

**Mercury and cognitive
dysfunction:
Are fish part of the problem or
part of the solution?**

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**CDID Annual Symposium
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Supplement
Serving Size
Servings Per

Selenium (as L-selenomethionine)

Other ingredients
cellulose, calcium

Does not contain

KEEP OUT OF REACH OF CHILDREN
STORE SEPARATELY FROM OTHER SUPPLEMENTS

Researchers Find Mercury Levels in Tuna Are Stubbornly High

by HIROKO TABUCHI

In the 1960s and 1970s, the horror stories of mercury poisoning in Japan and elsewhere shocked the world into curbing releases of the toxic metal. Since then, mercury levels have fallen from human activities, such as burning coal and mining, has declined in many parts of the world.

When a team of French researchers analyzed thousands of tuna samples from 1971 to 2022, they found that mercury levels in the fish remained virtually unchanged.

That's most likely because "legacy" mercury that has accumulated deep in the ocean is circulating into shallower depths where tuna swim and feed, the researchers posit in a study published this month in the journal *Environmental Science & Technology Letters*. Using modeling, they predicted that even with the most stringent mercury regulations, it would take an additional 10 to 25 years for mercury concentrations to start falling in the ocean. Drops in mercury in tuna would follow only decades after that.

The study shows that mercury levels in tuna would follow only decades after that. The study shows that mercury levels in tuna would follow only decades after that.

Unloading skipjack tuna at a market in Japan last fall. Researchers found little change in mercury levels in tuna in 51 years.

Tabuchi H. *The New York Times*, February 28, 2024

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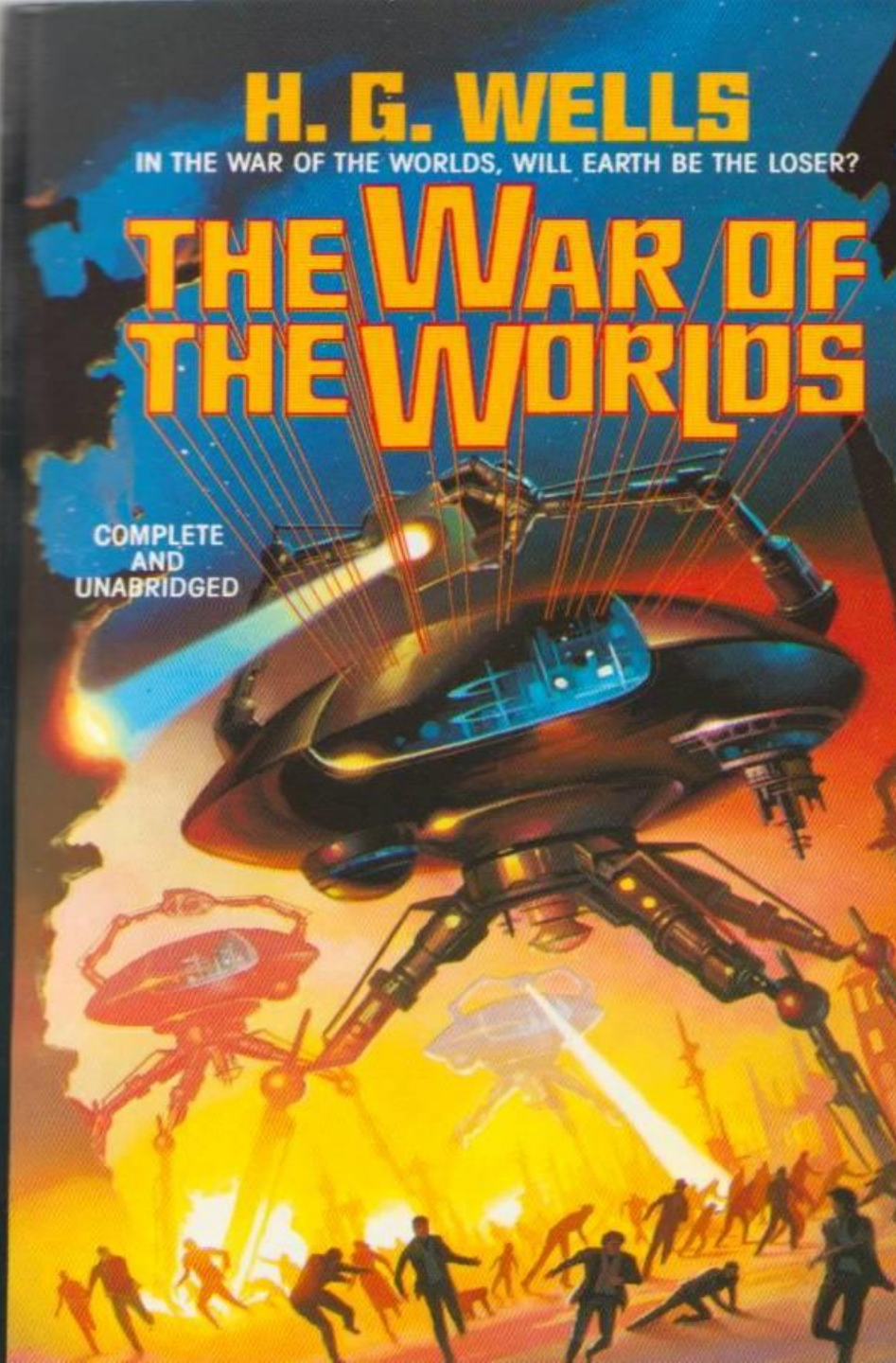
HIROKO MASUIKE/THE NEW YORK TIMES

