us consider, for example,

a prehistoric human brain discovered in 2008 at a construction site in York, UK. A paper published in 2011 in the *Journal of Archaeological Science* ("Exceptional preservation of a prehistoric human brain from Heslington, Yorkshire, UK") provides gross and histological observations as well as preliminary results of chemical assays in order to determine the extent and cause of preservation of the brain. Low-powered reflected light microscopy and electron microscopy were performed to explore the surviving morphology and histology of the brain, while highly sensitive neuroimmunological techniques and proteomic analyses were employed to explore brain chemistry.

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*"While interesting in its own right, few would argue that the Heslington brain represents a state of preservation amenable to resuscitation."*

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Examination of the skull indicated death by an abrupt trauma to the neck followed by deliberate dismemberment of the head between vertebrae C2 and C3. Significantly, the authors report "no trace of microbial activity, bacterial or fungal, with none of the porosity or 'tunneling'

that is characteristic of putrefactive microorganisms." Examination of the brain masses revealed recognizable sulci and gyri, but neither macroscopic nor CT evaluation could differentiate between grey and white matter.

Histological examination of the brain masses showed "a homogenous, amorphous substance that had not retained any cellular or matrix structure." Transmission electronic microscopy (TEM) also did not detect any surviving cellular structure, although it did reveal what appeared to be "numerous morphologically degraded structures characteristic of the myelin sheath of nerve fibres."

Preliminary biomolecular analysis found only 5% of the brain was detectable as hydrolysable amino acids, in contrast to fresh brain tissue of which proteins represent more than 1/3 of dry weight. When compared with a fresh brain, the Heslington brain was also depleted in polar amino acids and enriched in hydrophobic amino acids. Very little undegraded solvent-soluble brain lipid was preserved (0.8%-1.1% wet weight compared with 17.1% for rat brain). In addition, there was an almost complete absence of phospholipids and only a trace of cholesterol, while degradation products of a wide range of lipids were found in abundance.

Ultimately, the authors determined that the preservation of this brain was due to decapitation (thus eliminating the movement of putrefying bacteria from the

gut to the brain) followed by inhibition of postmortem putrefaction achieved through rapid burial into fine-grained wet sediment. They go on to argue that this type of preservation is not as unusual as one might think, citing several similar examples of preserved prehistoric human brains, almost always found in wet burial environments.

While interesting in its own right, few would argue that the Heslington brain represents a state of preservation amenable to resuscitation. The ability to infer anything beyond gross macro structure has been obliterated and the normal chemical constituents of the brain have dissolved almost completely into the surrounding environment. Clearly, much of the look of a brain can be retained while none of the person's identity remains (or is recoverable).

Let us then look at a situation that hits a little closer to home. Published in *Forensic Science International* in 2007, an article entitled "Autopsy at 2 months after death: Brain is satisfactorily preserved for neuropathology" provides us with considerable food for thought. In this

example, a 77-year-old woman's whole body was stored postmortem in a 3°C cooling chamber for 2 months prior to chemical fixation of her brain at autopsy.

The authors describe moderate autolysis of internal organs of the body, indicating the start of decomposition and putrefaction, as well as reduced tissue consistency and superficial areas of disintegration of the brain. Overall gross morphology was sufficiently preserved to allow macroscopic examination and application of neuropathological methods for diagnosis of neurological disorders. Importantly, they also report that "histologically, normal brain structures including all major parenchymal cell types (neurons, astrocytes, oligodendrocytes, microglia), neuropil, axons, and myelin sheaths were preserved."

In this case, the use of cold temperatures (3°C) drastically slowed, but did not stop, deterioration of the brain. However, enough of the brain's chemical constituents and physical structure remained to provide the basis for possible future resuscitation.

And while this woman's brain was preserved by chemical diffusion over the course of 9 weeks (allowing for continued degradation of subcortical tissues during the course of fixation), the use of cryogenic temperatures to quickly preserve her brain would also have been possible, as has been the situation for many "straight frozen" Alcor patients who were received in similar condition.

Exactly where the line between recoverability and non-recoverability — resulting in information-theoretic death — exists is yet to be determined. And while we push, rightfully, for ever greater preservation methods, we do well to remember that those preserved under less-than-optimal conditions are by no means lost causes. Preserved information, even in fractured and distorted form, may well be adequate to infer the original state. ■



## Alcor Member Forums

Discussion board of the Alcor Life Extension Foundation

Discuss Alcor and cryonics topics with other members and Alcor officials.

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- Cell Repair Technologies
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Other features include pseudonyms (pending verification of membership status) and a private forum.

<http://www.alcor.org/forums/>



# Lifespan Society of British Columbia Mini-Conference

By Julie Saucier

The Lifespan Society of British Columbia is a life extension advocacy organization established in the summer of 2012 in the province of British Columbia (B.C.), Canada. On December 8, 2012, the Lifespan Society hosted its first mini-conference in downtown Vancouver, B.C. The event brought in Society members and other interested persons from throughout the province to participate in a variety of longevity-related dialogues. The goal of this event was to broaden awareness of the organization and attract new members from a range of interest groups that have coalesced around the Society through its formation. Turnout for the conference (25!) exceeded initial expectations and allowed the organization to showcase new programs and initiatives to a wide audience. Lifespan Society President Luke Cockerham acted as host for the event.

Attendees were welcomed to the conference with a lunch social where members of the Lifespan Society were introduced and guests were encouraged to share a bit about their background and interest in the Society. Following lunch, Luke gave a welcoming address to introduce



the organization to new members and offer some background as to the Society's official founding last year. A brief presentation was given on the history of the organization's inception in 2010 and the programs currently being offered to its members, as well as several new projects being planned for 2013 to further the Society's mandate. Among the programs Lifespan Society intends to continue or offer anew in 2013 are life extension hikes in the Lower Mainland of B.C., recurring movie nights on life extension themes, and a life extension public speaker series. The Lifespan Society's annual letter to its stakeholders was also made available to the conference attendees, offering further updates on the continued growth of the organization.

Following his presentation, Luke introduced Keegan Macintosh as the conference's first speaker. Keegan is

currently completing his professional legal training on a fellowship from the Lifespan Society provided through a generous grant from the Life Extension Foundation. Keegan's discussion on access to cryonics engaged the audience, some of whom were uninitiated in cryonics and the legal impediments that currently exist in the U.S. and Canada. Following Keegan's address, Luke introduced Hans Wu as the second speaker for the day with his talk on "Evidence-based Nutrition." Hans is currently attending medical school in Vancouver and his presentation covered a variety of nutrition-based topics. Hans's informational approach to diet and supplements was very well received and generated a wealth of questions and feedback from the audience.

After the presentations and some more casual networking, conference attendees had the opportunity to choose between attending a meeting of the local cryonics interest group, CryoBC, or watching a BBC life extension documentary. There was more networking over dinner, then participants assembled once more in the media room of the conference venue to view a second life extension-themed film.

Feedback from the attendees was uniformly positive, pointing to the Lifespan Society's first mini-conference as a major success for the organization and setting the stage for an exciting year to come. To learn more, attend future events, or become a member of the Lifespan Society of B.C., visit <http://lifespanbc.ca>. ■



## THE MULTI-HEADED HYDRA

By Keegan Macintosh



*This article explores some of the regulatory challenges facing those who would bring rejuvenation biotechnologies, like those pursued by Dr. Aubrey de Grey and the SENS Foundation, to market. It does not presume familiarity with Dr. de Grey and his work; I've tried to make it informative to all alike.*

### The Conquest of Aging

Biomedical gerontologist Aubrey de Grey predicts that the first human being to live to 1,000 years old is alive today. Who exactly that person might be – or rather, how old they are today – is a detail that Dr. de Grey has wavered on, but he has remained firm in his commitment to that prediction, and is certainly one of the most prominent figures working towards realization of the technologies required to make his prophecy reality. In his book, *Ending Aging*, Dr. de Grey describes his proposed approach to the “problem” of aging, and how it differs from those presently in practice.[1]

In Dr. de Grey’s opinion, the current paradigm devotes a vast majority of resources to geriatric care, which attempts to cure or manage age-associated diseases only after they emerge as recognizable groupings of symptoms. To quote an apt metaphor from another longevity advocate:

“[T]he challenge of treating illnesses in the elderly must at times seem like Heracles’ trials of combating the multi-headed Hydra. Each time one head was severed, two more would sprout in its place. Likewise, a patient might survive a serious cardiac episode with help of antihypertensive

drugs only to succumb to cancer *and* dementia.”[2] [emphasis in original]

Meanwhile, the (significantly smaller) remaining portion of research dollars goes towards biogerontology, which studies the upstream causes of aging, any benefit of which is probably unrealizable for several human generations. However, Dr. de Grey argues that without necessarily knowing much more about the upstream causes of aging than is currently understood, it is already possible to categorize the different forms of aging “damage,” and devise therapies that will repair the damage at a sufficient rate to achieve what he terms “longevity escape velocity.”

