Food and Chemical Toxicology 57 (2013) 235–245

Contents lists available at SciVerse ScienceDirect

Food and Chemical Toxicology

journal homepage: www.elsevier.com/locate/foodchemtox

Selenium and mercury molar ratios in commercial fish from New Jersey and Illinois: Variation within species and relevance to risk communication

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ARTICLE INFO

Article history:
Received 22 August 2012
Accepted 12 March 2013
Available online 26 March 2013

Keywords:
Mercury
Selenium
Selenium:mercury molar ratios
Risk balancing

ABSTRACT

There is an emerging consensus that people consuming large amounts of fish with selenium:mercury ratios below 1 are at higher risk from mercury toxicity. As the relative amount of selenium increases compared to mercury, risk may be lowered, but it is unclear how much excess selenium is required. It would be useful if the selenium:mercury ratio was relatively consistent within a species, but this has not been the case in our studies of wild-caught fish. Since most people in developed countries and urban areas obtain their fish and other seafood commercially, we examined selenium:mercury molar ratios in commercial fish purchased in stores and fish markets in central New Jersey and Chicago. There was substantial interspecific and intraspecific variation in molar ratios. Across species the selenium:mercury molar ratio decreased with increasing mean mercury levels, but selenium variation also contributed to the ratio. Few samples had selenium:mercury molar ratios below 1, but there was a wide range in ratios, complicating the interpretation for use in risk management and communication. Before ratios can be used in risk management, more information is needed on mercury:selenium interactions and mutual bioavailability, and on the relationship between molar ratios and health outcomes. Further, people who are selenium deficient may be more at risk from mercury toxicity than others.

1. Introduction

Mercury is considered a global environmental problem because it is ubiquitous and undergoes biomethylation to methylmercury which in turn bioaccumulates and bioamplifies up the food chain. In aquatic food chains, the highest bioaccumulation of mercury generally occurs in fish-eating species, and in large-sized or older organisms (Sormo et al., 2011). All forms of mercury are toxic to probably all forms of life, but methylmercury has higher bioavailability from food and greater toxicity than elemental or inorganic species of mercury. The primary source of mercury exposure in humans is from fish consumption (Rice et al., 2000), and levels of methylmercury in some fish are high enough to cause toxic effects in the fish themselves and in top-level predators, including humans, who consume the fish (WHO, 1989). People who consume large amounts of such fish are at risk from chronic exposure to methylmercury (Grandjean et al., 1997; IOM, 2006; Gochfeld, 2003; Hites et al., 2004; Burger et al., 2007).

Effects from high methylmercury exposure include neurodevelopmental deficits (Steuerwald et al., 2000; NRC, 2000; Trasande et al., 2005), developmental and behavioral deficits in infants (JECFA, 2003; Stringari et al., 2008), and poorer cognitive test performance from fetal and childhood exposure (Oken et al., 2008; Freire et al., 2010). Methylmercury exposure in adults can counteract the cardioprotective effects of fish consumption (Rissanen et al., 2000; Guallar et al., 2002), promote development of cardiovascular disease (Choi et al., 2009; Roman et al., 2011), and result in neurological and locomotory deficits (Hightower and Moore, 2003; Zahir et al., 2005).

However, fish and seafood are an important source of protein and other nutrients (Brunner et al., 2009; NRC, 2000). Fish are not only a low-fat source of protein, but some species also contain high levels of omega-3 (n-3) polyunsaturated fatty acids (PUFAs) that are associated with positive pregnancy outcomes (Kris-Etherton et al., 2002; Daviglus et al., 2002), better child cognitive test performances (Oken et al., 2008), lowered asthma rates in children (Hodge et al., 1996), and lower incidences of cardiovascular