H. G. WELLS

IN THE WAR OF THE WORLDS, WILL EARTH BE THE LOSER?

THE WAR OF THE WORLDS

COMPLETE
AND
UNABRIDGED



- “...the Martians –*dead!* – slain by the putrefactive and disease bacteria against which their systems were unprepared; slain as the red weed was being slain; slain, after all man’s devices had failed, by the humblest things that God, in his wisdom, has put upon this earth.”
- “These germs of disease have taken toll of humanity since the beginning of things—taken toll of our prehuman ancestors since life began here. But by virtue of this natural selection of our kind we have developed resisting power; to no germs do we succumb without a struggle, and to many—those that cause putrefaction in dead matter, for instance—our frames are altogether immune.”

- “That they did not bury any of their dead, and the reckless slaughter they perpetrated, point also to an entire ignorance of the putrefactive process.”
- “For so it had come about, as indeed I and many men might have foreseen had not terror and disaster blinded our minds.”



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Original Research Article

Fish consumption and omega-3 polyunsaturated fatty acids from diet are positively associated with cognitive function in older adults even in the presence of exposure to lead, cadmium, selenium, and methylmercury: a cross-sectional study using NHANES 2011–2014 data

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ABSTRACT

Background: Long-chain omega-3 polyunsaturated fatty acids (ω -3 PUFAs) are reported to be beneficial for cognition, but limited consumption of some

Sasaki N et al. Fish consumption and omega-3 polyunsaturated fatty acids from diet are positively associated with cognitive function in older adults even in the presence of exposure to lead, cadmium, selenium and methylmercury: a cross-sectional study using NHANES 2011-2014 data, *Amer J Clin Nutr*, Vol. 119, pp. 283-293, 2024.

Introduction

Fish is an important source of long-chain omega-3 PUFAs, including DHA and EPA, which are essential for human health [1–3]. Humans cannot synthesize ω -3 PUFAs and must obtain them from dietary sources [2,4]. However, one study reported the median intake of ω -3 PUFAs in the United States as 0.11 g/d, lower than the recom-

consumption remains the primary source of biologically significant ω -3 PUFA intake, especially of EPA- and DHA-rich species, such as herring, sardines, pollock, flying fish, and salmon [1,2,5].

ω -3 PUFAs are particularly important in the central nervous system and the eyes [2,4]. DHA, the most abundant long-chain ω -3 PUFA in the human body, has been reported to reduce the risk of developing various diseases, including primary or secondary cardiovascular dis-

“Fish is an important source of long-chain omega-3 PUFAs, including DHA and EPA, which are essential for human health.”