Dr. de Grey calls his theory “Strategies for Engineered Negligible Senescence” (SENS). There are seven strategies, each related to one of the seven major categories of aging damage thus far discovered. Those categories (and proposed therapies) are: (1) cancer-causing nuclear mutations (removal of telomere-lengthening machinery, aka OncoSENS); (2) mitochondrial mutations (allotopic expression of 13 proteins, aka MitoSENS); (3) intracellular junk (novel lysosomal hydrolases, aka LysoSENS); (4) extracellular junk (immunotherapeutic clearance, aka AmyloSENS); (5) cell

loss & tissue atrophy (stem cells and tissue engineering, aka RepleniSENS); (6) cell senescence (targeted ablation, aka ApoptoSENS); and (7) extracellular crosslinks (AGE-breaking molecules and tissue engineering, aka GlycoSENS). The SENS Foundation was established in 2009, helped in part through seed funding provided by Peter Thiel, co-founder of PayPal and a managing partner of The Founders Fund. The SENS Foundation’s stated purpose is “to research, develop and promote comprehensive regenerative medicine solutions for the diseases and disabilities of aging.”[3]

Delving into the details of each of Dr. de Grey’s proposed strategies is beyond the scope of this article, but it is worth taking a closer look at one of the seven. LysoSENS aims at “junk” molecules which cannot be broken down by human lysosomal enzymes. Over time, these molecules accumulate within cells, contributing to conditions like macular degeneration, atherosclerosis, and Alzheimer’s disease (AD)[4]. Dr. de Grey’s proposition is to search for novel lysosomal enzymes (novel to humans, that is) in bacteria, molds, and other microbes that are involved in “recycling” dead animal bodies, since the “junk” inside our cells is — along with the

rest of us — food to them. SENS research being carried out at Rice University has already identified one such enzyme that, when targeted to the lysosome, decreases cytotoxicity of 7-ketocholesterol (7KC), an oxysterol associated with atherosclerosis and Alzheimer's disease.[5] Enzyme replacement therapy is already used for the treatment of lysosomal storage diseases not associated with aging, like Gaucher's disease. Insofar as it could be used to treat a condition (or conditions) remedially, a therapy targeting 7KC with a novel lysosomal enzyme might otherwise resemble traditional approaches to disease treatment, but it could also be used preventively. Other SENS pose even greater challenges to the traditional distinctions between cure, prevention and enhancement. The objective of MitoSENS, for instance, is to render the recipient immune to the fallout consequences of mitochondrial DNA mutations by placing backup copies of the thirteen mitochondrial genes — which naturally reside only inside the mitochondria — into the cell nuclei. Significant research progress is being made into this strategy as well.[6]

### The problem of normative definitions of aging

Dowsing for fountains of youth is well and good, but won't get us very far unless they can be tapped and piped to the marketplace, and while there are many scientific obstacles to overcome before rejuvenation biotechnologies are realized, there are also social, political and legal ones. Many of these problems are definitional. For one, what exactly distinguishes age-associated diseases and conditions from “normal” features of aging? In the words of one scholar: “[F]rom the perspective of modern biogerontology, there is little to distinguish biological ageing from a disease state.... To argue that ageing is not a disease by virtue of its universality is as misleading as it is to argue that the Basenji is not a dog because it does not bark.”[7] But to dismiss this debate as purely semantic or philosophical would be to misunderstand the true difficulty the definitional problem poses.

The U.S. Food, Drug and Cosmetic Act defines “drug” as, *inter alia*, “articles intended

for use in the diagnosis, cure, mitigation, treatment, or prevention of disease in man or other animals” and “articles (other than food) intended to affect the structure or any function of the body of man or other animals.” [8] So far so good, because even if the U.S. Food and Drug Administration (“FDA”) did not agree that a particular undesired physical state was a “disease” for the purposes of the first definition, it would be difficult to deny that a proposed therapeutic (whether a chemical entity or a biological product[9]) was not intended to affect the structure or functioning of the body, at some level. However, present regulatory approval pathways indirectly require that a drug be “indicated for the treatment, prevention, mitigation, cure, or diagnosis of a *recognized disease or condition* or of a *manifestation of a recognized disease or condition*, or for the relief of *symptoms associated with a recognized disease or condition*.”[10] [emphasis mine]. The phrase “recognized disease or condition” is not defined in this context[11], and the FDA is not itself the recognizer, but rather looks for consensus within the clinical and/or scientific communities regarding the existence of a particular disease or condition, and of clear criteria for clinical diagnosis thereof.[12] To quote one author: “To the extent that many problems of ageing have not been formally recognized by any of these processes, the FDA has no clear guidance on how to determine if a proposed indication would be acceptable.” [13]

For many age-associated conditions, the question of “recognition” is a value-laden debate. While some commentators will no doubt accuse longevity advocates of “disease-mongering”[14], Dr. de Grey would probably argue that that sort of reaction is a symptom of what he terms the “pro-aging trance”[15] — a terror management strategy that accepts and embraces the apparently unavoidable progressive wasting of one's body (and mind), instead of rejecting and resisting it. But the cognitively dissonant distinction between normal, “healthy” aging on the one hand, and “diseases” of aging on the other is not impermeable. For some historical perspective, it is worth considering the example of Alzheimer's disease. When it was first described in 1910, AD only

included what is now referred to as “early-onset Alzheimer's disease,” i.e., when the symptoms of “senile dementia” appeared in someone under 65.[16] Due to its vastly less frequent incidence, this “presenile dementia” was assumed to be distinct from the normal variety. However this normal/abnormal categorization broke down in 1977, due to professional recognition of their near identical symptomologies, making the early-onset subtype by far the minority of AD incidence.[17]

A present-day example of this process of recognizing “normal” features of aging as diseases or conditions of aging, is the case of sarcopenia. Sarcopenia (literally “poverty of the flesh”) describes the degeneration of skeletal muscle mass and strength that occurs with aging that contributes (in part) to disability, frailty, and morbidity in older persons.[18] Until fairly recently, sarcopenia and related conditions like sarcopenic obesity were considered “normal” aspects of aging, much like senile dementia prior to 1977. To be fair, both sarcopenia and senile dementia *are* normal, insofar as they are common conditions in older persons — but that does not mean they are untreatable, nor that they should be left untreated. A number of potential drug targets have been identified that may be of use in treating sarcopenia[19], but if consensus recognition of the condition is lacking there may not yet be a regulatory pathway for licensing therapeutics to treat it.[20]

Thus, as it stands, forging a regulatory pathway for treatments of a common, disabling (and in some cases indirectly lethal) feature of aging involves two distinct steps: first, persuade the scientific and clinical communities that a particular symptomology of aging can and should be treated, and second, persuade the FDA that everyone else is persuaded. But this is not insurmountable. The European Working Group on Sarcopenia in Older People published a “practical clinical definition and consensus diagnostic criteria for age-related sarcopenia” in 2010[21], which was followed by a consensus definition from The International Working Group on Sarcopenia in 2011[22]. In the U.S., the Foundation for the National Institutes of Health, the National Institute on Aging,

and the FDA held a Sarcopenia Consensus Summit on May 8-11, 2012.[23] A number of clinically meaningful end points have been proposed for assessing treatment efficacy[24], including patient-reported outcomes.[25] Under appropriate regulatory supervision, medicalization of sarcopenia would help older persons maintain or even regain functional independence and quality of life — and perhaps boost lifespan, via a reduction in comorbidity with diseases like osteoporosis.

### The problem of causally interrelated disease states

There is another definitional problem: What distinguishes one age-associated disease from another? This may seem like a facetious question, given the obvious symptomatic differences between atherosclerosis and AD. However, as mentioned above, the oxysterol 7KC has been implicated in the pathogenesis of both those disease states. If 7KC is indeed a metabolic byproduct that is causally related to both atherosclerosis and AD then, in addition to being a promising drug target itself, it could conceivably qualify as a surrogate endpoint for clinical trials of new drugs indicated for those diseases. FDA has issued a draft guidance regarding qualification of biomarkers as drug development tools[26], but surrogate endpoints may only be used in lieu of direct measures of clinical benefit under the FDA's "Fast-Track Program," which is only available for new drugs intended for the treatment of a serious or life-threatening condition and that demonstrate the potential to address unmet medical needs for such a condition.[27] However, it would not be necessary to qualify 7KC reduction as a surrogate endpoint for *both* AD and atherosclerosis. Doing so for one or the other based on which is thought to be the more serious condition and/or the greater unmet need would allow its use in a fast-tracked New Drug Application for the one indication, and then if safety and efficacy in humans is established and the therapeutic is approved, data from (likely compulsory) post-marketing studies could be used in a new indication claim for the other condition.

Surrogate endpoints need only be "reasonably likely to predict clinical benefit"[28], and some commentators have pointed out that applying this lower standard to the screening of surrogate endpoints may result in drugs approved on the basis of evidence of an effect on a biomarker which, while related to the disease, is not actually causally related to any clinical benefit.[29] Of course, given its ambitious objective, the SENS Foundation has a strong vested interest in tying 7KC to clinical benefit, and the fact that FDA-qualified biomarkers are released into the public domain also fits within the Foundation's public interest mandate, and could enhance perceptions of the legitimacy of its research goals. But more importantly, it could shorten clinical trials, an oft-criticized source of delay and drug costs. While its work to date has primarily been proof-of-concept research, in the future the SENS Foundation might devote some of its resources to running forms of aging damage like 7KC through the biomarker qualification process. Although publishing both the proof-of-concept *and* such valuable drug development tools might cut out some of the traditional patenting opportunities[30], it also offsets costs ordinarily borne by pharmaceutical companies. A little low-hanging fruit might stir up some productive competition in an industry sometimes criticized for chasing after minor therapeutic improvements and patent trolling.

