

Aging On Hold

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Peter Gorner and Ronald Kotulak for the Chicago
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Scientists try to tame molecular "shark's"

By Peter Gorner
and Ronald Kotulak
Chicago Tribune

Aging On Hold

Bethesda, MD. - They zip madly through the cellular sea of our bodies and rip and tear at our molecules like sharks. Their name - oxygen free radicals - makes them sound like political terrorists or 60's yuppies. They mean nothing to most people.

Yet they possess the power to sustain life by fueling the body's chemical reactions and to destroy it when they get out of hand.

We couldn't exist without free radicals, the by-product of burning oxygen in our cells. But growing evidence suggests that free radicals make us age, hurt and die.

Research also is beginning to show how the damage can be prevented and the effects of aging reversed.

Scientists are making strong connections between free-radical damage and such killers as heart disease and cancer. Free radicals also have been implicated in brain damage, arthritis, cataracts and emphysema. As if that weren't enough, they give us hangovers and dandruff... The list seems to be growing by the day.

"Free radical damage is a very important part of the aging process — much more important than scientists were willing to accept in the past," said Dr. Earl Stadtman, chief of the laboratory of biochemistry of the National Heart, Lung and Blood Institute in Bethesda.

Now the free radical theory of aging is the hottest in biogerontology, the science that studies why we age, in hopes of retarding or even reversing the process.

Recent discoveries suggest we can minimize free-radical damage with dietary and other supplements of key nutrients to bolster the body's defenses as they weaken with age.

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The march of the free radical theory to center stage delights its long-suffering founder, Dr. Denham Harman. He came up with his grand notion in 1954 and has been fighting the establishment view, which failed to give his theory any credence until recently.

"Chances are 99 percent [free radicals] are the basis for aging," said the 75-year-old University of Nebraska emeritus professor of medicine and biochemistry. "Aging is the ever-increasing accumulation of changes caused or contributed to by free radicals."

Stadtman agrees. His research at the National Institutes of Health shows that when aged cells reach the end of their lives, 30 percent of their proteins are junk, irreparably fractured by free radicals.

"What the human life span reflects is simply the level of [free radicals] oxidative damage that accumulates in cells," Stadtman has decided. "when enough damage has accumulated, the body's cells can't survive

properly and they just give up."

As you read these words, free radicals are being mass-produced in your body, but most will be squelched by one of nature's most ferocious protective systems. Called antioxidants, this army of enzymes degrades, neutralizes and detoxifies free radicals. Plants, fortunately for the humans who eat them, produce antioxidants..."Most of your cells get repaired," Harman said.

But enough free radicals survive our protective system and sabotage the proteins that make up cells at such an astounding rate that the body must replace each of its billions of proteins every three days on average.

Proteins, composed of and orchestrated by genes, are the notes in the body's basic symphony. All our parts, inside and out — our brain, flesh, blood, nails, lymph, hair — are all made of proteins.

Working in counterpoint, proteins also make up the operating systems that keep us humming: our muscles and nerves; the enzymes that whisk about harmoniously making important things happen; our choruses of immune cells and antibodies; our hormones and neurotransmitters that carry chemical tunes from cell to cell, tissue to tissue, organ to organ.

But the proteins' fuel, oxygen, can be very toxic. Humans can tolerate breathing pure oxygen for no more than 48 hours before sustaining lung damage that can kill them. We survive the air we breathe only because it is just 20 percent oxygen.

As iron oxide, oxygen rusts metal. Oxygen free radicals make butter turn bad; they eat away the toughest granite of the Canadian Rockies; they love to catch fire.

Nature intended us to breathe oxygen only about 30 years, biogerontologists estimate. That's sufficient time for us to mature, breed and pass on our genes according to nature's plan.

If scientists like Harman and Stadtman are right, the longer we breathe, the more we age because of oxygen. What would happen if the tired genes that protect us against oxidations damage could be rejuvenated, or if the proteins they generate could be synthesized in the laboratory and taken as

drugs? Could the aging process be forestalled?

The answers are being sought by biogerontologists around the world as they test the free radical theory.

Free radicals result when molecules are torn apart and thrown out of electrical balance. In chemical terms, a free radical is a molecule with an unpaired electron; simply put, it is a confused particle that has lost its mate. Electrons - electrically charged parti-

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cles that whirl about all atoms and molecules - ordinarily orbit in pairs. But when an oxygen atom is being broken down by the body as it produces energy, the reaction strips an electron away. That leaves an unpaired electron - a 'Free Radical'. The impaired molecule desperately looks for another electron to mate with and make itself whole again. The only way it can is by stealing an electron from somewhere else, thereby throwing another molecule out of balance.