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The downside of fish

- **“Fish often contain contaminants that pose a hazard to cognitive function, such as persistent organic pollutants and methylmercury. Persistent organic pollutants concentrate in the fat of fatty fish, such as salmon and trigger advisories for limited consumption for some species.”**

The downside of fish

- **“Large carnivorous fish often contain high concentrations of methylmercury, a potent neurotoxin in both children and adults, causing decrements in remote memory, mental manipulation, and orientation in older adults. Fish also often contain lower concentrations of other toxic metals. Therefore, the overall health effects of fish consumption remain unclear.”**

Study protocol

- **“In this study, we used the same NHANES data to better understand the relationships between fish consumption, dietary omega-3 PUFAs, and heavy metal exposures on cognitive function.”**
- **“Daily intake of dietary fatty acids was estimated using data from 2 dietary interviews conducted on separate days. The first dietary interview was conducted in-person at a mobile examination center, whereas the second interview was by telephone 3-10 d after the initial interview. For this study, we created continuous variables by summing the average values of EPA and DHA.”**

Study protocol

- **“All concentrations of metals in whole blood or urine were measured using certified laboratory methods, quality assurance, and control procedures with inductively coupled plasma-mass spectrometry.”**

Study participants

- **“The participants were grouped by age: 60-65 (36%), 66-74 (36%), and ≥ 75 (28%). The study included approximately equal numbers of male and female participants (49% and 51% respectively), and half of participants received at least some college education (50%). The majority of participants were non-smokers (87%) and either nondrinkers (47%) or moderate to heavy drinkers (50%).”**

Study participants

- **“The median dietary omega-3 PUFA intake was 0.035 g, and 71% of participants consumed at least one fish meal per month, an equal frequency of >0.23 times a week. The median concentrations of blood metals were 14.80 µg/L for lead, 0.41 µg/L for cadmium, 192.40 µg/L for selenium, and 0.87 µg/L for methylmercury.”**

Study results

“The results from linear regression models revealed that consuming 2 or more fish meals per month was associated with significantly higher cognitive function scores for all cognitive tests compared with not consuming any fish.”

Study results

“There were significantly better cognitive scores with increased fish consumption for participants aged 66-74 y as well as for female participants in all age groups.”

Study results

- **“The most striking result from this study is how strongly fish consumption is associated with better performance on immediate recall, delayed recall, and executive function in older adults. Interestingly, results are consistently stronger for reported fish consumption than dietary omega-3 PUFAs for all cognitive scores.”**

Study results

- **“Although the frequency of fish consumption is self-reported, and omega-3 PUFA intake was estimated partly from the reported frequency and species of fish consumption alongside other dietary sources, the benefit of both is apparent. Even when comparing no fish with 2 fish meals per month, cognitive scores increased significantly.”**

Sasaki et al emphatically assert:

“...the present results suggest that, at least for older adults, the benefit of fish consumption may outweigh the hazards of methylmercury and persistent organics.”

Could selenium be the reason for the findings?

- **“Our results are consistent with the conclusion that the positive associations of fish consumption and blood selenium on cognitive function are independent factors. Selenium is known to protect against mercury toxicity, especially selenoenzymes of glutathione peroxidase and thioredoxin reductase, which irreversibly bind to methylmercury and prevent toxicity.”**

Could selenium be the reason for the findings?

- **“Glutathione peroxidase and thioredoxin reductase prevent and reverse oxidative and inflammatory damage in the human body and play important roles in the brain. Molar ratios of selenium to methylmercury have been shown to be more important than methylmercury concentrations, and fish species with higher molar ratio of selenium to methylmercury (≥ 1), are considered safe to eat.”**

Could selenium be the reason for the findings?

- **“Morris et al. found that seafood consumption was correlated with less severe Alzheimer’s disease (mean age 89.9 y) and higher mercury in older adults’ brains, but these mercury levels were not correlated with brain neuropathology.”**

Could selenium be the reason for the findings?

- **“Our results suggest that fish consumption is not the main source of selenium, but selenium concentrations are weakly correlated with selenium-rich fish species such as tuna, mackerel, salmon, and sardines. Nevertheless, participants who had adequate selenium intake and consumed selenium-rich fish could activate selenoenzymes and this may help mitigate the negative associations of methylmercury exposures on cognitive function.”**

Could there be an additive effect of omega-3 fats and selenium?

- **“Our study also demonstrates that fish consumption is an important factor, contributing to the positive associations of omega-3 PUFAs with cognitive function beyond blood selenium concentration. In older adults, decreasing plasma selenium is associated with declining cognitive function.”**

Could there be an additive effect of omega-3 fats and selenium?

- **“Frequent fish consumption is positively associated with plasma selenium, red blood cell fatty acid omega-3 PUFAs, DHA, and EPA. In animal models, the combination of omega-3 PUFAs and selenium was the only treatment condition that significantly increased levels of superoxide dismutase and catalase and neutralized adverse neurotoxic effects of paraquat, a potent and toxic herbicide.”**

Could there be an additive effect of omega-3 fats and selenium?