Another option that is very in line with the social agenda of longevity advocates would be to promote the rebranding of multiple disease states with significantly overlapping low-level chemistry as single unified conditions that present varied symptom groupings based on exposure to particular environmental factors (including the endogenous "environment," like certain genes or epigenetic variations, along with more traditional exogenous contributors like diet, exercise, etc). Admittedly, this would be the more difficult path, because it relies on the two-step process of disease recognition, discussed above, and considering how long it took AD and senile dementia to be folded into AD with an early-onset subtype, trying to replicate

this process with diseases that present as differently as atherosclerosis and AD may be a Sisyphean task. On the other hand, academic pressure of this kind could have trickle-out effects on the public, re-situating the discourse of age-associated diseases further upstream, pressing on towards greater recognition of aging *as* disease.

Finally, slight augmentations to the SENS branding could be in order. Dr. de Grey gave unique names to his proposed *strategies* (LysoSENS, MitoSENS, etc.), but not to the categories of damage which are the *targets* of those strategies. Devising and promoting novel medical names for these categories of damage, like *idiocytotoxicosis*[31] for the "intracellular junk" targeted by LysoSENS, might prompt frame-shifting in the academic and clinical communities that could consequently (albeit indirectly, and thus probably slowly) broaden the scope of "recognized disease or condition". Sadly for the planet, 'junk' doesn't seem to be something humans take terribly seriously — *idiocytotoxicosis*, on the other hand, well that's clearly a monster. Perhaps this suggestion borders on "disease-mongering" — but isn't that term itself equally agenda-driven, given the not-so-subtle association with war-mongering? Dr. de Grey and other longevity advocates consider themselves to be on moral high ground, so these kinds of accusations are only of consequence if they provoke counter-productive public response, and reframing well-recognized diseases like AD and atherosclerosis as symptoms of underlying "metabolic pathology" can hardly be characterized as "questionable new diagnoses — like [premenstrual dysphoric dysfunction] and social anxiety disorder — which are hard to distinguish from normal life," the likes of which give at least one critic concern. [32] And perhaps it is the very idea that "normal" is the ultimate objective — as opposed to simply "better" — that is the problem.

### What's the alternative?

If the perceived burden is too high, and the cost of doing nothing too great, stakeholders may look to circumvent the FDA. The SENS Foundation characterizes the assault on aging as the next space race.

If the U.S. doesn't take action to foster local development of what will assuredly be highly sought-after therapies, the movement may simply move underground

(i.e. further in the vein of DIYbio), and overseas (medical tourism, and seasteads), which will only hamper the FDA's mandate to protect Americans from harm. ■

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## Endnotes

[1]: Aubrey de Grey & Michael Rae, *Ending Aging: The Rejuvenation Breakthroughs That Could Reverse Human Aging in Our Lifetime*, (New York: St Martin's Press, 2007).

[2]: David Gems, "Tragedy and delight: the ethics of decelerated aging" (2011) 366 *Philosophical Transactions of the Royal Society B [Phil Trans R Soc B]* 108 at 110.

[3]: SENS Foundation, *SENS Foundation*, online: <<http://www.sens.org/about-the-foundation>>.

[4]: Jacques M Mathieu et al, "7-Ketocholesterol Catabolism by *Rhodococcus jostii* RHA1" (2010) 76:1 *Applied and Environmental Microbiology* 352.

[5]: Jacques M Mathieu et al, "Increased resistance to oxysterol cytotoxicity in fibroblasts transfected with a lysosomally targeted *Chromobacterium* oxidase" (2012) *Biotechnology and Bioengineering*, online: <<http://www.wileyonlinelibrary.com>> DOI 10.1002/bit.24506.

[6]: SENS Foundation, *Research Report 2011*, online: <<http://images.sens.org/reports/SENS%20Research%20Report%202011.pdf>>.

[7]: *Supra* note 2 at 109.

[8]: 21 USC § 321(g)(1).

[9]: 42 USC § 262(i). The phrase "analogous product" has been used to justify the extension of the FDA's regulatory authority to human cells, tissues, and cellular and tissue-based products (HCT/PS). See also Areta L Kupchyk, "Approval of Products for Human Use" in HB Wellons et al, *Biotechnology and the Law* (ABA, 2007) 591 at 617, note 41

[10]: 21 CFR § 201.57(c)(2) Specifically, this is a labeling requirement, but if a drug cannot be labeled according to the

regulation, the New Drug Application cannot be approved. See also 21 CFR § 201.56.

[11]: The term disease is defined in 21 CFR §101.93(g) for the purposes of disease claims relating to dietary supplements, but that is only applicable in that context. See also 21 USC 343(r)(6).

[12]: William J Evans, "Drug discovery and development for ageing: opportunities and challenges" (2011) 366 *Phil Trans R Soc B* 113 at 114.

[13]: *Ibid* at 114.

[14]: Barbara Mintzes, "Disease Mongering in Drug Promotion: Do Governments Have a Regulatory Role?" (2006) 3:4 *PLoS Medicine* e198.

[15]: Aubrey de Grey, "Combating the Tihonus Error: What Works?" (2008), 11:4 *Rejuvenation Research* 713.

[16]: GE Berrios, "Alzheimer's disease: a conceptual history" (1990) 5:6 *International Journal of Geriatric Psychiatry* 355.

[17]: Robert Katzman et al, *Alzheimer's disease: senile dementia and related disorders* (NY: Raven Press, 1978) at 595.

[18]: Eric P Brass & Kathy E Sietsema, "Considerations in the Development of Drugs to Treat Sarcopenia" (2011) 59:3 *Journal of the American Geriatrics Society* 530.

[19]: *Ibid* at 531.

[20]: *Supra* note 12 at 116.

[21]: Alfonso J Cruz-Jentoft et al, "Sarcopenia: European consensus on definition and diagnosis" (2010) 39:4 *Age and Ageing* 412 (Abstract).

[22]: Roger A Fielding et al, "Sarcopenia: An Undiagnosed Condition in Older Adults. Current Consensus Definition: Prevalence,

Etiology, and Consequences" (2011)12:4 *Journal of the American Medical Doctors Association [JAMDA]* 249 (Abstract).

[23]: See Marco Brotto, "Lessons from the FNIH-NIA-FDA sarcopenia consensus summit" (2012) 9 *IBMS BoneKEy* 210.

[24]: *Supra* note 18 at 531-533.

[25]: *Ibid* at 533. See also Christopher J Evans et al, "Development of a New Patient-Reported Outcome Measure in Sarcopenia" (2011) 12:3 *JAMDA* 226.

[26]: Center for Drug Evaluation and Research, "Guidance for Industry – Qualification Process for Drug Development Tools," *FDA* (October 2010) online: <<http://www.fda.gov/downloads/Drugs/GuidanceComplianceRegulatoryInformation/Guidances/UCM230597.pdf>>.

[27]: 21 USC § 356(a)(1).

[28]: 21 CFR § 314.510.

[29]: Thomas R Fleming, "Surrogate Endpoints And FDA's Accelerated Approval Process" (2005) 24:1 *Health Affairs* 67. See also Thomas R Fleming and David L DeMets, "Surrogate end points in clinical trials: are we being misled?" (1996) 125:7 *Annals of Internal Medicine* 605.

[30]: There may be other intellectual property issues implicated in this shift of paradigm in drug development and regulation, but they are beyond the scope of this article.

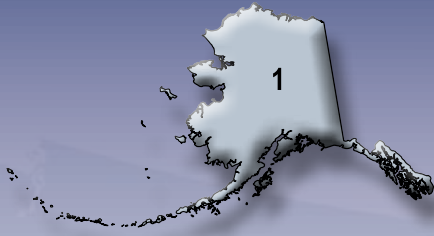
[31]: Meaning "self, one's own" + "cell" + "toxin" + "condition of increase".

[32]: *Supra* note 14 at 0463.