"These things keep going down the line, causing a chain reaction," Harman said. "Eventually two free radicals came together and form a stable molecule." But before that happens, countless electrons crash about in search of mates, wreaking molecular havoc.

Plants, fortunately for the humans who eat them, produce antioxidants

In their frenzy, Harman said, free radicals explode the fragile equilibrium of cells. They shatter the intricate process in which the messages of genes are transcribed into proteins. They demolish enzymes and other molecules.

Free radicals are especially devastating to the tiny energy plants in each cell called mitochondria, where the radicals are produced in abundance. When our body burns food for energy, it ships oxygen molecules through the mitochondria, which use them to furnish the electrical energy that cells need. Thus mitochondria are at the front lines of the free radical attack, said Emory University researcher Douglas Wallace, who has pioneered research into mitochondrial damage.

One result of mitochondrial damage is an eye disease called ocular myopathy. Wallace has been using antioxidants to help treat ocular myopathy patients, who often have droopy eyelids and can't move their eyeballs because the muscles that control movement have become de-energized.

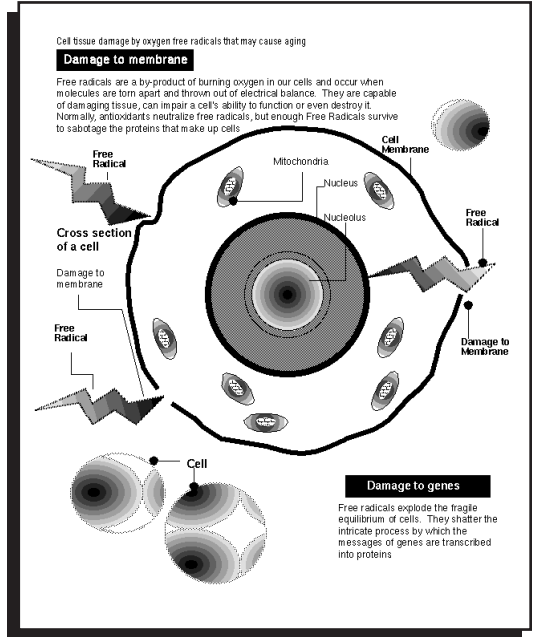
Not everything that free radicals do is unhealthy. When our immune system eats toxic organisms, like viruses, it destroys them with a burst of free radicals. But if this important defense mechanism is missing because of a genetic defect, a child's white blood cells can't form free radicals, and hence are ineffective, leaving the youngster open to death from infections.

Other radicals make their presence known from birth on. "One of life's most beautiful and dramatic moments occurs when a newborn infant takes its first breath," said Dr. Howard Halperin of the University of Chicago. The baby's ability to switch quickly from its mother's circulation and take oxygen into its lungs is generated by a type of free radical.

While some free radicals inside us come from the environment they cause cell dam-

age when we are exposed to radiation such as sunlight, for example — most are produced by our own cellular processes, like sparks leaping from a generator.

Until recently it was impossible to detect radicals because they never stick around long enough to be seen; They live only millionths of a second. But free radical "footprints" may be tracked, and researchers are identifying the wreckage their rampages leave behind.



It's an imprecise list. Free radicals now are being linked to a host of diseases associated with aging, and foremost among them are cancer and heart disease, the major killer of Americans. When free radicals attack genetic material of DNA, they cause gene damage that over time is believed to trigger the eruption of cancer. Dr. Donald Malins, a cancer researcher at the Pacific Northwest Research Foundation in Seattle, reported recently that he has found the first "smoking gun" in the form of free radicals still attached, like spent bullets, to damaged DNA

in human breast cancer cells.

One out of 9 American women will develop breast cancer, and of these, 1 out of 4 will not survive. Until now, the cause was a mystery.

Malins, who reported his findings in the October issue of the journal 'Cancer Research', said a free radical known as hydroxyl inflicts severe damage on the breast's DNA impairing its ability to construct healthy cells.

The hydroxyl radical is kept under tight control in normal tissues, Malins said. "Otherwise, if it elevates, all hell breaks loose and it reacts with proteins." Eventually the DNA becomes so damaged that some cells become metastatic—they spread throughout the body.

Breast cancer, he suggested, is a disease in which the body's defense mechanisms have lost the capacity to control the hydroxyl radical and treatment would involve strategies to restore it.

The hottest antioxidant under investigation, the one that kicked off the current 'Free Radical' hoopla, is Superoxide Dismutase, or SOD, which controls the free radical called 'Superoxide'.

Malins doesn't claim that hydroxyl radicals are the sole cause of cancer. "But we believe the damage to DNA is part of the cause." He said.