- **“Therefore, the mixture of omega-3 PUFAs and selenium may activate antioxidant defense systems, alter biochemical parameters for lipid peroxidation and oxidative stress, increase superoxide dismutase and catalase, and protect cognitive function from the adverse effects of neurotoxic compounds.”**

Could there be an additive effect of omega-3 fats and selenium?

- **“Our results from mixture analyses indicate that dietary omega-3 PUFAs or fish consumption, in combination with concentrations of selenium, are critical in maintaining cognitive health in older adults, reducing the negative impacts of lead, cadmium, and methylmercury on cognitive function. Furthermore, our results show that the positive associations of fish consumption and selenium are independent.”**

A caveat from Sasaki et al

- **“...this is a cross-sectional study with unweighted analysis, and although we cannot prove causality, our results provide important insights into the potential benefits of fish consumption and selenium intake on cognitive function.”**

Special Feature: Methylmercury

Forum

Mercury Toxicity and the Mitigating Role of Selenium

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Berry MJ & Ralston NVC. Mercury toxicity and the mitigating role of selenium, *EcoHealth*, Vol. 5, pp. 456-459, 2008

its attendant risks in the environment and food sources, including the studies reported in this issue. However, studies of mercury intoxication have frequently failed to consider the protective effects of the essential trace element, selenium. Mercury binds to selenium with extraordinarily high affinity, and high maternal exposures inhibit selenium-dependent enzyme activities in fetal brains. However, increased maternal dietary selenium intakes preserve these enzyme activities, thereby preventing the pathological effects that would otherwise arise in their absence. Recent evidence indicates that assessments of mercury exposure and tissue levels need to consider selenium intakes and tissue distributions in order to provide meaningful risk evaluations.

Keywords: mercury, selenium, toxicity, environment, heavy metals

Mercury is a naturally occurring element that originates from geological materials, but readily distributes into the air, water, soil, and biomass of the environment. Mercury is a naturally occurring element that originates from geological materials, but readily distributes into the air, water, soil, and biomass of the environment. Mercury is a naturally occurring element that originates from geological materials, but readily distributes into the air, water, soil, and biomass of the environment.

“Mercury is a naturally occurring element that originates from geological materials, but readily distributes into the air, water, soil, and biomass of the environment.”

Hg⁰ eventually becomes charged and precipitates as inorganic mercury (Hg²⁺) deposited on ground and water surfaces. Bacteria convert Hg²⁺ to form methylmercury (MeHg⁺) that is largely found in association with the sulfur

deposition is released back into the environment when coal is burned. As a result, coal-burning power plants are a dominant source of human-caused mercury emissions to the atmosphere.

Coal-fired power plants in the United States account for ~40% of domestic (~5% of global) human-caused mercury emission (Driscoll et al. 2007); the U.S. Environmental Protection Agency (EPA) estimates that ~25% of

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Sources of metallic mercury (Hg^0)

- **“The uncharged metallic mercury form (Hg^0) is highly volatile and enters the atmosphere where it can reside for extended periods. Substantial releases of Hg^0 from natural sources such as solar irradiation, combustion (grass and forest fires), and volcanism cannot be controlled.”**

Getting to methylmercury

- **“Regardless of the source, atmospheric Hg^0 eventually becomes charged and precipitates as inorganic mercury (Hg^{2+}) deposited on ground and water surfaces. Bacteria convert Hg^{2+} to form methylmercury (MeHg^+) that is largely found in association with sulfur and cysteine and similar thiomolecules in the aquatic biomass. As a result, the most common organic mercury compound found in the environment is MeHg-cysteine.”**

Coal as a source

- **“This is also the predominant form accumulated in the aquatic biomass that eventually became coal.”**

We are not defenseless against this onslaught: Selenium to the rescue

- **“Studies have demonstrated the binding of complexes of mercury-selenium, silver-selenium, and cadmium-selenium by plasma selenoprotein P, leading to the proposal that this protein may function to chelate heavy metals, reducing their toxicity.”**

Evidence from research on miners

- **“In miners exposed to high concentrations of mercury, expression of both selenoprotein P protein and glutathione peroxidase activity was increased. These increments were accompanied by elevated selenium concentrations in serum. In addition, selenoprotein P bound more mercury at higher mercury exposure concentrations.”**

Selenium protects many life forms from mercury toxicity

- **“The ability of selenium compounds to decrease the toxicity of mercury has been established in all species of mammals, birds, and fish investigated.”**

Due to selenium:mercury ratios fish are beneficial

- **“Therefore, it is important to consider the molar relationships between mercury and selenium when investigating neurodevelopmental outcomes of maternal mercury exposure during pregnancy. Since free-ranging marine fish are rich sources of selenium in substantial molar excess of mercury, this may explain why the largest and most recent studies of effects of maternal seafood consumption (and associated MeHg⁺ exposures) on child neurodevelopmental outcomes find substantial benefits (~5-10 IQ points) instead of harm.”**

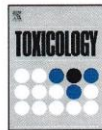
Could mercury adversely affect other selenium functions?