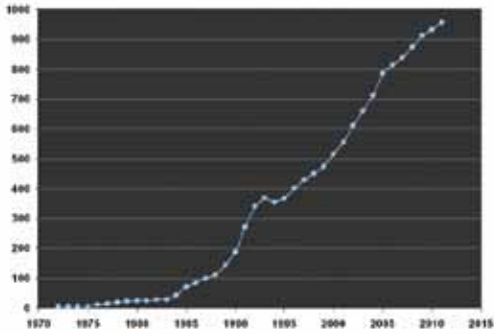
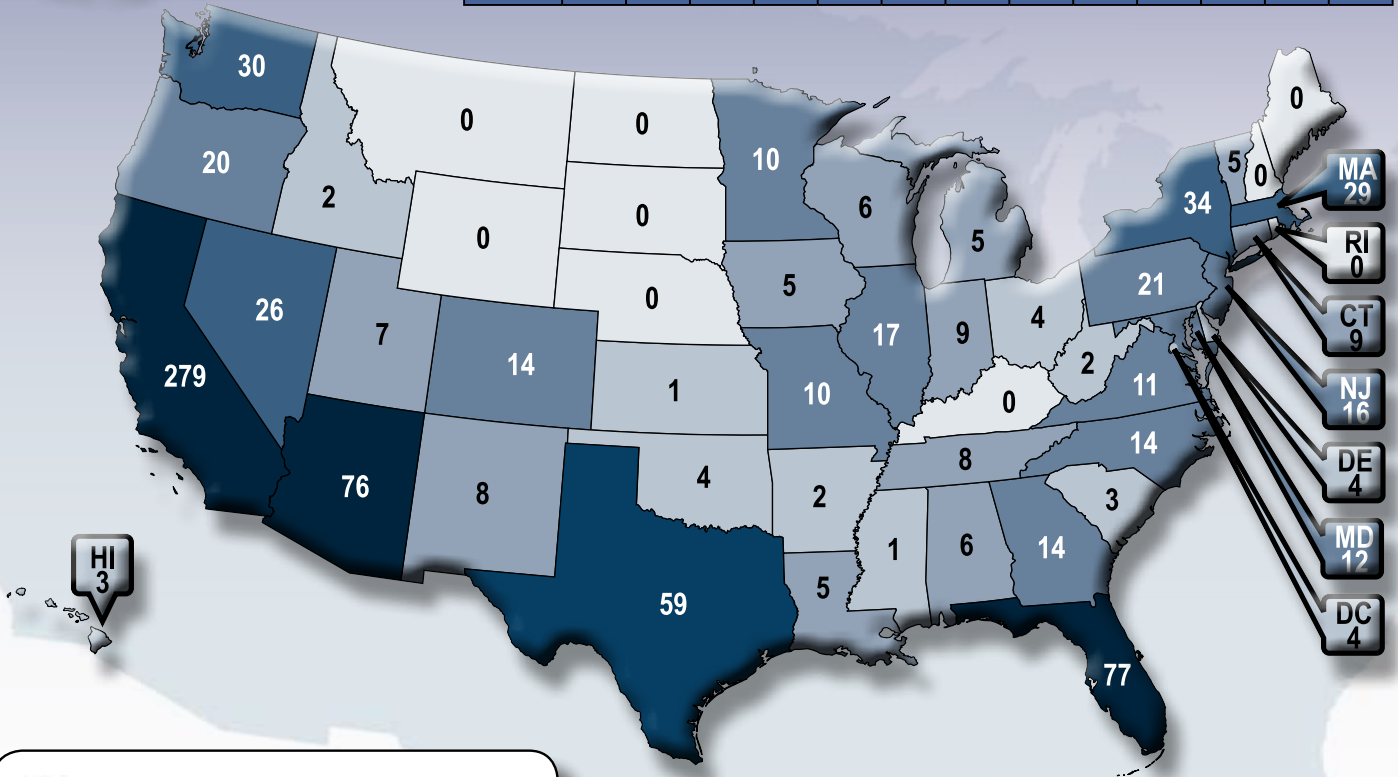
[33]: SENS Foundation, *Annual Report 2011*, online: <<http://www.sens.org/sites/srf.org/files/SENS%20Foundation%20Annual%20Report%202011.pdf>>.



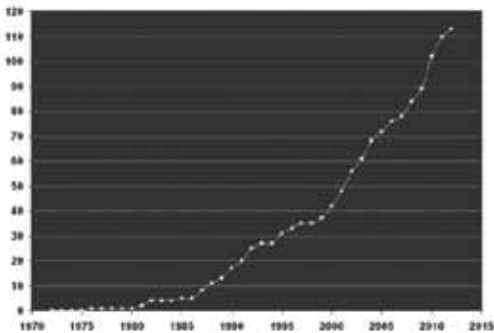
# Membership Statistics



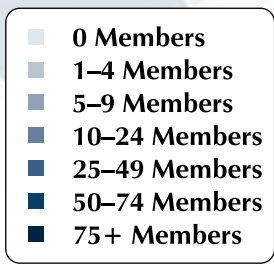
2012	01	02	03	04	05	06	07	08	09	10	11	12	
Members	956	959	963	967	968	974	974	975	975	982	976	980	980
Patients	110	110	111	111	111	111	112	112	112	112	113	113	113
Associate	0	0	0	8	9	13	16	20	21	24	30	33	33
Total	1066	1069	1074	1086	1088	1098	1102	1107	1108	1118	1119	1126	1126



Number of Alcor members



Number of Alcor patients



## International

Country	Members	Patients
Australia	11	2
Canada	42	2
Denmark	2	0
France	1	0
Germany	5	0
Israel	0	1
Italy	1	0
Luxembourg	1	0
Mexico	4	0
Monaco	2	0
Netherlands	2	0
New Zealand	2	0
Portugal	4	0
Spain	2	1
Thailand	3	0
United Arab Emirates	1	0
United Kingdom	24	2
<b>TOTAL</b>	<b>107</b>	<b>8</b>

## JEAN FINOT: PROLONGEVITY ADVOCATE OF THE EARLY TWENTIETH CENTURY

By R. Michael Perry



*“The possibility of the prolongation of our existence well beyond a century, the narrowing of the limits of old age, the removal of the horrors of departure, the resurrection of the body in the many forms of infinite life, are all things which tend to bring peace to our saddened and fearful minds.”<sup>1</sup>*

*“... some fine day we shall destroy the diseases peculiar to old age.”<sup>2</sup>*

—Jean Finot

Jean Finot (1858-1922) was a philosopher and social commentator whose interests ranged from relations among fellow humans to the deep issues of life and death. Overall he was an optimist who advocated peace and world harmony based on equality of races and genders, and urged the use of science and technology for human betterment, including lengthening the life span. Born in Warsaw, Poland to Jewish parents of the surname Finkelhaus, he became a naturalized French citizen and adapted his name, one suspects to avoid some of the very race prejudice he would rebut in one of his major works. He attended the University of Cambridge and the Faculty of Letters in Paris. He edited a number of magazines, most importantly the *Revue des Revues* which, under his direction after 1890, published some of the most provocative social, political, and philosophical studies appearing in the French press of his day. He is most known for his own monographs in the French

language including (titles in English) *Race Prejudice* (1905), *Prejudice and Problems of the Sexes* (1913), *The Science of Happiness* (1913), and *Modern Saints and Seers* (1918).<sup>3</sup>

Here we focus mainly on Finot’s efforts in the field of prolongevity (a term coined in 1955 by Gerald J. Gruman meaning “the significant extension of the length of life by human action”<sup>4</sup>). His two important works in this field were *The Philosophy of Long Life* (1900) and a shorter, posthumously-published sequel, *How to Prolong Life* (1924). In these writings, especially the first, he also delves into other matters of interest to modern transhumanists such as the creation of artificial living organisms, as usual with an optimistic forecast. It was his firm belief that a normal human life span could be increased beyond 150 years if we would only take proper steps. He also foresaw other advances in life prolongation such as the use of cold to extend life by putting living things “on hold” for a period of time then rewarming to reanimate them — the essence of cryonics.

In making his arguments Finot was dutiful and generous in his citations of supporting sources, as would be hoped. Sometimes these appear perfectly reasonable, as in the experiments reported on the use of cold and desiccation to extend the lives of small organisms. Other times, however, his sources are hardly credible and he is to be taken to task for rudely ignoring the maxim, “extraordinary claims require extraordinary evidence.” There are many pages of alleged instances of persons living beyond 150 years, sometimes far beyond, including numerous ancient anecdotes, all presented without question or independent confirmation. (Finot more credibly denies that past generations on average lived longer than present generations and presents evidence that life span was gradually increasing. He also refuses to accept the near-millennial life spans recorded in Genesis, on grounds of recent biblical criticism.) There are other fantastic reports, including that of the creation by a human experimenter of

small intelligent creatures or “homunculi,” which Finot took seriously.

In all, his prolongevity writings are a strange potpourri of the credible and the fantastic; we can imagine that the uncritical reader would be reassured and encouraged, as many must have been. (That *Philosophy of Long Life* was a publishing success is confirmed in the preface to the English edition of 1909. Less than a decade from its original publication the book had gone into 14 editions “as well as being translated into nearly all languages.”<sup>25</sup>) Aging can be counteracted, says Finot, at least for a long time, and why be vexed about death anyway? It happens to us all in the end, yet there is about it an overarching unreality: the soul exists and is immortal, though we know very little about what it is or how it survives; enough seems evident, however, to be hopeful and reassured. Besides, he argues, the body actually “lives on” after death anyway, being the host of numerous soil organisms, which he enumerates in scientific detail and which take their deserved place in the whole of nature. (For this reason he was opposed to cremation and devotes a section of *Philosophy of Long Life* to that topic.)

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*“It was his firm belief that a normal human life span could be increased beyond 150 years if we would only take proper steps.”*

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There are other reasons he urges for not being too exercised about death. What is it that dies anyway? Each day of our lives we are effectively a new person, not the same as we were before, and not the same as we will be after this day, ever. We die all the time — and are reborn. Is not this in some way the overall plan of nature? Is death — any person’s — ever to be considered absolute and final? In all we have reason to seek the good life but be content with what we get if we have put forth a good effort.

In his last work, *How to Prolong Life*, Finot abbreviates the philosophical discourse (though some wild longevity claims are

still left in), and focuses more on practical matters. The advice he gives is sound enough in broad outlines: eat frugally, exercise regularly, and by all means stay active, pressing forward with plans and purposes and accomplishments rather than imploding in idleness. That is the key to living long and well — and science, he reminds us, is the stepping-stone to doing better yet, whatever may have happened in the past.

In summary, Finot might be called an “opportunistic optimist,” choosing his topics and arguments to offer an appealing message to readers of his time who were concerned about death, as we still are today. He approaches his subject from the standpoint of a rationalist philosopher who hardly rules out anything that might lurk in the murky backwaters of our existence, especially if it makes for an appealing point, but is not dogmatically attached to any narrow beliefs. He is open-minded — too much so — but ever cheerful and encouraging. Much of what he says might gain traction with certain readers of today; the sort who are “spiritual but not religious” come to mind. For many transhumanists on the other hand, especially cryonicists, his uncritical assertions will be a bit much. Extraordinary claims *do* demand extraordinary evidence, particularly if they are highly appealing and address our deeper anxieties. Writings that fail to observe this cannot command unlimited respect, but can still be of interest as historical milestones and for other reasons.

