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Evidence Based Literature to Protect and Explore Natural Medicine since 1996

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2016 Issue on Homeopathy
Hormesis is Homeopathy—Poison is Medicine

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Open Letter to Homeopaths:

Homeopathy has had a bad reputation lately due to a lack of respect for science and statistics. Math-Phobia has put Homeopathy into trouble. This journal is dedicated to help point out Homeopathy’s Myth and Mis-Conceptions. And we hope to point the way forward to help the art of Medicine to regain it former glory. Please let’s work as adults to help our art of Homeopathy face it critics and present a valid evidence based way into the future.

Brad Victor Johnson
Hormesis is Homeopathy - Poison is Medicine

Poison as Medicine

Contents

Dilute Poisons can be medicine This proves Homeopathy ............................................................................................................................................................................. 4
Snake venoms in science and clinical medicine 2. Applied immunology in snake venom research .................................................................................................................................................................... 17
Abstract ................................................................................................................................................................. 17
Hormesis is Homeopathy ............................................................................................................................................. 17
History .................................................................................................................................................................. 18
Examples ................................................................................................................................................................. 19
Physical exercise .................................................................................................................................................... 19
Alcohol ................................................................................................................................................................. 20
Methylmercury and mallard eggs .......................................................................................................................... 20
Effects in aging ..................................................................................................................................................... 20
Mitochondria .......................................................................................................................................................... 20
Ionizing Radiation ................................................................................................................................................ 21
Chemical and Ionizing Radiation combined ...................................................................................................... 21
Controversy .......................................................................................................................................................... 22
Policy consequences .............................................................................................................................................. 22
See also ............................................................................................................................................................... 23
References ............................................................................................................................................................ 23

A dilute poison will produce a reverse effect

This proves Homeopathy
Homeopathy Works and it has a basis in Traditional Pharmacology

The Pharmacology art of Hormesis proves Homeopathy’s use of toxic substances for reversal of action, known as Isode Homeopathy. This also proves Nosode Homeopathy (using diseased or disease causing tissue or organism).

Nosode Homeopathy is the same as Vaccination.

----Let’s Repeat----

Vaccination is an example of Homeopathy Principles.

Herbal Pharmacology and Pharmacology’s Law of Initial Values validates using Dilute herbs in most of Classical Homeopathy.

This also proves the use of dilute Hormones from endocrine organs, known as Sarcode Homeopathy.

Allergy Desensitization using dilute substances proves Allersode Homeopathy.

Reverse Psychology, Intention Medicine, Imponderables, and Emotive Homeopathy all are other forms with scientific explanations of safety and efficacy.

THERE IS MASSIVE EVIDENCE FOR HOMEOPATHY
Hormesis is Homeopathy - Poison is Medicine

The Effects a Poison has in a large dose are Reversed if we use a very dilute form of that Poison

Graphical representation of the Arndt-Schulz Law

Periodic succussion increase the effect

Arndt-Schulz law for an open ulcer: Doses between 0.01 and 10 J/cm² are stimulating the wound healing but higher doses are inhibitory.

Applies to Radiation as well

A dilute poison will produce a reverse effect
“I moved to California to die.” Said Ellie Lobel, 27 when she was bitten by a tick and contracted Lyme disease. And she was not yet 45 when she decided to give up fighting for survival. Caused by corkscrew-shaped bacteria called Borrelia burgdorferi, which enter the body through the bite of a tick, Lyme disease is diagnosed in around 300,000 people every year in the United States. It kills almost none of these people, and is by and large curable – if caught in time. If doctors correctly identify the cause of the illness early on, antibiotics can wipe out the bacteria quickly before they spread through the heart, joints and nervous system.

But back in the spring of 1996, Ellie didn’t know to look for the characteristic bull’s-eye rash when she was bitten – she thought it was just a weird spider bite. Then came three months with flu-like symptoms and horrible pains that moved around the body. Ellie was a fit, active woman with three kids, but her body did not know how to handle this new invader. She was incapacitated. “It was all I could do to get my head up off the pillow,” Ellie remembers.

Her first doctor told her it was just a virus, and it would run its course. So did the next. As time wore on, Ellie went to doctor after doctor, each giving her a different diagnosis. Multiple sclerosis. Lupus. Rheumatoid arthritis. Fibromyalgia. None of them realized she was infected with Borrelia until more than a year after she contracted the disease – and by then, it was far too late. Lyme bacteria are exceptionally good at adapting, with some evidence that they may be capable of dodging both the immune system and the arsenal of antibiotics currently available. Borrelia are able to live all over the body, including the brain, leading to neurological symptoms. And even with antibiotic treatment, 10–20 percent
of patients don’t get better right away. There are testimonies of symptoms persisting –
sometimes even resurfacing decades after the initial infection – though the exact cause of
such post-treatment Lyme disease syndrome is a topic of debate among Lyme scientists.