- **“Since mercury is uniquely able to inhibit selenium-dependent enzyme activities in brain tissues, the risks of oxidative brain damage as a result of mercury toxicity directly correspond to Hg:Se molar ratios in tissues. Converging evidence from cell culture studies indicates a progressive decrease in the activity of selenium-dependent glutathione peroxidase enzyme activities in cells exposed to mercury.”**

The real issue for mercury toxicity is mercury:selenium ratios

- **“In animal studies where mercury toxicity has been observed, mercury has consistently been present in substantial molar excess of selenium in the affected tissues. Since the binding affinity between mercury and selenium is a million times greater than the affinity between sulfur and mercury, it is easy to understand why Hg:Se molar ratios in excess of 1:1 stoichiometry are increasingly toxic.”**

Evidence to support the role of selenium in reducing the toxicity of methylmercury



Dietary selenium's protective effects against methylmercury toxicity

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ABSTRACT

Dietary selenium (Se) status is inversely related to vulnerability to methylmercury (MeHg) toxicity. Mercury exposures that are uniformly neurotoxic and lethal among animals fed low dietary Se are far less serious among those with normal Se intakes and are without observable consequences in those fed Se-enriched diets. Although these effects have been known since 1967, they have only lately become well

Ralston NVC & Raymond LJ. Dietary selenium's protective effects against methylmercury toxicity, *Toxicology*, Vol. 278, pp. 112-123, 2010.

populations exposed to foods that contain Hg in molar excess of Se, such as shark or pilot whale meats, have found adverse child outcomes, but studies of populations exposed to MeHg by eating Se-rich ocean fish observe improved child IQs instead of harm. However, since the Se contents of freshwater fish are dependent on local soil Se status, fish with high MeHg from regions with poor Se availability may be cause for concern. Further studies of these relationships are needed to assist regulatory agencies in protecting and improving child health.

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1. Introduction

A nutraceutical is a food known to have a medical or health benefit, including the prevention and treatment of disease. Selenium (Se), an essential trace element for humans and animals, is increasingly recognized for its biological importance and is increasingly considered a nutraceutical component. As reviewed by Rayman (2000), many metabolic processes and, hence, many diseases and clinical conditions directly or indirectly involve disruptions of Se physiology. Selenium can act as a growth factor (Ramauge et al., 1996), has powerful antioxidant (Behne et al., 2000) and anticancer (Combs and Lu, 2001) properties, and is essential for normal thyroid hormone homeostasis (Ramauge et al., 1996) and immunity (Roy et al., 1995). Studies indicate Se has important roles in development, reproduction, cardiovascular disease, and mood disorders (see Rayman, 2000; Taylor et al., 2009, for reviews).

The role of defects of Se physiology in the etiologies of certain diseases is also becoming apparent. Links have been found between compromised Se-dependent metabolic processes and congenital muscular dystrophy, autism, Alzheimer's and Down syndromes,

brain tumors, diabetes, liver diseases, and conditions associated with increased oxidative stress or inflammation, such as rheumatoid arthritis, pancreatitis, asthma, and obesity (Whanger, 2001; Rayman, 2000; Köhrle et al., 2000).