In Finot’s case there certainly are “other reasons.” I think most, even among present-day, hard-bitten cryonicists would agree that he was not totally wrong, far from it. His scientific stance especially foreshadows our hopes of deliverance through technology, something however that we still cannot be sure of. And cryonics, even if it works, will not get back the many dead who were not preserved. Finot I think would have quickly seen this, and would have proposed an answer that many today might respect. His heart was in the right place, even if his head at times was a bit off in the clouds. ■

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## Notes

1. Finot, *The Philosophy of Long Life*, v
2. Finot, *op. cit.*, 119
3. Biographical data in this paragraph summarized from Alain Leroy Locke, *Lectures on the Theory and Practice of Race*, n. 120, which cites *Dictionnaire de Biographie Française*, vol. 13 (Paris: Librairie Letouzey et Ane, 1975), 1376-77. Online, 1 Dec. 2012: <http://www.negroartist.com/writings/Race%20Contacts%20and%20Interracial%20Relations.htm>
4. Gerald J. Gruman, “A History of Ideas about the Prolongation of Life.” *Transactions of the American Philosophical Society* 56, no. 9 (December 1966), 6.
5. Finot, *op. cit.*, v

# TAX BENEFITS OF LIFE INSURANCE

By Rudi Hoffman

## EXECUTIVE SUMMARY:

*Life insurance has numerous tax and non-tax benefits. The cash value in a life insurance policy both enables level premiums, and also provides a unique long term savings with safe and relatively high returns. The cash inside the policy can be borrowed out tax free in a "wash loan." Rather than trying to determine the least one can put into a policy, it is smart to "overfund" a policy so it accumulates more internal cash. Death benefits, whether used to fund a cryonics perpetual trust, or go to loved ones, or fund your cryonic suspension, are completely tax free in a properly set up life insurance policy.*

- Growth of dollars inside life insurance is tax free.
- Money inside policies has creditor protection.
- Growth of dollars can be not just tax deferred, but can be tax free.
- Why smart and wealthy people get high tax free returns using life insurance, and how you can be one of them.

Life insurance has some unique tax benefits, which have historically been made available due to its perceived and actual benefit to society.

The following article briefly describes the tax treatment of life insurance, and provides examples of how this may be applied as part of your financial plan. Excuse the necessary disclaimer, but it must be pointed out that the following information is subject to change, represents accurate but generic information, and one should consult a tax advisor for specific information.

## 1. BENEFIT ONE: INCOME TAX FREE DEATH PROCEEDS

Life insurance death benefits are generally tax free. Perhaps you or someone you know has been the beneficiary of a life insurance policy. You may remember the difference a lump sum can make for loved ones and survivors. You may also remember that the lump sum coming from life insurance proceeds is not subject to federal or state income tax. Life insurance proceeds also have the benefit of going directly to the named beneficiary, by operation of law. This enables them to be creditor-proof... that is, not subject to the claims of creditors. Direct naming of beneficiaries also enables one to bypass probate, so there is no delay, uncertainty, or costs associated with this potentially long process.

In nearly every instance, barring unusual corporate funded policies, the death benefit of a life insurance is a tax free event. Think what a difference this makes to the beneficiaries! Instead of having an extra \$300,000 or \$500,000 added to their taxable income, the whole amount of the life insurance proceeds goes directly where you want it to go, exactly when it is needed.

## 2. BENEFIT TWO: ESTATE TAX FREE DEATH PROCEEDS

Life insurance proceeds in a properly drafted program are also not subject to estate tax, a separate and potentially devastating tax.

A life insurance policy can be structured to pay a tax free death benefit to a cryonics personal revival trust. In fact, nearly all well designed cryonics trusts have a dedicated life insurance policy which

enables enormous leverage of the dollars committed to the trust. This means that a cryonics trust funded with a million dollars may well be affordable to many cryonicists. How exciting is that? Even an extra \$100,000 policy could grow and compound into millions over time. This could mean the difference between resuscitation happening or not, or perhaps a huge difference in your future choices.

By having a life insurance policy which funds a cryonics trust, you get the leverage of life insurance AND a tax free death benefit AND the money does not reduce what you have going to your loved ones! This is simply a smart way of structuring your arrangements.

## 3. BENEFIT THREE: TAX FREE ACCUMULATION OF DOLLARS INSIDE THE POLICY

On permanent policies...whole life and universal life type policies, there is an internal cash accumulation which enables the policies to maintain a levelized premium. This cash value grows at rates of return generally 2-4% higher than equivalent "guaranteed, ultra low risk" investment options like bank CDs, money market funds, and investment grade bonds.

However, unlike bank Certificates of Deposits, savings accounts, and even bond funds, there is a tax benefit unique to life insurance. The cash values of life insurance are NOT taxed as they grow. Perhaps more importantly, these values can be WITHDRAWN on a tax-free basis by accessing the dollars through a policy loan. Although the policy loan provisions generally allow for a so-called "Wash"

interest rate...i.e. the interest paid on the loan is credited back to you at about the same rate you pay to borrow the money... this is still a legitimate LOAN in the eyes of the IRS.

What this means in practice is that you can have ten thousand dollars of growth in your cash value of your life insurance, borrow the dollars from the policy, and have zero tax on both growth and even withdrawal of the dollars.

Let's contrast this with a traditional Individual Retirement Account, for instance. While IRA contributions are deducted from your taxable income and life insurance premiums are not, there is a difference in how the cash accumulation is taxed when you actually GET and USE the money.

When you withdraw the dollars from your IRA, they are fully taxable at the then current rates. Do you think tax rates will probably be higher or lower in the future? While the definite answer to this question may be unknown, it may be instructive to note that the top tax rates have in the past been much higher than they are now.

In contrast, dollars can be withdrawn TAX FREE from a life insurance policy. Conceptually, this is more like the tax considerations of a ROTH Individual Retirement Account. However, the Roth has some limitations and penalties that a life insurance policy is not subject to. For instance, a Roth has contribution limits, is disallowed for higher wage earners, and has penalties for withdrawal prior to age 59.5. Life insurance is not subject to any of these limitations.

There IS the tradeoff of the internal life insurance cost being deducted from the cash accumulation. However, this internal cost is also what enables the policy to be taxed as life insurance, and create the tax free lump sum that can leverage the dollars put into a policy by many times the amount put into the policy. Additionally, there are disincentives to early withdrawal of life insurance cash values, to help us remember that they are designed as long

term programs and not short term "put and take" accounts.

#### 4. BENEFIT FOUR: ABILITY TO ADJUST AMOUNTS OF CONTRIBUTIONS

Many higher earners are phased out of traditional tax-advantaged plans like traditional IRA plans as well as Roth IRA plans. Even if you are not one of these people, the maximum you can put into an IRA or a ROTH is \$5,000 a year (plus a \$1,000 a year "catch up" if you are over 50).

What if you want to put \$25,000 into a plan this year, and zero the next, and still want tax advantaged growth? A life insurance plan will let you do this.

#### 5. ABILITY TO BECOME SELF COMPLETING

While not technically a tax benefit, a life insurance policy is the only savings program that creates a huge lump sum immediately upon pronouncement of legal "death." If you are saving up to have a million dollars at age 68, but you die in a car crash at age 32, only one plan type can create that large lump sum for your loved ones or to fund your cryostasis. Your banker won't say, "Well, I am pretty sure Harry would have continued his contributions of \$8,000 a year, so here is the million dollars he would have had at age 68." Neither will your stockbroker, or your fund manager.

#### 6. SOME CONSIDERATIONS ABOUT TAX BENEFITS OF LIFE INSURANCE

Like any legitimate program, you have to "play by the rules" to utilize the benefits of life insurance.

In order to be taxed as life insurance and not taxed like an annuity, a life insurance policy must meet certain criteria established by the IRS. You don't need to know the technical criteria, because these are already built into the software illustrations insurance companies provide, but the basic concept is this.

A. There is relationship between the face amount of the policy and the premium put in, which defines whether a policy allows both tax deferred growth and tax free withdrawal of the cash value.

You can put a SINGLE PREMIUM into a policy, for instance, which will immediately pay the policy up...i.e., the death benefit will be paid and no further premiums are required.

However, this policy will be considered a MEC, a "Modified Endowment Contract." This is not a scary or bad thing, it simply means the following. The tax treatment of a MEC is that one cannot borrow the cash value out in a tax-free way.... However, the death benefit of a MEC is STILL tax free!

In order to avoid being a MEC, it is possible to pay a policy up in seven years. This will meet an IRS guideline called, not surprisingly, the "seven pay test" which defines whether a life insurance policy can have the tax benefits of a life insurance policy.

B. Many cryonicists have a "Guaranteed Universal Life" policy to fund their cryonics needs. This is an appropriate funding vehicle, because the policy has an intrinsic built in GUARANTEE that the death benefit will never go down, and the premium never increase, all the way to age 120.

However, if we have simply asked the computer to generate the LOWEST premium that will enable the policy to stay in place with a level premium even with "worst case" assumptions, the policy will generally not generate much cash value. This does not mean you have a bad policy. It simply means you are paying the lowest premium to get the guaranteed death benefit you want.