“I just kept doing this treatment and that treatment,” says Ellie. Her condition was
constantly worsening. She describes being stuck in bed or a wheelchair, not being able to
think clearly, feeling like she’d lost her short-term memory and not feeling “smart”
anymore. Ellie kept fighting, with every antibiotic, every pharmaceutical, every holistic
treatment she could find. “With some things I would get better for a little while, and then I
would just relapse right back into this horrible Lyme nightmare. And with every relapse it
got worse.”

After fifteen years, she gave up. “Nothing was working any more, and nobody had any
answers for me,” she says. “Doctors couldn’t help me. I was spending all this cash and was
going broke, and when I got my last test results back and all my counts were just horrible, I
knew right then and there that this was the end.”

“I had outlived so many other people already,” she says, having lost friends from Lyme
support groups, including some who just couldn’t take the suffering any more. “I didn’t care
if I was going to see my next birthday. It’s just enough. I was ready to call it a life and be
done with it.”

So she packed up everything and moved to California to die. And she almost did. Less than a
week after moving, Ellie was attacked by a swarm of Africanized bees.

**Hormesis is Homeopathy- Poison is Medicine**

**Lethal Swarm**

Ellie was in California for three days before her attack. “I wanted to get some fresh air and
feel the sun on my face and hear the birds sing. I knew that I was going to die in the next
three months or four months. Just laying there in bed all crumpled up... It was kind of depressing.”

At this point, Ellie was struggling to stand on her own. She had a caregiver on hand to help her shuffle along the rural roads by her place in Wildomar, the place where she had chosen to die.

She was just standing near a broken wall and a tree when the first bee appeared, she remembers, “just hitting me in the head”. “All of a sudden – boom! – bees everywhere.”

Her caregiver ran. But Ellie couldn’t run – she couldn’t even walk. “They were in my hair, in my head, all I heard was this crazy buzzing in my ears. I thought: wow, this is it. I’m just going to die right here.”

Ellie, like 1–7 percent of the world’s population, is severely allergic to bees. When she was two, a sting put her into anaphylaxis, a severe reaction of the body’s immune system that can include swelling, nausea and narrowing of the airways. She nearly died. She stopped breathing and had to be revived by defibrillation. Her mother drilled a fear of bees into her to ensure she never ended up in the same dire situation again. So when the bees descended, Ellie was sure that this was the end, a few months earlier than expected.

Bees – and some other species in the order Hymenoptera, such as ants and wasps – are armed with a potent sting that many of us are all too aware of. This is their venom, and it’s a mixture of many compounds. Perhaps the most important is a tiny 26-amino-acid peptide called melittin, which constitutes more than half of the venom of honey bees and is found in a number of other bees and wasps. This little compound is responsible for the burning pain associated with bee stings. It tricks our bodies into thinking that they are quite literally on fire.

When we experience high temperatures, our cells release inflammatory compounds that activate a special kind of channel, TRPV1, in sensory neurons. This ultimately causes the neurons to send a signal to the brain that we’re burning. Melittin subversively makes TRPV1 channels open by activating other enzymes that act just like those inflammatory compounds.

Jellyfish and other creatures also possess TRPV1-activating compounds in their venoms. The endpoint is the same: intense, burning pain.
“I could feel the first five or ten or fifteen but after that... All you hear is this overwhelming buzzing, and you feel them hitting your head, hitting your face, hitting your neck,” says Ellie. “I just went limp. I put my hands up and covered my face because I didn’t want them stinging me in the eyes... The next thing I know, the bees are gone.”

When the bees finally dissipated, her caregiver tried to take her to the hospital, but Ellie refused to go. “This is God’s way of putting me out of my misery even sooner,” she told him. “I’m just going to accept this.” “I locked myself in my room and told him to come collect the body tomorrow.”

But Ellie didn’t die. Not that day, and not three to four months later. “I just can’t believe that was three years ago, and I just can’t believe where I am now,” she tells me. “I had all my blood work done. Everything. We tested everything. I’m so healthy.” She believes the bees, and their venom, saved her life.