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http://dx.doi.org/10.1016/j.fct.2013.03.021

disease (Virtanen et al., 2008; Ramel et al., 2010). Some fish also contain high levels of selenium, an essential trace element that, among other functions, plays an antioxidant role and may confer some protection against mercury (Kaneko and Ralston, 2007; Ralston, 2009; Ralston and Raymond, 2010). Human, and particularly pre-natal, exposure to methylmercury can be lowered by reducing mercury in the environment (e.g. cutting emissions from coal-fired power plants), harvesting fish from low-mercury environments, or by modifying human fish consumption behavior. In the United States, many states have responded to high mercury levels in freshwater fish by issuing consumption advisories, and the US Food and Drug Administration (US FDA, 2001) has issued advisories for saltwater fish. EPA also issues guidance and warnings about high mercury levels in fish (US FDA-EPA, 2004, 2005). However, advisories are often ignored or misunderstood (Burger, 2000). The FDA warnings about fish consumption may have resulted in decreased fish consumption, especially canned fish (Shimshack et al., 2007). However, commercial statistics indicate that fish species with high mercury levels actually make up only a small share of seafood consumption, at least in the United States (Groth, 2010).

Determining the toxicity of methylmercury to humans and other vertebrates is not always clearcut since a number of factors affect uptake, toxicokinetics, and toxicity, including co-occurrence with other metals and vitamins, nutritional status and probably genetic susceptibility (Haley, 2005; Beyrouty and Chan, 2006; Ralston, 2008; Borderias and Sanchez-Alonso, 2011). From the mid-1960s to the early 1980s some studies showed that selenium could protect against mercury toxicity (Pařizek and Oštádalová, 1967; Lindh and Johansson, 1987), and also suggested that mercury might protect against selenium toxicity. Although most mercury toxicity has been attributed to binding to sulphur, mercury also binds to selenium with a high affinity.

Low levels of selenium are associated with increased coronary heart disease (Seppanen et al., 2004), while higher (but subtoxic) levels of selenium are associated with lower levels of nonfatal heart attacks (Mozaffarian, 2009). High maternal exposure to methylmercury in animals inhibits selenium-dependent enzyme activity in the brain while selenium supplementation is protective (Berry and Ralston, 2008). Sormo et al. (2011) have proposed that selenium moderates mercury toxicity in free-ranging fish (Sormo et al., 2011). Selenium and mercury interact in complex ways to influence egg hatchability and chick defects in ducks (Heinz et al., 2011).

Mercury acts on multiple endpoints. Mercury and methylmercury are irreversible selenoenzyme inhibitors that impair selenoprotein form and function (Watanabe et al., 1999; Carvalho et al., 2008). Therefore one proposed mechanisms of toxicity is whether binding to mercury produces a relative selenium-deficiency, resulting in inadequate synthesis of seleno-enzymes or inhibition of their activity (Ralston, 2008, 2009; Ralston et al., 2008). Selenoenzymes play an important role in antioxidant defenses, which may explain the oxidative damage attributable to methylmercury (Cabanero et al., 2007; Pinheiro et al., 2009; Ralston and Raymond, 2010). The toxicokinetics and toxicodynamics of the selenium and mercury interactions require extensive study as effects differ depending on the forms or species of selenium and of mercury (Dang and Wang, 2011; Khan and Wang, 2009), administration methods (Klimstra et al., 2011), and relationship among them (Falnoga et al., 2006; Farina et al., 2011). There is a limit to the protection of selenium on mercury toxicity, and selenium itself can be highly toxic (Klimstra et al., 2011).

Table 1
Total mercury and selenium levels (ppm, wet weight) (μg/g) and molar ratios in fish species collected from fish markets and grocery stores in New Jersey and Chicago, Illinois. Given are arithmetic means ± SE, and Kendall Tau correlation coefficient of the ratio with mercury concentration. Hg:Se, the reciprocal of Se:Hg is shown for comparison with papers reporting mercury:selenium ratios. Kruskal-Wallis one way ANOVA comparison among species.