Recall from your experience or reading my other articles, there are TWO illustrations shown in most Universal Life policy illustrations. One, usually the left three columns, shows what happens if two worst case scenarios were to occur and would continue to occur every year. This is the "Guaranteed" side of the illustration. The "Current", sometimes called "nonguaranteed" side of the illustration shows what the insurance company is ACTUALLY doing now, the actual interest rate they are crediting to the cash value.

Because prevailing interest rates remain at historic lows, the guaranteed rates are currently at 3% (some companies have gone to 2%) while the current rates may be 3-5%.

C. The bottom line is this: Rather than thinking "what is the LEAST I am to put into my policy?" wealthy people have historically and continue to ask "what is the MOST money I can afford to put into this policy, to enable the cash value to grow at excellent, risk free and tax free rates?"

**7. AN EXAMPLE OF CASH VALUE BUILDING POLICY**

Mr. G. is a software engineer, aged 30, preferred nontobacco, making good money and wanting a smart place to put it for later use. While we can't use the name, this is an actual policy and situation from the real world.

Mr. G can invest \$10,000 a year for seven years in a cash rich no-load Universal Life plan. In addition to the immediate benefit of \$500,000 of life insurance, an amount that actually INCREASES over time as the cash accumulation grows, he has, even if interest rates never rise, a remarkable \$363,000 he can withdraw TAX FREE at his age 70. In later years it goes to millions. Because it happens to be a cryonics policy, this increase in cash value ALSO increases the death benefit, so he has a policy that keeps up with and even outpaces inflation!

He will have even more if he elects to continue to invest in the program after seven years, but the above figure assumes he stops and never contributes another dime to the plan.

His dollars are creditor protected, do not reduce contributions he can make to other retirement plans, growing at an effective rate of 4.5% tax free, in a risk free, worry free, hassle free manner. If the future interest rates go up, his credited rate will also rise commensurately to maintain a rate about 3% higher than most bank rates.

**IN SUMMARY**

Life insurance remains one of the last genuine tax shelters available. Tax law changes have reduced other kinds of tax shelter investments, while increasing many people's effective tax rate. Our questions to our financial planners and life insurance brokers should not just be "What is the least

I can pay?" but now should include "What is the most I can put into this program?" ■



**Rudi Hoffman**  
CFP CLU ChFC  
is the leading writer of funding programs for cryopreservation in the world.  
Personally signed up with Alcor

since 1994, he specializes in making the occasionally daunting world of insurance and cryonics funding as straightforward, understandable, and affordable as possible.

With over 500 million dollars of life insurance written since over 34 years, Rudi's main goal is to make the cryonics option accessible for a much larger segment of the population.

**A Life Insurance Policy Illustration**  
Flexible Premium Adjustable Death Benefit Universal Life Insurance

		Guaranteed Assumptions Guaranteed Interest 3.00 %			Not Guaranteed Current Interest 3.75 %			
Age	Year	Premium Outlay	Accum Value	Cash Surrender Value	Death Benefit	Accum Value	Cash Surrender Value	Death Benefit
<b>George Goodfellow</b> Male, Age 30 Prof. Elite Nontobacco Init DB: \$500,000.00								
<b>Issue State:</b> FL								
<b>Initial Death Benefit:</b> \$500,000.00								
<b>Initial Death Benefit Coverage Option:</b> Option A								
<b>Initial Premium:</b> \$10,000.00 Annual								
<b>Riders/Benefits on Primary Insured:</b> LBR								
<b>Defra Option:</b> CVAT								
31	1	10,000.00	9,378	3,063	500,000	9,863	3,548	500,000
32	2	10,000.00	19,053	13,054	500,000	20,153	14,153	500,000
33	3	10,000.00	29,028	23,029	500,000	30,864	24,865	500,000
34	4	10,000.00	39,288	33,362	500,000	42,015	36,079	500,000
35	5	10,000.00	49,879	43,943	500,000	53,621	47,685	500,000
36	6	10,000.00	60,775	54,839	500,000	65,691	59,755	500,000
37	7	10,000.00	71,984	66,300	500,000	78,235	72,551	500,000
38	8	0.00	73,209	67,528	500,000	80,864	75,181	500,000
39	9	0.00	74,434	68,813	500,000	83,568	77,947	500,000
40	10	0.00	75,662	70,168	500,000	86,352	80,858	500,000
41	11	0.00	76,890	72,280	500,000	89,219	84,609	500,000
42	12	0.00	78,106	74,443	500,000	92,177	88,514	500,000
43	13	0.00	79,296	76,517	500,000	95,250	92,471	500,000
44	14	0.00	80,451	78,620	500,000	98,446	96,615	500,000
45	15	0.00	81,558	80,611	500,000	101,770	100,823	500,000
46	16	0.00	82,603	82,603	500,000	105,211	105,211	500,000
47	17	0.00	83,589	83,589	500,000	108,747	108,747	500,000
48	18	0.00	84,506	84,506	500,000	112,380	112,380	500,000
49	19	0.00	85,394	85,394	500,000	126,279	126,279	500,000
50	20	0.00	86,244	86,244	500,000	142,972	142,972	500,000
51	21	0.00	87,026	87,026	500,000	148,993	148,993	500,000
52	22	0.00	87,721	87,721	500,000	155,248	155,248	500,000
53	23	0.00	88,284	88,284	500,000	161,740	161,740	500,000
54	24	0.00	88,700	88,700	500,000	168,474	168,474	500,000
55	25	0.00	88,917	88,917	500,000	175,455	175,455	500,000
56	26	0.00	88,878	88,878	500,000	182,638	182,638	500,000
57	27	0.00	88,570	88,570	500,000	190,074	190,074	500,000
58	28	0.00	87,962	87,962	500,000	197,773	197,773	500,000
59	29	0.00	87,084	87,084	500,000	205,746	205,746	500,000
60	30	0.00	85,886	85,886	500,000	213,998	213,998	500,000
61	31	0.00	84,296	84,296	500,000	222,537	222,537	500,000
62	32	0.00	82,219	82,219	500,000	231,369	231,369	500,000
63	33	0.00	79,538	79,538	500,000	240,500	240,500	500,000
64	34	0.00	76,155	76,155	500,000	249,933	249,933	500,000
65	35	0.00	72,000	72,000	500,000	259,676	259,676	500,000
66	36	0.00	66,993	66,993	500,000	269,497	269,497	500,000
67	37	0.00	61,068	61,068	500,000	279,614	279,614	500,000
68	38	0.00	54,154	54,154	500,000	290,010	290,010	503,272
69	39	0.00	46,124	46,124	500,000	300,672	300,672	508,992
70	40	0.00	36,866	36,866	500,000	311,606	311,606	514,743

*\* 10,000 A YEAR FOR SEVEN YEARS*

*\*\* CASH VALUE AGE 50*

*\*\*\* DEATH BENEFIT MAY INCREASE AS SHOWN*

## Hybrid Tunnel May Help Guide Severed Nerves Back to Health

Building a tunnel made up of both hard and soft materials to guide the reconnection of severed nerve endings may be the first step toward helping patients who have suffered extensive nerve trauma regain feeling and movement, according to a team of biomedical engineers. The researchers, who published their results in the current issue of *Advanced Healthcare Materials*, developed a novel hybrid conduit that consisted of a soft material, called a hydrogel, as an external wall along with an internal wall made of an electrically-active conducting polymer to serve as a tunnel that guides the regrowth and reconnection of the severed nerve endings. The method could offer advantages over current surgeries that are used to reconnect severed nerves according to Mohammad Reza Abidian, assistant professor of biomedical engineering, Penn State.

Pennsylvania State University  
17 Dec. 2012  
<http://live.psu.edu/story/63267>

## Study Unmasks Regulator of Healthy Life Span

A new series of studies in mouse models by Mayo Clinic researchers uncovered that the aging process is characterized by high rates of whole-chromosome losses and gains in various organs, including heart, muscle, kidney and eye, and demonstrates that reducing these rates slows age-related tissue deterioration and promotes a healthier life span. The findings appear in today's online issue of *Nature Cell Biology*. "We've known for some time that reduced levels of BubR1 are a hallmark of aging and correspond to age-related conditions, including muscle weakness, cataract formation and tumor growth," says co-author Jan van Deursen, Ph.D., of Mayo Clinic. "Here we've shown that a high abundance of BubR1, a regulator

of chromosome segregation during mitosis, preserves genomic integrity and reduces tumors, even in the face of some genetic alterations that promote inaccurate cell division. Our findings suggest that controlling levels of this regulator provides a unique opportunity to extend healthy life span."

Mayo Clinic (Minnesota)  
17 Dec. 2012  
<http://www.mayoclinic.org/news2012-rst/7212.html>

## First Map of How the Brain Organizes Everything We See

Our eyes may be our window to the world, but how do we make sense of the thousands of images that flood our retinas each day? Scientists at the University of California, Berkeley, have found that the brain is wired to put in order all the categories of objects and actions that we see. They have created the first interactive map of how the brain organizes these groupings. The result — achieved through computational models of brain imaging data collected while the subjects watched hours of movie clips — is what researchers call "a continuous semantic space." The researchers found that different people share a similar semantic layout. "Our methods open a door that will quickly lead to a more complete and detailed understanding of how the brain is organized. Already, our online brain viewer appears to provide the most detailed look ever at the visual function and organization of a single human brain," said Alexander Huth, a doctoral student in neuroscience at UC Berkeley and lead author of the study published Dec. 19 in the journal *Neuron*.