Left, a witch placing a scorpion into a pot in order to make a potion. Right, two Shiva devotees known for extracting the poison from a scorpion's bite.
Toxin as Medicine

The idea that the same venom toxins that cause harm may also be used to heal is not new. Bee venom has been used as a treatment in East Asia since at least the second century BCE. In Chinese traditional medicine, scorpion venom is recognized as a powerful medicine, used to treat everything from eczema to epilepsy. Mithradates VI of Pontus, a formidable enemy of Rome (and also an infamous toxinologist), was said to have been saved from a potentially fatal wound on the battlefield by using steppe viper venom to stop the bleeding.

“Over millions of years, these little chemical engineers have developed a diversity of molecules that target different parts of our nervous system,” says Ken Winkel, Director of the Australian Venom Research Unit at the University of Melbourne. “This idea of applying these potent nerve toxins to somehow interrupt a nervous disease has been there for a long time. But we haven’t known enough to safely and effectively do that.”

Despite the wealth of history, the practical application of venoms in modern therapeutics has been minimal. That is, until the past ten years or so, according to Glenn King at the University of Queensland in Brisbane, Australia. In 1997, when Ellie was bouncing around from doctor to doctor, King was teasing apart the components of the venom from the Australian funnel-web, a deadly spider. He’s now at the forefront of venom drug discovery.

King’s group was the first to put funnel-web venom through a separation method called high-performance liquid chromatography (HPLC), which can separate out different components in a mixture based on properties like size or charge. “I was just blown away,” he says. “This is an absolute pharmacological goldmine that nobody’s really looked at. Clearly hundreds and hundreds of different peptides.”

Over the course of the 20th century, suggested venom treatments for a range of diseases have appeared in scientific and medical literature. Venoms have been shown to fight cancer, kill bacteria, and even serve as potent painkillers – though many have only gone as far as animal tests. At the time of writing, just six had been approved by the US Food and Drug Administration for medical use (one other – Baltrodibin, adapted from the venom of the Lancehead snake – is not FDA approved, but is available outside the US for treatment of bleeding during operations).

The more we learn about the venoms that cause such awful damage, the more we realize, medically speaking, how useful they can be. Like the melittin in bee venom.
Melittin does not only cause pain. In the right doses, it punches holes in cells’ protective membranes, causing the cells to explode. At low doses, melittin associates with the membranes, activating lipid-cutting enzymes that mimic the inflammation caused by heat. But at higher concentrations, and under the right conditions, melittin molecules group together into rings creating large pores in membranes, weakening a cell’s protective barrier and causing the entire cell to swell and pop like a balloon.

Because of this, melittin is a potent antimicrobial, fighting off a variety of bacteria and fungi with ease. And scientists are hoping to capitalize on this action to fight diseases like HIV, cancer, arthritis and multiple sclerosis.

For example, researchers at the Washington University School of Medicine in St Louis, Missouri, have found that melittin can tear open HIV’s protective cell membrane without harming human cells. This envelope-busting method also stops the virus from having a chance to evolve resistance. “We are attacking an inherent physical property of HIV,” Joshua L Hood, the lead author of the study, said in a press statement. “Theoretically, there isn’t any way for the virus to adapt to that. The virus has to have a protective coat.” Initially envisioned as a prophylactic vaginal gel, the hope is that melittin-loaded nanoparticles could someday be injected into the bloodstream, clearing the infection.

**Starting Sting Therapy**

Ellie is the first to admit that her tale sounds a little tall. “If someone were to have come to me and say, ‘Hey, I’ll sting you with some bees, and you’ll get better’, I would have said, ‘Absolutely not! You’re crazy in your head!’” But she has no doubts now.

After the attack, Ellie watched the clock, waiting for anaphylaxis to set in, but it didn’t. Instead, three hours later, her body was racked with pains. A scientist by education before Lyme took its toll, Ellie thinks that these weren’t a part of an allergic response, but instead indicated a Jarisch–Herxheimer reaction – her body was being flooded with toxins from dying bacteria. The same kind of thing can happen when a person is cured from a bad case
of syphilis. A theory is that certain bacterial species go down swinging, releasing nasty compounds that cause fever, rash and other symptoms.

For three days, she was in pain. Then, she wasn’t.

“I had been living in this... I call it a brown-out because it’s like you’re walking around in a half-coma all the time with the inflammation of your brain from the Lyme. My brain just came right out of that fog. I thought: I can actually think clearly for the first time in years.”

With a now-clear head, Ellie started wondering what had happened. So she did what anyone else would do: Google it. Disappointingly, her searches turned up very little. But she did find one small 1997 study by scientists at the Rocky Mountain Laboratories in Montana, who’d found that melittin killed *Borrelia*. Exposing cell cultures to purified melittin, they reported that the compound completely inhibited *Borrelia* growth. When they looked more closely, they saw that shortly after melittin was added, the bacteria were effectively paralyzed, unable to move as their outer membranes were under attack. Soon after, those membranes began to fall apart, killing the bacteria.