Common name	*n*	Mercury μg/g Mean ± SE	Selenium μg/g Mean ± SE	Hg nmol/g wet wt.	Se nmol/g wet wt.	Se:Hg ratio (means)[a]	Se:Hg ratio correlation with Hg tau (*p*)	Hg:Se[b]
New Jersey markets								
Yellow fin tuna	(49)	0.65 ± 0.1	0.75 ± 0.05	3.22	9.44	2.93	−0.7 (<0.0001)	0.34
Chilean sea bass	(7)	0.38 ± 0.06	1.02 ± 0.12	1.87	12.89	6.90	−0.7 (0.02)	0.15
Bluefish	(50)	0.26 ± 0.02	0.51 ± 0.04	1.31	6.51	4.96	−0.5 (<0.0001)	0.20
Red snapper	(4)	0.24 ± 0.01	0.91 ± 0.09	1.20	11.56	9.66	−0.3 (NS)	0.10
Croaker	(14)	0.14 ± 0.02	0.77 ± 0.11	0.72	9.79	13.64	−0.6 (0.006)	0.07
Cod	(7)	0.11 ± 0.01	0.70 ± 0.13	0.54	8.87	16.47	−0.2 (NS)	0.06
Porgy	(16)	0.10 ± 0.01	0.95 ± 0.11	0.47	11.97	25.27	−0.6 (0.002)	0.04
Flounder	(54)	0.05 ± 0.001	0.31 ± 0.03	0.23	3.94	17.18	−0.5 (<0.0001)	0.06
Whiting	(16)	0.04 ± 0.004	0.93 ± 0.14	0.17	11.73	67.21	−0.3 (0.08)	0.01
Shrimp (small)	(12)	0.02 ± 0.001	0.16 ± 0.03	0.07	2.08	27.78	0.0 (NS)	0.04
Scallops	(12)	0.01 ± 0.001	0.05 ± 0.01	0.06	0.68	10.55	−0.1 (NS)	0.09
Shrimp (large)	(12)	0.01 ± 0.01	0.23 ± 0.03	0.05	2.89	57.92	−0.2 (NS)	0.02
Kruskal Wallis X^2 (*p*)		203 (<0.0001)	145 (<0.0001)	145 (<0.0001)				
Chicago markets								
Swordfish	(18)	1.31 ± 0.19	0.63 ± 0.05	6.54	8.03	1.23	−0.67 (<0.0001)	0.81
Orange roughy	(19)	0.57 ± 0.06	0.75 ± 0.04	2.84	9.46	3.33	−0.68 (<0.0001)	0.30
Walleye pollock	(18)	0.51 ± 0.13	0.47 ± 0.03	2.53	5.95	2.35	−0.74 (<0.0001)	0.43
Tuna steak	(18)	0.35 ± 0.06	0.82 ± 0.03	1.72	10.41	6.05	−0.84 (<0.0001)	0.17
Canned tuna (White)	(21)	0.31 ± 0.03	0.83 ± 0.04	1.54	10.57	6.89	−0.66 (<0.0001)	0.15
Grouper	(18)	0.26 ± 0.06	0.59 ± 0.06	1.29	7.46	5.80	−0.93 (<0.0001)	0.17
Canned tuna (Light)	(19)	0.10 ± 0.02	0.89 ± 0.05	0.49	11.32	22.96	−0.84 (<0.0001)	0.04
Canned tuna (Gourmet)	(18)	0.06 ± 0.01	1.02 ± 0.05	0.30	12.89	42.96	−0.83 (<0.0001)	0.02
Salmon	(18)	0.03 ± 0.01	0.35 ± 0.03	0.15	4.45	28.86	−0.62 (0.0004)	0.03
Kruskal Wallis X^2 (*p*)		104 (<0.0001)	100 (<0.0001)	103 (<0.0001)				

[a] The Se/Hg molar ratios are calculated on unrounded mean Hg and Se values.
[b] The correlations for Hg:Se ratio with mercury and length are the same as Se:Hg ratio correlations with mercury and length, only positive.