Another cancer researcher investigating the effects of free radicals is Dr. Bruce Ames, director of the National Institute of

By reinforcing the body's arsenal against free radicals, scientists hope to extend youthfulness

What causes the loss of control of hydroxyl radicals in breast tissue? "One possibility is that it results from increases in the female hormone, estrogen," Malins said. "And drinking alcohol is, in my view one of the strongest relationships yet proposed."

The National Institutes of Health has shown a correlation between the life span of a dozen mammalian species and their levels of SOD. Their work suggests that the life span of an animal is directly related to the total amount of SOD it can produce.

Environmental Health Sciences Center at the University of California in Berkeley.

According to Ames' latest research, the genes in each human cell receive an estimated 10,000 damaging hits from potentially dangerous oxidants each day. Special enzymes and other proteins routinely race to the accident scenes in the cell, splice out the damage and fix the genes. Our repair rate is 99 to 99.9 percent effective, Ames has shown.

"Normally we're OK." He said. "But it doesn't quite keep up. As you get older, more and more of this junk accumulates, Sunshine, for instance, is a carcinogen, and 99 percent of its damage is repaired. But if you get too much sunshine, you get skin cancer and melanoma."

His research shows that the life spans of animals are directly related to their ability to repair free radical damage. Humans come equipped with far more of the needed

oxidants than any other animals, said Ames, whose latest work shows that vitamin C may help prevent genetic defects.

Recent research into heart disease has found that free radicals give us high blood pressure and make our blood clot abnormally. When they ricochet through the lining of arteries, they trigger the chain of events that permit clumps of cholesterol to build up, block the arteries and cause heart attacks.

A few months ago Dr. Daniel Steinberg of the University of California in San Diego showed the reason so-called "good cholesterol"—HDL—benefits the body is because it acts as an antioxidant to prevent free radicals from damaging arterial walls and producing the deadly buildup of cholesterol-filled plaques.

Steinberg said there's "a reasonable amount of evidence" that intervention with supplements of antioxidants may help prevent arterial disease.

ment to accumulate. It is seen on the body as liver spots, though they are harmless and cannot be used to assess aging due to free radical damage.

In their brief life spans, free radicals also create waste products like aldehydes, chemicals that jam together—or "cross-link"—proteins and other cellular material, much like the tanning of hides. When that happens to collagen, the connective tissue of our bones and muscles, it becomes rigid and doesn't work right—we age. Or so the theory goes.

"The theory has intuitive appeal," noted Roy Walford of the University of California in Los Angeles, one of the leading scientists in aging research. "Most people as they grow older feel more and more like they were being cross-linked."

Harman said his free radical theory evolved because he was looking for something pervasive in all living organisms, something that is essential to life and also makes

Antioxidant Supplementation may be key to longevity

The evidence includes a World Health Organization study of men in 16 European cities. Those with low levels of [antioxidants] in their blood were more likely to suffer fatal heart attacks. The link was stronger than for any other factor studies, including cholesterol levels, and scientists recommended vitamin supplements along with low cholesterol diets.

Hoping to develop therapies to head off diseases caused by free radicals, The National Institutes of Health is launching an unprecedented five-year study involving more than 40,000 women... The \$17 million study, scheduled to begin next year, will observe the role of vitamin E and another antioxidant, beta carotene, in preventing cancer and heart disease.

Beside being linked to those two diseases, free radicals demolish brain cells and contribute to premature senility and other maladies by allowing sludgy yellow age pig-

ment to accumulate, "I think I found it," he said.

He stalked up and down the Berkeley hills buttonholing other scientists, trying to get them to listen. "They wouldn't," he recalled dryly. "It sounded like too simple an answer to something as complex as aging."

Since then by feeding antioxidants to lab animals over the years, Harman showed he could increase their average life expectancy, though not their maximum life span.

Many gerontologists still aren't convinced that boosting protection against free radicals by dietary supplements makes people live healthier longer. But Harman has been patient, laboring to find antioxidants that might work better in animals and support his theory.

It is the molecular biologists—the gene splicer—who are taking Harman seriously now.

Using their powerful new tools, they're measuring the cumulative effects of free radicals in cells and developing ways to counter them.

For example, as researchers identify more of the body's natural antioxidants, genetic engineering techniques often can be used in the laboratory to produce the chemicals in large quantities. These synthetic antioxidants then can be used as drugs when the body's natural supply begins to dwindle with age.

The cell's arsenal against free radicals includes enzymes and other proteins bearing such formidable names as Superoxide Dismutase, Catalase, Glutathione Peroxidase— and the more familiar vitamins A, C and E.

But, the hottest antioxidant under investigation, the one that kicked off the current free radical hoopla, is Superoxide Dismutase, or SOD, which controls the radical called superoxide.