Convinced by her experience and the limited research she found, Ellie decided to try apitherapy, the therapeutic use of materials derived from bees.

Her bees live in a “bee condo” in her apartment. She doesn’t raise them herself; instead, she mail orders, receiving a package once a week. To perform the apitherapy, she uses tweezers to grab a bee and press it gently where she wants to be stung. “Sometimes I have to tap them on the tush a little bit,” she says, “but they’re usually pretty willing to sting you.”

She started on a regimen of ten stings a day, three days a week: Monday, Wednesday, Friday. Three years and several thousand stings later, Ellie seems to have recovered miraculously. Slowly, she has reduced the number of stings and their frequency – just three stings in the past eight months, she tells me (and one of those she tried in response to swelling from a broken bone, rather than Lyme-related symptoms). She keeps the bees around just in case, but for the past year before I talked to her, she’d mostly done just fine without them.
Unraveling the Secret of Venoms

Modern science has slowly begun to take apart venoms piece by piece to understand how they do the things they do, both terrible and tremendous. We now know that most venoms are complex cocktails of compounds, with dozens to hundreds of different proteins, peptides and other molecules to be found in every one. The cocktails vary between species and can even vary within them, by age, location or diet. Each compound has a different task that allows the venom to work with maximum efficiency – many parts moving together to immobilise, induce pain, or do whatever it is that the animal needs its venom for.

The fact that venoms are mixtures of specifically targeted toxins rather than single toxins is exactly what makes them such rich sources of potential drugs – that’s all a drug is, really, a compound that has a desired effect on our bodies. The more specific the drug’s action, the better, as that means fewer side-effects.

“It was in the 2000s that people started saying well, actually, [venoms] are really complex molecular libraries, and we should start screening them against specific therapeutic targets as a source of drugs,” says King.

Of the seven venom-derived pharmaceuticals on the international market, the most successful, captopril, was derived from a peptide found in the venom of the Brazilian viper (Bothrops jararaca). This venom has been known for centuries for its potent blood-thinning ability – one tribe are said to have coated their arrow tips in it to inflict maximum damage – and the drug has made its parent company more than a billion dollars and become a common treatment for hypertension.
Bryan Fry, a colleague of Glenn King’s at the University of Queensland and one of the world’s most prolific venom researchers, says the captopril family and its derivatives still command a market worth billions of dollars a year. Not bad for something developed in 1970s. “It’s not only been one of the top twenty drugs of all time,” he says, “it’s been one of the most persistent outside of maybe aspirin.”

And it’s not just captopril. Fry points to exenatide, a molecule found in the venom of a lizard, the gila monster, and the newest venom-derived pharmaceutical on the US market. Known by the brand name Byetta, this has the potential to treat type 2 diabetes, stimulating the body to release insulin and slow the overproduction of sugar, helping reverse the hormonal changes caused by the disease.

Rare cases like Ellie’s are a reminder of the potent potential of venoms. But turning folk knowledge into pharmaceuticals can be a long and arduous process. “It could take as long as ten years from the time you find it and patent it,” says King. “And for every one that you get through, ten fail.”

A New Era of Drug Discovery

Since the 1997 study, no one had looked further into bee venom as a potential cure for Lyme disease, until Ellie.

Ellie now runs a business selling bee-derived beauty products called BeeVinity, inspired after, she says, noticing how good her skin looked as she underwent apitherapy. “I thought, ‘Well, people aren’t going to want to get stung with bees just to look good.’”

Ellie has partnered with a bee farm that uses a special electrified glass plate to extract venom. As the bees walk across the plate on the way to and from their hive, harmless currents stimulate the bees to release venom from their abdomens, leaving teeny little droplets on the glass, which are later collected. Ellie says it takes 10,000 bees crossing that plate to get 1 gram of venom (other sources, such as the Food and Agriculture Organization of the UN, quote 1 million stings per gram of venom), but “those bees are not harmed.”
For her, it is more than just a way to make a living: it’s “an amazing blessing”. Proceeds from her creams and other products support bee preservation initiatives, as well as Lyme disease research. In addition, she sends some of the venom she purchases – which, due to the cost of the no-harm extraction method she uses, she says is “more expensive than gold” – to Eva Sapi, Associate Professor of Biology and Environmental Science at the University of New Haven, who studies Lyme disease.