Less known, but equally important, is the fact that Se is a natural methylmercury (MeHg) and inorganic Hg antagonist that potentially counteracts or eliminates symptoms of toxicity that would otherwise accompany high MeHg/Hg exposures. Because Hg's binding affinities for Se (10^{45}) are up to a million times higher than its affinity for sulfur (10^{39}) in analogous forms (Dyrssen and Wedborg, 1991), Se's "protective effect" was initially presumed to involve Se sequestration of Hg, thereby preventing its harmful effects. However, as more has become understood about Se physiology, the mechanism of MeHg/Hg toxicity and the mechanism of Se's protective effect have also become clear. The high affinity between Hg and Se results in Hg binding to Se, thus compromising Se's biological functions and availability. By biochemical definition, MeHg is a highly specific irreversible inhibitor of selenoenzymes. Since MeHg has the ability to cross the placental and blood–brain barriers, its high affinity for Se enables it to specifically sequester Se at the active sites of essential Se-dependent enzymes (selenoenzymes) in fetal neuroendocrine tissues that lack adequate reserves of Se because of their rapid growth. As intracellular concentrations of MeHg approach, and especially as they exceed 1:1

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Epidemiologic evidence

- **“The previously unexplained ‘silent latency,’ or unique delay between exposure to lethal or damaging doses of MeHg (or dimethylmercury) and onset of clinical signs, appears likely to be attributable to the time-dependent, cumulative sequestration of Se in biologically unavailable HgSe forms. The delay between exposure to a lethal dose and onset of clinical symptoms, the affected tissue locations, and characteristic signs of toxicity are all entirely consistent with predictions regarding progressive MeHg inhibition of selenoenzymes and increasing sequestration of Se as HgSe.”**

The role of supplemental selenium: to negate constant MeHg exposure

- **“...the ‘protective effect’ of supplemental Se is readily understood. Since additional Se ensures adequate Se is available to replace the Se lost to Hg sequestration, thereby maintaining normal selenoprotein synthesis, adverse effects that would otherwise have occurred in the absence of these enzyme activities are averted.”**

The relationship between selenium and mercury is vastly underappreciated

“Recognizing the effects of high Hg exposures on Se physiology is vital in understanding Hg toxicity risks, yet it has been seriously overlooked.”

Fish are one of the best sources of dietary selenium

- **“Almost all varieties of ocean fish and seafoods are rich sources of Se. In a survey of 1100 foods (U.S. Department of Agriculture National Nutrient Database, 2008), seafoods comprised 17 of the top 25 dietary sources of Se. Therefore, most ocean fish offer abundant natural protection against the MeHg that they also contain. This has important implications for human health since fish contain many essential nutrients and are often needlessly avoided because of misguided fears associated with their consumption.”**

Few other animal sources of selenium

- **“The levels of Se present in livestock reflect the Se contents of the plants they consume. Unfortunately, low-Se soils are prevalent in many areas of the world, thus compromising the Se status of indigenous populations that exclusively eat locally produced foods.”**

Plant-based sources

- **“Garlic, mushrooms, and certain types of nuts are also good sources, but fruits, vegetables, and dairy products tend to have low-Se contents.”**

Selenium status in human populations

- **“The whole blood Se concentrations observed in various countries around the world are often below the level that is known to be necessary to support ‘optimal’ blood levels of the glutathione peroxidase, a selenoenzyme commonly measured to assess Se status. Because of centralized food distribution, blood Se contents in the United States are generally rich, but tend to be variable because of diversity in food choices.”**

The importance of supplemental selenium in today's world

- **“...Hg’s propensity for Se sequestration in brain and endocrine tissues can inhibit formation of essential Se-dependent proteins (selenoproteins). Therefore, the ‘protective effect’ of supplemental Se can be explained as ensuring adequate levels of Se are available to replace the amount of Se lost to Hg sequestration, thereby maintaining normal selenoprotein levels.”**

Selenium levels in the brain

- **“Under normal conditions, homeostatic regulation prevents the Se contents of brain from falling below levels approximating 60% of normal. However, consumption of diets with high MeHg contents (50 $\mu\text{mol MeHg/kg}$: ~ 10 ppm Hg) cause the brain Se to diminish to 43% of normal brain Se in weanling rats raised on low-Se diets. These remarkably low brain Se contents apparently occurred because MeHg impairs redistribution of somatic Se reserves in other tissues.”**

How much brain MeHg is too much?

- **“For the sake of clarity, high MeHg exposures are defined as those that result in disproportionately high Hg:Se (<5:1) molar ratios.”**
- **“Dietary Hg:Se ratios will need to be significantly lower than 1:1 in order to prevent impaired maternal transport of Se to the fetus and prevent neurodevelopmental outcomes in the offspring.”**

Mercury and selenium from an intracellular standpoint

- **“Intracellular Se is usually present in substantial excess of Hg, and brain cells can, therefore, easily access sufficient free Se to support optimal rates of selenoenzyme synthesis and activity. However, when the intracellular concentration of MeHg in the cells approaches or exceeds that of Se (1-2.5 μM), the amounts of Se available for normal selenoenzyme synthesis diminish.”**

The problem is not too much mercury but too little selenium

“Contrary to earlier expectations, it has been shown that Hg does not cause oxidative damage directly. Instead, high MeHg exposures cause increased oxidative damage secondary to their inhibition of selenoenzyme activities. Selenium’s ability to counteract toxic effects of high Hg exposures has been recognized since 1967, and subsequent studies have confirmed that supplemental Se counteracts the otherwise toxic consequences of Hg exposure.”