Both superoxide and SOD were discovered by Duke University biochemist Irwin Fridovich and colleagues. Superoxide, found in 1968 provided the first evidence that the body made free radicals. Similarly, discovery of SOD showed that the body produced its own antioxidant against onslaughts by the dangerous free radical.

"It has now been well established that superoxide radicals, can kill," Fridovich said. "They damage enzymes that synthesize amino acids and metabolize sugar."

Dr. Richard Cutler, at the Gerontology Research Center of the National Institute of Health in Baltimore, has shown a correlation between the life span of a dozen mammalian species and their levels of SOD, which protects their genes. His work suggest that the life span of an animal is directly related to the total amount of SOD it can produce to manage the by-products of its own metabolism.

Scientist's long have been testing the possible protection offered by SOD against free-radical damage in animals, and a preparation of bovine SOD has been used as an anti-inflammatory agent in veterinary practice. SOD also reportedly may help fight rheumatoid arthritis and Duchenne muscu-

lar dystrophy.

Human SOD now is being produced by gene-splicing techniques, and trials are under way to determine whether it can help in kidney transplants and play a role in preventing lung damage in premature infants and in preventing heart attacks.

Other developments involve attaching chemicals to make SOD last longer in the body, and putting it in tiny globules of artificial fat to permit it to reach sites inside the cells.

Scientists say that the [bovine source] SOD that is available in health food stores is useless when taken orally. "It merely gets

The cell's arsenal against free radicals includes enzymes and other proteins bearing such formidable names as Superoxide Dismutase (SOD), Catalase, (CAT) and Glutathione.

digested like any other protein." Fridovich said. *[However; supplements that increase endogenous levels of human SOD are being used with remarkable success.]*

Despite the wealth of circumstantial laboratory evidence, the link between free radicals and aging has yet to be proved. What is needed is a way to assess biological damage uniquely associated with free radicals that can be shown to increase over time.

Particularly promising is a simple urine test, being developed by Ames and colleagues, that would show how fast people are aging. The test measures a chemical that indicates the rate at which genes are being damaged by free radicals and repaired.

The most dramatic evidence so far linking free radicals to aging has been found in aged gerbils that recovered their ability to remember after they were treated for two weeks with an industrial antioxidant call PBN.

Robert Floyd, a molecular toxicologist at the Oklahoma Medical Research Foundation, and John Garney of the University of Kentucky showed that PBN improved the animals' brain-cell chemistry.

By inhibiting the runaway free radical process, the antioxidant was able to increase the level of critical brain chemicals the cells need to communicate with one another and to clean out protein damaged by free radicals.

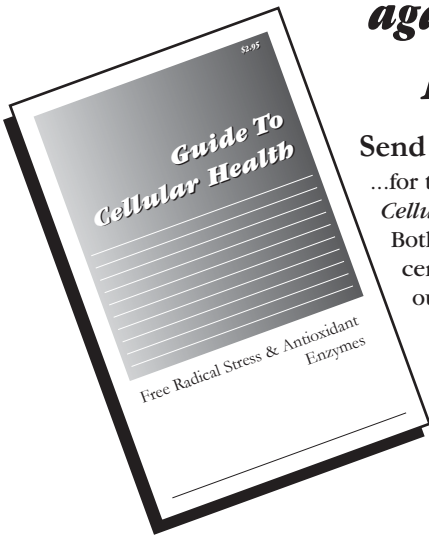
When placed in a special maze, the PBN-treated gerbils [which previously made 2.5 more mistakes than young gerbils] now performed as well as young-

sters. PBN given to young gerbils had no effect on their memories. But when the antioxidant treatment was stopped in the old animals, their earlier forgetfulness returned.

"The experiment," explained the NIH's Stadtman, "marked the first time that a physiological function—the return of youthful brain chemistry and the restoration of short-term memory—has been cleanly linked to the level of oxidized protein in the cell."

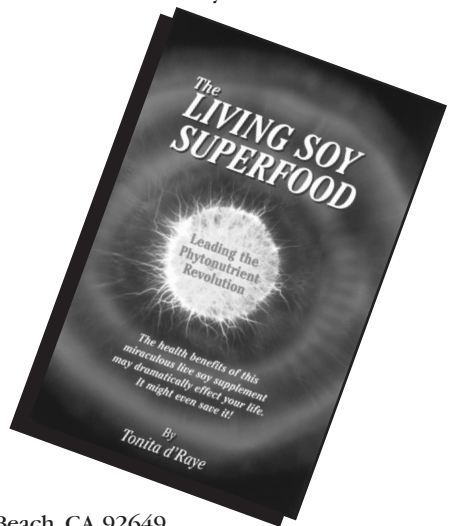
And the potential for humanity may be profound: "The loss of memory with age can apparently be overcome," Stadtman said.

Antioxidants are Winning the battle against the dangerous FREE RADICAL!



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