Yasmin Anwar, Media Relations,  
UC Berkeley  
19 Dec. 2012  
<http://newscenter.berkeley.edu/2012/12/19/semanticspace/>

## A Nanoscale Window to the Biological World

Investigators at the Virginia Tech Carilion Research Institute have invented a way to directly image biological structures at their most fundamental level and in their natural habitats. The technique is a major advance toward the ultimate goal of imaging biological processes in action at the atomic level. "It's sort of like the difference between seeing Han Solo frozen in carbonite and watching him walk around blasting stormtroopers," said Deborah Kelly, an assistant professor at the VTC Research Institute and a lead author on the paper describing the first successful test of the new technique. "Seeing viruses, for example, in action in their natural environment is invaluable." The technique involves taking two silicon-nitride microchips with windows etched in their centers and pressing them together until only a 150-nanometer space between them remains. The researchers then fill this pocket with a liquid resembling the natural environment of the biological structure to be imaged, creating a microfluidic chamber, with antibody "tethers" to hold viruses in place for observation.

Ken Kingery, Virginia Tech Carilion  
Research Institute  
20 Dec. 2012  
<http://research.vtc.vt.edu/news/2012/dec/20/nanoscale-window-biological-world/>

## Eyes May Provide a Look into Multiple Sclerosis Progression

New research suggests that thinning of a layer of the retina in the eyes may show how fast multiple sclerosis (MS) is progressing in people with the disease. The study is published in the January 1, 2013, online issue of *Neurology*. "This study suggests that retinal thinning, measured by in-office eye scans, called OCT, may occur

at higher rates in people with earlier and more active MS,” said Robert Bermel, MD, with the Cleveland Clinic Mellen Center for MS and a member of the American Academy of Neurology, who wrote an accompanying editorial. For the study, 164 people with MS from the Johns Hopkins MS Center, including 59 who had no disease activity, underwent eye scans that measured thinning of a portion of their retinas every six months for an average of 21 months. Participants were also given MRI brain scans at the start of the study and yearly. The study found that people with MS relapses had 42 percent faster thinning than people with MS who had no relapses.

American Academy of Neurology  
26 Dec. 2012  
<http://www.aan.com/press/index.cfm?fuseaction=release.view&release=1125>

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## New MRI Method May Help Diagnose Dementia

A new way to use MRI scans may help determine whether dementia is Alzheimer’s disease or another type of dementia, according to new research published in the December 26, 2012, online issue of *Neurology*. Alzheimer’s disease and frontotemporal lobar degeneration (FTLD) often have similar symptoms, even though the underlying disease process is much different. “Diagnosis can be challenging,” said study author Corey McMillan, PhD, of the Perelman School of Medicine and Frontotemporal Degeneration Center at the University of Pennsylvania. “If the clinical symptoms and routine brain MR are equal, an expensive positron emission tomography (PET) scan might be needed. Or, a lumbar puncture, which involves inserting a needle into the spine, would be needed to help make the diagnosis. Analysis of the cerebrospinal fluid gives us reliable diagnostic information, but this is not something patients look forward to and is also expensive. Using this new MRI method is less expensive and definitely less invasive.”

American Academy of Neurology  
26 Dec. 2012  
<http://www.aan.com/press/index.cfm?fuseaction=release.view&release=1126>

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## Compound Restores Memory Loss and Reverses Symptoms of Alzheimer’s

A new ray of hope has broken through the clouded outcomes associated with Alzheimer’s disease. A new research report published in the January 2013 print issue of the *FASEB Journal* by scientists from the National Institutes of Health shows that when a molecule called TFP5 is injected into mice with disease that is the equivalent of human Alzheimer’s, symptoms are reversed and memory is restored—without obvious toxic side effects. “We hope that clinical trial studies in AD patients should yield an extended and a better quality of life as observed in mice upon TFP5 treatment,” said Harish C. Pant, Ph.D., a senior researcher involved in the work from the Laboratory of Neurochemistry at the National Institute of Neurological Disorders and Stroke at the National Institutes of Health in Bethesda, MD. “Therefore, we suggest that TFP5 should be an effective therapeutic compound.” To make this discovery, Pant and colleagues used mice with a disease considered the equivalent of Alzheimer’s. One set of these mice were injected with the small molecule TFP5, while the other was injected with saline as placebo.

Federation of American Societies for Experimental Biology / EurekAlert  
2 Jan. 2013  
[http://www.eurekalert.org/pub\\_releases/2013-01/foas-pcr010213.php](http://www.eurekalert.org/pub_releases/2013-01/foas-pcr010213.php)

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## First Known “Social Chromosome” Found

To humans, all fire ants may look alike. But the tiny, red, stinging bugs known as *Solenopsis invicta* have two types of social

organization, and these factions are as recognizable to the ants as rival football teams are to us. Researchers once thought that the groups’ distinct physiological and behavioral profiles stemmed from a variant in a single gene. Now, a new study provides the first evidence that the gene in question is bound up in a bundle of some 600 other genes, versions of which are all inherited together. This “supergene” takes up a large chunk of what may be the first known social chromosome, analogous to the chromosomes that determine sex in humans. The differences between the two types of fire ants start with the winged queens, according to evolutionary geneticist Laurent Keller of the University of Lausanne in Switzerland. “This is a spectacular piece of work,” says University of Georgia, Athens geneticist Kenneth Ross, who was not involved in the study. “They’ve unlocked a whole new mechanism for how a supergene can determine something as complex as behavior.”



*Is rivalry in the genes?  
Differences in fire ant  
groups are governed by  
“social chromosome.”  
Credit: Alex Wild/Visuals  
Unlimited Inc./SPL*

Elizabeth Norton / Science  
16 Jan. 2013  
<http://news.sciencemag.org/sciencenow/2013/01/first-known-social-chromosome-fo.html?ref=hp>



# PURE, HEART HEALTHY Super Omega-3 EPA/DHA



Item # 01482

There's no debating the power of **omega-3** fatty acids. From support for **heart health** and **brain function** to help with **inflammation**, their broad-spectrum benefits have been firmly established in a wealth of studies.<sup>1-9</sup>

To ensure the purest, most stable, and easy-to-tolerate fish oil supplement, **SUPER OMEGA-3 EPA/DHA** is *molecularly distilled*. This proprietary technology ensures any environmental pollutants are reduced to extremely low levels. The result? Our fish oil enjoys a **5-star rating for purity, quality, and concentration** from the **International Fish Oil Standards** program (IFOS)—the highest possible ranking from the world's *premier* testing laboratory.

## Sesame Lignans and Standardized Olive Fruit Extract for Enhanced Benefits

Fish oils (and other fatty acids) have a tendency to **oxidize**, rendering them nutritionally inferior. Scientific studies show that when added to fish oil, **sesame lignans** safeguard against oxidation **and** direct fatty acids toward pathways that help with inflammatory reactions.<sup>10</sup>

To further emulate the benefits of a **Mediterranean diet**, **Super Omega-3** delivers standardized, high-potency **olive fruit extract**. Research shows that **fish oil** combined with **olive oil** helps with inflammation **better** than fish oil alone.<sup>11</sup>

Olive also contains the compounds **hydroxytyrosol**, **tyrosol**, and **oleuropein**. Together these nutrients counter the action of free radicals, delay aging in specialized skin cells, prevent undesirable LDL oxidation, and help maintain normal platelet activation.<sup>12-15</sup>

**Super Omega-3** (4 regular size softgels) supplies the equivalent content of **6 tablespoons of extra virgin olive oil**. Take **two** softgels twice daily with meals.

A bottle containing 120 softgels of **Super Omega-3 EPA/DHA with Sesame Lignans and Olive Fruit Extract** retails for \$32. If a member buys four bottles, the price is reduced to **\$21** per bottle. If **10 bottles** are purchased, the cost is **\$18.68** per bottle. (Item # 01482)

Just one serving of **SUPER OMEGA-3 EPA/DHA** with Sesame Lignans & Olive Fruit Extract provide:

<b>EPA Pure+™ Extract</b> (eicosapentaenoic acid)	<b>1400 mg</b>
<b>DHA Pure+™ Extract</b> (docosahexaenoic acid)	<b>1000 mg</b>
<b>Olive Fruit Extract</b> [std. to 6.5% polyphenols (39 mg), 1.73% hydroxytyrosol/tyrosol (10.4 mg), 0.5% verbascoside/oleuropein (3 mg)]	<b>600 mg</b>
<b>Sesame Seed Lignan Extract</b>	<b>20 mg</b>

## A SMALLER SOFTGEL for easier swallowing!

Some members have requested we make **Super Omega-3** available in a smaller capsule for easier swallowing. We have accomplished this by making **half-size softgels** available.

A bottle containing 240 half-size softgels of **Super Omega-3 EPA/DHA with Sesame Lignans and Olive Fruit Extract** retails for \$32. If a member buys four bottles, the price is reduced to **\$21** per bottle. If **10 bottles** are purchased, the cost is **\$18.68** per bottle. (Item # 01619)

For those with sensitive stomachs, **Super Omega-3** is also available with **enteric coating** and retails for **\$34**. If a member buys four bottles, the price is reduced to **\$23.25** per bottle. If **10 bottles** are purchased, the cost is **\$21** per bottle. (Item # 01484)

To order the most advanced fish oil supplement, **Super Omega-3 EPA/DHA with Sesame Lignans and Olive Fruit Extract** (with or without enteric coating), call **1-800-544-4440** or visit **www.LifeExtension.com**



Ratings based on results of the 2012 ConsumerLab.com Survey of Supplement Users. More information at [www.consumerlab.com](http://www.consumerlab.com).