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Original Research Article

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Introduction

Fish is an important source of long-chain omega-3 PUFAs, including DHA and EPA, which are essential for human health [1–3]. Humans cannot synthesize ω -3 PUFAs and must obtain them from dietary sources [2,4]. However, one study reported the median intake of ω -3 PUFAs in the United States as 0.11 g/d, lower than the recommended intake of 0.5 g/d [1]. Although green vegetables, nuts, and algae contain α -linolenic acid, a less potent 18-carbon ω -3 PUFA, fish

consumption remains the primary source of biologically significant ω -3 PUFA intake, especially of EPA- and DHA-rich species, such as herring, sardines, pollock, flying fish, and salmon [1,2,5].

ω -3 PUFAs are particularly important in the central nervous system and the eyes [2,4]. DHA, the most abundant long-chain ω -3 PUFA in the human body, has been reported to reduce the risk of developing various diseases, including primary or secondary cardiovascular disease [6–9]. However, recent clinical trials have failed to demonstrate reductions in cardiac arrhythmias [9], adverse cardiovascular events

Abbreviations: CERAD, Consortium to Establish a Registry for Alzheimer's Disease; DAG, directed acyclic graph; DSST, Digit Symbol Substitution Test; NCHS, National Center for Health Statistics.

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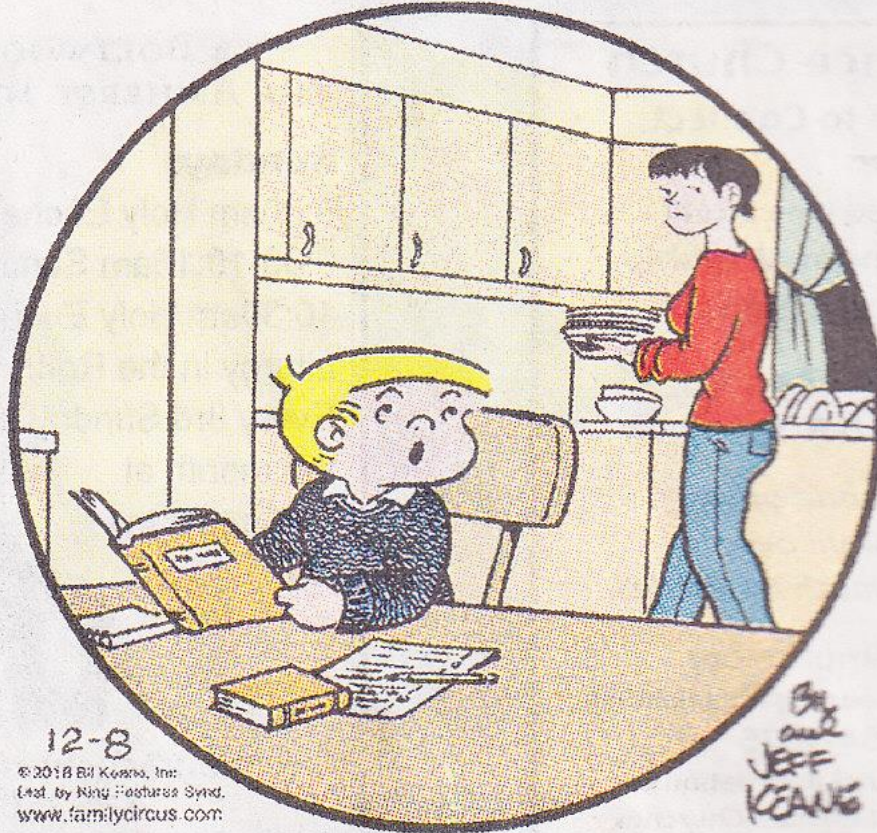
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By
and
JEFF
KEANE

**“My teacher said no man can be
wise on an empty stomach.
I think I need a cookie!”**