Sapi’s research into the venom’s effects on Lyme bacteria is ongoing and as yet unpublished, though she told me the results from preliminary work done by one of her students look “very promising”. *Borrelia* bacteria can shift between different forms in the body, which is part of what makes them so hard to kill. Sapi has found that other antibiotics don’t actually kill the bacteria but just push them into another form that is more dormant. As soon as you stop the antibiotics, the *Borrelia* bounce back. Her lab is testing different bee venoms on all forms of the bacteria, and so far, the melittin venom seems effective.

The next step is to test whether melittin alone is responsible, or whether there are other important venom components. “We also want to see, using high-resolution images, what exactly happens when bee venom hits *Borrelia,*” Sapi told me.

She stresses that much more data is needed before any clinical use can be considered. “Before jumping into the human studies, I would like to see some animal studies,” she says. “It’s still a venom.” And they still don’t really know why the venom works for Ellie, not least because the exact cause of post-treatment Lyme disease symptoms remains unknown. “Is it effective for her because it’s killing *Borrelia,* or is it effective because it stimulates the immune system?” asks Sapi. It’s still a mystery.

There’s a long way to go for bee venom and melittin. And it takes a lot of work – and money – to turn a discovery into a safe, working medicine. But labs like King’s are starting to tap the pharmaceutical potential that lies in the full diversity of venomous species. And King, for one, believes that scientists are entering a new era of drug discovery.

In the past, venoms have been investigated because of their known effects on humans. Such investigations required both knowledge of the venom’s clinical effects and large volumes of venom, so until now only large species, like snakes, with easily extracted venoms have been studied in any depth. But that’s changing. Technological advances allow for more efficient venom extraction as well as new ways to study smaller amounts of venom. The preliminary tests for pharmaceuticals can now start with nothing more than a genetic sequence. “We can now genomically look at the toxins in these animals without having to actually even purify the venom,” says King, “and that changes everything.” Ken Winkel thinks venomous animals will be excellent drug resources for devastating neurological diseases, as so many of
their venoms target our nervous system. “We really don’t have great drugs in this area,” he says, “and we have these little factories that have a plethora of compounds...” No one knows exactly how many venomous species there are on this planet. There are venomous jellyfish, venomous snails, venomous insects, even venomous primates. With that, however, comes a race against time of our own making. Species are going extinct every year, and up to a third may go extinct from climate change alone.

“When people ask me what’s the best way to convince people to preserve nature, your weakest argument is to talk about how beautiful and wonderful it is,” says Bryan Fry. Instead, he says, we need to emphasize the untapped potential that these species represent. “It’s a resource, it’s money. So conservation through commercialization is really the only sane approach.” Ellie couldn’t agree more. “We need to do a lot more research on these venoms,” she tells me emphatically, “and really take a look at what’s in nature that’s going to help us.”

This is best homeopathic bible to use today for venoms. 

http://www.downloads.imune.net/medicalbooks/HORMESIS.pdf
http://www.downloads.imune.net/medicalbooks/Scientists%20are%20unlocking%20the%20medical%20potential%20of%20venoms%20with%20Homeopathy.pdf
Snake venoms in science and clinical medicine 2. Applied immunology in snake venom research

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Abstract

Enzyme-linked immunosorbent assay (ELISA) is a very important tool for studying both the epidemiology and clinical effects of snake bite in man. For epidemiology ELISA depends on the development and persistence of specific humoral venom antibody in previous snake bite victims. In the Nigerian savanna 63% of previous bite victims possessed specific venom antibodies against *Echis carinatus* venom; in Ecuador, where there is a 5% annual mortality due to snake bite in a population of Waorani Indians, venom antibodies against a wide range of different venoms were identified in previous bite victims using ELISA. In certain areas it is often not possible, using the symptoms of envenoming, to determine which species of snake has bitten the patient. Field studies using ELISA in Nigeria and Thailand have been successful in establishing the species responsible for envenoming. Current studies are in progress on the development of a rapid immunoassay which should be capable of detecting the biting species within 5–10 min of sampling from the admission patient. This will be useful for the clinician as it will enable the rapid detection of the species responsible for envenoming and, therefore, the use of the correct antivenom. Experimental work on the development of new methods of antivenom production includes immunization of experimental animals with venom/ liposome preparations, the preparation of venom antigens using monoclonal antibodies on affinity columns, and recombinant deoxyribonucleic acid technology. Liposomal immunization requires only a single injection of venom to obtain a rapid, high level and protective immune response. Venom liposomes may also be given orally resulting in a serum immunoglobulin G immune response in experimental animals. Use of such a system may eventually result in immunization of man in areas of high snake bite incidence and mortality.