**CAUTION:** If you are taking anti-coagulant or anti-platelet medications, or have a bleeding disorder, consult your healthcare provider before taking this product. Contains fish (anchovy, mackerel), sesame, and corn.

Supportive but not conclusive evidence shows that consumption of EPA and DHA omega-3 fatty acids may reduce the risk of coronary heart disease. IFOS™ certification mark is a registered trademark of Nutrasource Diagnostics, Inc. These products have been tested to the quality and purity standards of the IFOS™ program conducted at Nutrasource Diagnostics, Inc.

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These statements have not been evaluated by the Food and Drug Administration. These products are not intended to diagnose, treat, cure or prevent any disease.

# MEETINGS

## About the Alcor Foundation

The Alcor Life Extension Foundation is a nonprofit tax-exempt scientific and educational organization dedicated to advancing the science of cryopreservation and promoting cryonics as a rational option. Being an Alcor member means knowing that—should the worst happen—Alcor's Emergency Response Team is ready to respond for you, 24 hours a day, 365 days a year.

Alcor's Emergency Response capability includes specially trained technicians and customized equipment in Arizona, northern California, southern California, and south Florida, as well as many additional certified technicians on-call around the United States. Alcor's Arizona facility includes a full-time staff, and the Patient Care Bay is personally monitored 24 hours a day.

## ARIZONA

### Flagstaff:

Arizona without the inferno. Cryonics group in beautiful, high-altitude Flagstaff. Two-hour drive to Alcor. Contact [eric@flagstaffcryo.com](mailto:eric@flagstaffcryo.com) for more information.

### Scottsdale:

This group meets the third Friday of each month and gatherings are hosted at a home near Alcor. To RSVP, visit <http://cryonics.meetup.com/45/>.

### At Alcor:

Alcor Board of Directors Meetings and Facility Tours — Alcor business meetings are generally held on the first Saturday of every month starting at 11:00 AM MST. Guests are welcome. Facility tours are held every Tuesday and Friday at 2:00 PM. For more information or to schedule a tour, call D'Bora Tarrant at (877) 462-5267 x101 or email [dbora@alcor.org](mailto:dbora@alcor.org).

The Alcor Volunteer Network, Scottsdale Chapter has a variety of meetings on topics including: member education, training, community outreach, and fundraising. To RSVP, visit: <http://www.meetup.com/AVNScottsdale/members/>

## CALIFORNIA

### Los Angeles:

Alcor Southern California Meetings—For information, call Peter Voss at (310) 822-4533 or e-mail him at [peter@optimal.org](mailto:peter@optimal.org).

Although monthly meetings are not held regularly, you can meet Los Angeles Alcor members by contacting Peter.

### San Francisco Bay:

Alcor Northern California Meetings are held quarterly in January, April, July, and October. A CryoFeast is held once a year. For information on Northern California meetings, call Mark Galeck at (408) 245-4928 or email [Mark\\_galeck@pacbell.net](mailto:Mark_galeck@pacbell.net).

## FLORIDA

Central Florida Life Extension group meets once a month in the Tampa Bay area (Tampa and St. Petersburg) for discussion and socializing. The group has been active since 2007. Email [arcturus12453@yahoo.com](mailto:arcturus12453@yahoo.com) for more information.

## NEW ENGLAND

### Cambridge:

The New England regional group strives to meet monthly in Cambridge, MA — for information or to be added to the Alcor NE mailing list, please contact Bret Kulakovich at 617-824-8982, [alcor@bonfireproductions.com](mailto:alcor@bonfireproductions.com), or on FACEBOOK via the Cryonics Special Interest Group.

## PACIFIC NORTHWEST

Cryonics Northwest holds regular meetings for members of all cryonics organizations living in the Pacific Northwest.

For information about upcoming meetings and events go to: <http://www.facebook.com/cryonics.northwest>

A Yahoo mailing list is also maintained for cryonists in the Pacific Northwest at <http://tech.groups.yahoo.com/group/CryonicsNW/>.

## British Columbia (Canada):

The contact person for meetings in the Vancouver area is Keegan Macintosh: [keegan.macintosh@me.com](mailto:keegan.macintosh@me.com)

## Oregon:

The contact person for meetings in the Portland area is Chana de Wolf: [chana.de.wolf@gmail.com](mailto:chana.de.wolf@gmail.com)

## ALCOR PORTUGAL

Alcor Portugal is working to have good stabilization and transport capabilities. The group meets every Saturday for two hours. For information about meetings, contact Nuno Martins at [n-martins@n-martins.com](mailto:n-martins@n-martins.com). The Alcor Portugal website is: [www.alcorportugal.com](http://www.alcorportugal.com).

## TEXAS

### Dallas:

North Texas Cryonauts, please sign up for our announcements list for meetings (<http://groups.yahoo.com/group/cryonauts-announce>) or contact David Wallace Croft at (214) 636-3790 for details of upcoming meetings.

### Austin/Central Texas:

We meet at least quarterly for training, transport kit updates, and discussion. For information: Steve Jackson, 512-447-7866, [sj@sjgames.com](mailto:sj@sjgames.com).

## UNITED KINGDOM

There is an Alcor chapter in England. For information about meetings, contact Alan Sinclair at [cryoservices@yahoo.co.uk](mailto:cryoservices@yahoo.co.uk). See the web site at [www.alcor-uk.org](http://www.alcor-uk.org).

If you are interested in hosting regular meetings in your area, contact Alcor at 877-462-5267, ext. 113. Meetings are a great way to learn about cryonics, meet others with similar interests, and introduce your friends and family to Alcor members!

# WHAT IS CRYONICS?

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Cryonics is an attempt to preserve and protect human life, not reverse death. It is the practice of using extreme cold to attempt to preserve the life of a person who can no longer be supported by today's medicine. Will future medicine, including mature nanotechnology, have the ability to heal at the cellular and molecular levels? Can cryonics successfully carry the cryopreserved person forward through time, for however many decades or centuries might be necessary, until the cryopreservation process can be reversed and the person restored to full health? While cryonics may sound like science fiction, there is a basis for it in real science. The complete scientific story of cryonics is seldom told in media reports, leaving cryonics widely misunderstood. We invite you to reach your own conclusions.

# HOW DO I FIND OUT MORE?

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The Alcor Life Extension Foundation is the world leader in cryonics research and technology. Alcor is a non-profit organization located in Scottsdale, Arizona, founded in 1972. Our website is one of the best sources of detailed introductory information about Alcor and cryopreservation ([www.alcor.org](http://www.alcor.org)). We also invite you to request our FREE information package on the "Free Information" section of our website. It includes:

A fully illustrated color brochure

- A sample of our magazine
- An application for membership and brochure explaining how to join
- And more!

**Your free package should arrive in 1-2 weeks.**

(The complete package will be sent free in the U.S., Canada, and the United Kingdom.)

# HOW DO I ENROLL?

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Signing up for a cryopreservation is easy!

**Step 1:** Fill out an application and submit it with your \$150 application fee.

**Step 2:** You will then be sent a set of contracts to review and sign.

**Step 3:** Fund your cryopreservation. While most people use life insurance to fund their cryopreservation, other forms of prepayment are also accepted. Alcor's Membership Coordinator can provide you with a list of insurance agents familiar with satisfying Alcor's current funding requirements.

**Finally:** After enrolling, you will wear emergency alert tags or carry a special card in your wallet. This is your confirmation that Alcor will respond immediately to an emergency call on your behalf.

**Call toll-free today to start your application:**

**877-462-5267 ext. 132**

**[info@alcor.org](mailto:info@alcor.org)**

**[www.alcor.org](http://www.alcor.org)**





# Will You Be Alive and Healthy 10...20...30 Years from now?

Your best chance at achieving future immortality is to protect your precious health now so you can benefit from future medical breakthroughs. Staying informed about the latest health discoveries can mean the difference between life and premature death.

And the **Life Extension Foundation** can be your passport to the future. As the largest anti-aging organization in the world, we are dedicated to finding scientific ways to prevent disease, slow aging, and eventually stop death.

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- **A subscription to *Life Extension* magazine** (\$59.88 yearly newsstand value)...Over 100 full-color pages every month are filled with medical research findings, scientific reports, and practical guidance about using diet, nutrients, hormones, and drugs to prevent disease and slow aging.
- Access to a toll-free phone line to speak with **knowledgeable health advisors**, including naturopathic doctors, nutritionists, and a cancer expert, about your individual health concerns. You can also receive help in developing your own personal life extension program.
- **Discounts on prescription drugs, blood tests, and pharmaceutical quality supplements** that will greatly exceed your membership dues. You'll receive a directory listing

the latest vitamins and supplements, backed by scientific research and available through a unique buyers club.

**FREE BONUS!**

- ***Disease Prevention and Treatment* book** (\$49.95 cover price)...this hardbound fourth edition provides novel information on complementary therapies for 133 diseases and illnesses—from Alzheimer's disease to cancer, from arthritis to heart disease—that is based on thousands of scientific studies.

Life Extension Foundation funds advanced vitrification and gene-chip research. Your \$75 membership fee helps support scientific projects that could literally save your life.

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