**Hormesis is Homeopathy**

_Hormesis_ is Homeopathy- Poison is Medicine

Professor of Medicine Desire' Dubounet for IMUNE

A low dose of a chemical agent may trigger from an organism the opposite response to a very high dose.

**Hormesis** is the term for generally favorable biological responses to low exposures to _toxins_ and other stressors. It comes from Greek _hörmēsis_ "rapid motion, eagerness", itself from ancient Greek _horēsein_ "to set in motion, impel, urge on". A pollutant or _toxin_ showing hormesis thus has the opposite effect in small doses as in large doses. A related concept is _Mithridatism_, which refers to
the willful exposure to toxins in an attempt to develop immunity against them. Hormetics is the term proposed for the study and science of hormesis.

In toxicology, hormesis is a dose response phenomenon characterized by a low dose stimulation, high dose inhibition, resulting in either a J-shaped or an inverted U-shaped dose response. Such environmental factors that would seem to produce positive responses have also been termed "eustress".

The hormesis model of dose response is vigorously debated. The notion that hormesis is important for chemical risks regulations is not widely accepted.

The biochemical mechanisms by which hormesis works are not well understood. It is conjectured that low doses of toxins or other stressors might activate the repair mechanisms of the body. The repair process fixes not only the damage caused by the toxin, but also other low-level damage that might have accumulated before without having triggered the repair mechanism.

History

German Homeopathic pharmacologist Hugo Schulz first described such a phenomenon in 1888 following his own observations that the growth of yeast could be stimulated by small doses of poisons. This was coupled with the work of German homeopathic physician Rudolph Arndt, who studied animals given low doses of drugs, eventually giving rise to the Arndt-Schulz rule. Arndt's advocacy of homeopathy contributed to the rule's diminished credibility in the 1920s and 1930s. The term "hormesis" was coined and used for the first time in a scientific paper by C.M. Southam and J. Ehrlich in 1943 in the journal: Phytopathology, volume 33, pp. 517–541. Recently, Edward Calabrese has revived the hormesis theory through his research on peppermint plants. The evidence for and importance of hormesis in physiology and health was advanced by Mark Mattson, who elucidated cellular and molecular mechanisms by which the nervous system responds adaptively to mild bioenergetic stresses such as fasting and exercise. Cells respond to such challenges by increasing their production of neurotrophic factors, DNA repair proteins and antioxidant enzymes. Mattson also proposed that the reason that vegetables, fruits, tea and coffee can improve brain health is that they contain 'noxious' chemicals that are produced by the plants to protect themselves from being eaten by insects and other organisms. Such phytochemicals trigger hormetic responses in brain cells which can improve brain function and may increase the resistance of neurons to injury and age-related neurodegenerative disorders such as Alzheimer’s disease and Parkinson’s disease.
Examples

**Physical exercise**

Individuals with low levels of physical activity are at risk for high levels of oxidative stress, as are individuals engaged in highly intensive exercise programs; however, individuals engaged in moderately intensive, regular exercise experience lower levels of oxidative stress. High levels of oxidative stress have been linked by some with the increased incidence of a variety of diseases.\[^{11}\]

It has been claimed that this relationship, characterized by positive effects at an intermediate dose of the stressor (exercise), is characteristic of hormesis.\[^{13}\] However, it is important to point out that there is evidence that the oxidative stress associated with intensive exercise may have long-term health
benefits. This would imply that oxidative stress, itself, provides an example of hormesis (see section on Mitochondrial hormesis), but physical exercise does not.[12]

Alcohol
Main articles: Alcohol consumption and health, Alcohol and cancer and Alcohol and cardiovascular disease

Alcohol is believed to be hormetic in preventing heart disease and stroke,[13] although the benefits of light drinking may have been exaggerated.[14][15]

In 2012, researchers at UCLA found that tiny amounts (1 mM, or 0.005%) of ethanol doubled the lifespan of Caenorhabditis elegans, a round worm frequently used in biological studies, that were starved of other nutrients. Higher doses of 0.4% provided no longevity benefit.[16] However, worms exposed to 0.005% did not develop normally (their development was arrested). The authors argue that the worms were using ethanol as an alternative energy source in the absence of other nutrition, or had initiated a stress response. They did not test the effect of ethanol on worms fed a normal diet.

Methylmercury and mallard eggs

In 2010, a paper published in the journal Environmental Toxicology & Chemistry showed that low doses of methylmercury, a potent neurotoxic pollutant, improved the hatching rate of mallard eggs.[17] The author of the study, Gary Heinz, who led the study for the U.S. Geological Survey at the Patuxent Wildlife Research Center in Beltsville, Md., stated that other explanations are possible. For instance, it is possible that the flock he studied might have harbored some low, subclinical infection and that mercury, well known to be antimicrobial, might have killed the infection that otherwise hurt reproduction in the untreated birds.[17]

Effects in aging

One of the areas where the concept of hormesis has been explored extensively with respect to its applicability is aging.[18][19] Since the basic survival capacity of any biological system depends on its homeodynamic (homeostatic) ability, biogerontologists proposed that exposing cells and organisms to mild stress should result in the adaptive or hormetic response with various biological benefits. This idea has now gathered a large body of supportive evidence showing that repetitive mild stress exposure has anti-aging effects.[20][21] Exercise is a paradigm for hormesis in this respect.[21] Some of the mild stresses used for such studies on the application of hormesis in aging research and interventions are heat shock, irradiation, prooxidants, hypergravity and food restriction.[20][21][22] Some other natural and synthetic molecules, such as celasterols from medicinal herbs and curcumin from the spice turmeric have also been found to have hormetic beneficial effects.[21] Such compounds which bring about their health beneficial effects by stimulating or by modulating stress response pathways in cells have been termed "hormetins". Hormetic interventions have also been proposed at the clinical level,[22] with a variety of stimuli, challenges and stressful actions, that aim to increase the dynamical complexity of the biological systems in humans.[23]

Mitochondria

Mitochondria are sometimes described as "cellular power plants" because they generate most of the cell's supply of adenosine triphosphate (ATP), a source of chemical energy. Reactive oxygen species (ROS) have been regarded as unwanted by-products of oxidative phosphorylation in mitochondria by the proponents of the free-radical theory of aging promoted by Denham Harman. The free-radical theory suggests that the use of compounds which inactivate ROS, such as antioxidants, would lead to a reduction of oxidative stress and thereby produce an increase in lifespan.

ROS may perform an essential and potentially lifespan-promoting role as redox signaling molecules which transduce signals from the mitochondrial compartment to other compartments of the
Increased formation of ROS within the mitochondria may cause an adaptive reaction which produces increased stress resistance and a long-term reduction of oxidative stress. This kind of reverse effect of the response to ROS stress has been named mitochondrial hormesis or mitohormesis and is hypothesized to be responsible for the respective lifespan-extending and health-promoting capabilities of glucose restriction and physical exercise.\[29\]

Hormesis may also be induced by endogenously produced, potentially toxic agents. For example, mitochondria consume oxygen which generates free radicals (reactive oxygen species) as a by-product. It was previously proposed on a hypothetical basis that such free radicals may induce an endogenous response culminating in increased defense capacity against exogenous radicals (and possibly other toxic compounds).\[27\] Recent experimental evidence strongly suggests that this is indeed the case, and that such induction of endogenous free radical production extends lifespan of a model organism. Most importantly, this extension of life span is prevented by antioxidants, providing direct evidence that toxic radicals may mitohormetically exert life extending and health promoting effects.\[28\] Since mitochondrial activity was found to be increased in the previously mentioned studies, this effect cannot be explained by an excess of free radicals that might mark mitochondria for destruction by lysosomes, with the free radicals acting as a signal within the cell to indicate which mitochondria are ready for destruction, as proposed by Nick Lane.\[29\]

Whether this concept applies to humans remains to be shown, although recent epidemiological findings support the process of mitohormesis, and even suggest that some antioxidant supplements may increase disease prevalence in humans.\[30\]

### Ionizing Radiation

**See also:** Radiation hormesis

Hormesis has been observed in a number of cases in humans and animals exposed to chronic low doses of ionizing radiation. In Taiwan recycled radiocontaminated steel was inadvertently used in the construction of over 100 apartment buildings causing the long-term (10 years) exposure of 10,000 people. The average dose rate was 50 mSv/year and a subset of the population (1,000 people) received a total dose of over 4,000 mSv over ten years. In the widely used Linear No Threshold (LNT) theory used by regulatory bodies, the expected cancer deaths in this population would have been 302 with 70 caused by the extra ionizing radiation with the remainder caused by natural background radiation. However the observed cancer rate was quite low at 7 cancer deaths when 232 would be predicted by the LNT theory. Ionizing radiation hormesis appears to be at work. Described by Professor Charles L. Sanders, Korea Advanced Institute of Science and Technology.\[31\]

Taiwan's National Cancer Registry (first study)

Cancer risks in a population with prolonged low dose-rate gamma-radiation exposure in radiocontaminated buildings, 1983-2002. The results suggest that prolonged low dose-rate radiation exposure appeared to increase risks of developing certain cancers in specific subgroups of this population in Taiwan.\[32\]

Taiwan's National Cancer Registry (second study, two years later)

A significant radiation risk was observed for leukemia excluding chronic lymphocytic leukemia (HR(100mGy) 1.19, 90% CI 1.01-1.31). Breast cancer exhibited a marginally significant dose response (HR(100mGy) 1.12, 90% CI 0.99-1.21). The results further strengthen the association between protracted low-dose radiation and cancer risks, especially for breast cancers and leukemia, in this unique cohort population.\[33\]

### Chemical and Ionizing Radiation combined

No experiment can be performed in perfect isolation. Thick lead shielding around a chemical dose experiment to rule out the effects of ionizing radiation is built and rigorously controlled for in the laboratory, and certainly not the field. Likewise the same applies for ionizing radiation studies because by definition ionization radiation creates a cascade of new chemicals in the body which
combine with whatever other environmental exposure is occurring during the measurement period. The resulting confusion in the low dose exposure field (radiation and chemical) arise from lack of consideration of this concept as described by Mothersill and Seymory. Mothersill and Seymory state "Most of the arguments about whether radiation is ‘good for you’ or ‘bad for you’ fail due to lack of consideration of the hierarchical level at which the effect occurs and because most of the arguments are anthropocentric. For example cell death is seen as a ‘bad’ effect but if it removes a potentially carcinogenic cell from the population of cells in a tissue it could prevent cancer starting and could be seen as ‘good’.”

Controversy

Whether hormesis is common or important is controversial. At least one peer-reviewed article accepts the idea, claiming that over 600 substances show a U-shaped dose-response relationship. Calaberese and Baldwin wrote: "One percent (195 out of 20,285) of the published articles contained 668 dose-response relationships that met the entry criteria.”

The idea that low dose effects may be (sometimes strikingly) different is accepted, but that the low dose effect is positive is questionable. The hypothesis of hormesis has generated the most controversy when applied to ionizing radiation. This theory is called radiation hormesis. For policy making purposes, the commonly accepted model of dose response in radiobiology is the linear no-threshold model (LNT), which assumes a strictly linear dependence between the risk of radiation-induced adverse health effects and radiation dose.

The United States National Research Council (part of the National Academy of Sciences), the National Council on Radiation Protection and Measurements (a body commissioned by the United States Congress) and the United Nations Scientific Committee on the Effects of Ionizing Radiation (UNSCEAR) all agree that radiation hormesis is not clearly shown, nor clearly the rule for radiation doses.

A report commissioned by the French National Academy concluded that there is sufficient evidence for hormesis occurring at low doses and that LNT should be reconsidered as the methodology used to estimate risks from low level sources of radiation, like deep geological repositories for nuclear waste. On the other hand, the United States-based National Council on Radiation Protection and Measurements states that there is insufficient evidence for radiation hormesis and that radiation protection authorities should continue to apply the LNT model for purposes of risk estimation.

Policy consequences

Regulatory agencies such as the Environmental Protection Agency (EPA), the Food and Drug Administration (FDA), and the Nuclear Regulatory Commission (NRC) traditionally use a linear no-threshold model for carcinogens (including radiation). In the linear model, the assumption is that there is no dosage that has no risk of causing cancer. While this linear approach remains the default, with sufficient mechanistic evidence suggesting a non-linear dose-response, EPA allows for the derivation of a threshold dose (also known as reference dose) below which it is assumed that there is no risk for cancer.

While proponents of hormesis argue that changing to a hormesis model would likely change exposure standards for these toxicants in air, water, food and soil, making the standards less strict, other scientists point out that low dose stimulation can have extremely adverse effects. Similarly, low doses of the phthalate DEHP cause increased allergic responses to allergens, while higher doses have no effect. Wider use of the hormesis model would affect how scientists design and conduct studies and the selection of models that estimate risk. In all likelihood, recognizing that low dose effects can’t be predicted from high dose experiments would force a strengthening of public health standards, not their weakening, as hormesis proponents would argue.
Hormesis remains largely unknown to the public. Any policy change ought to consider hormesis first as a public health issue (versus an industrial regulatory issue). This would include the assessment of the public concern regarding exposure to small toxic doses. In addition, impact of hormesis policy change upon the management of industrial risks should be studied.[39]

See also

- Calorie restriction
- Michael Ristow
- Petkau effect
- Radiation hormesis
- Stochastic resonance
- Arndt–Schulz rule
- Mithridatism

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Evidence"

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