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Mercury and selenium levels, and selenium:mercury molar ratios of brain, muscle and other tissues in bluefish (*Pomatomus saltatrix*) from New Jersey, USA

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Abstract

A number of contaminants affect fish health, including mercury and selenium, and the selenium:mercury molar ratio. Recently the protective effects of selenium on methylmercury toxicity have been publicized, particularly for consumption of saltwater fish. Yet the relative ameliorating effects of selenium on toxicity within fish have not been examined, nor has the molar ratio in different tissues, (i.e. brain). We examined mercury and selenium levels in brain, kidney, liver, red and white muscle, and skin and scales in bluefish (*Pomatomus saltatrix*) from New Jersey to determine whether there were toxic levels of either metal, and we computed the selenium:mercury molar ratios by tissues. Total mercury averaged 0.32 ± 0.02 ppm wet weight in edible muscle and 0.09 ± 0.01 ppm in brain. Selenium concentration averaged 0.37 ± 0.03 in muscle and 0.36 ± 0.03 ppm in brain. There were significant differences in levels of mercury, selenium, and selenium:mercury molar ratios, among tissues. Mercury and selenium levels were correlated in kidney and skin/scales. Mercury levels were highest in kidney, intermediate in muscle and liver, and lowest in brain and skin/scales; selenium levels were also highest in kidney, intermediate in liver, and were an order of magnitude lower in the white muscle and brain. Mercury levels in muscle, kidney and skin/scales were positively correlated with fish size (length). Selenium levels in muscle, kidney and liver were positively correlated with fish length, but in brain; selenium levels were negatively correlated with fish length. The selenium:mercury molar ratio was negatively correlated with fish length for white muscle, liver, kidney, and brain, particularly for fish over 50 cm in length, suggesting that older fish experience less protective advantages of selenium against mercury toxicity than smaller fish, and that consumers of bluefish similarly receive less advantage from eating larger fish.

Keywords

Mercury; Selenium; Molar ratios; Risk to fish; Marine

1. Introduction

Fish are an important component of marine food chains, including those leading to humans. Not only are fish an important source of protein throughout the world, but fishing is an important commercial enterprise, and provides valuable recreational, cultural, and esthetic benefits (Toth and Brown, 1997; Burger, 2000, 2002; Burger et al. 2007). In many places, fish and shellfish provide the only readily available protein. Fish consumption is associated with lower blood cholesterol, positive pregnancy outcomes, better child cognitive test performance, lower blood pressure, and reduced incidences of stroke, heart disease, and pre-term delivery (Anderson and Wiener, 1995; Guallar et al., 2002; Daviglus et al., 2002; Patterson, 2002; Oken et al., 2008; Virtanen et al., 2008; Ramel et al., 2010), mainly due to omega-3 fatty acids. Thus, consuming fish confers a number of health benefits besides providing protein.

However, high levels of some contaminants in fish, such as methylmercury, can pose health risks to human consumers (IOM 1991, 2006; NRC, 2000; Hightower and Moore, 2003; Burger and Gochfeld, 2007), and to other top-level predators consuming them (Eisler, 1987; Frederick et al., 1999; Spalding et al., 2000a, 2000b; Heinz et al., 2009; Frederick and Jayasena, 2010; Jackson et al., 2011). Mercury can counteract the positive effects of omega-3 fatty acids in fish (Khan and Want, 2009). In humans, levels of methylmercury can be sufficiently high in some fish to cause adverse human health effects (IOM, 1991; Gochfeld, 2003; Hites et al., 2004), particularly to developing fetuses (NRC, 2000; JECFA, 2011). Much less attention has been devoted to demonstrating the toxic effects of mercury on the fish themselves, although Weis and colleagues found several effects of mercury on predator/prey behavior and foraging behavior (Weiss, 2009; Candelmo et al., 2010; Weis and Candelmo, 2012).

In contrast to mercury, selenium is an essential trace element, and it has a protective role on genetic damage and possibly on cancer (El-Bayoumy, 2001). Selenoproteins confer antioxidant properties and are necessary for thyroid function. Selenium deficiencies can cause problems if human intake levels fall below 0.1 ug/g per day (Banuelos and Meek, 1989; Wang and Gao, 2001; ATSDR, 2003; Mozaffarian and Rimm, 2006; Yang et al., 2008). However, high levels of selenium can be toxic, particularly for birds (Ohlendorf and Hothem 1995; Ohlendorf 2000), and for other vertebrates (Eisler, 2000). In fish, metabolic stress increases the toxicity of selenium (Lemly, 1993). The range of beneficial action is relatively small (Diaz-Alarcon et al. 1994; WHO, 2004). Selenium is also known to ameliorate the toxic effects of methylmercury (Satoh et al., 1985; Raymond and Ralston, 2004; Ralston, 2008, 2009; Ralston et al., 2008), and mercury can protect against the toxic effects of selenium (Cuvin-Aralar and Furness, 1991).

Most of the attention on the beneficial effects of selenium on methylmercury toxicity has focused on laboratory studies with mammalian models (e.g. Cabanero et al., 2007) and on

field studies to assess a protective benefit that selenium might confer on mercury toxicity in fish consumed by humans. Laboratory studies have demonstrated that mercury binds to selenium with a high affinity, and high mercury exposure has an adverse impact on selenoenzymes (Beyrouthy and Chan, 2006; Cabanero et al., 2005, 2007; Pinheiro et al., 2009). Field studies generally examine the levels of selenium and mercury, and sometimes include the molar ratios (e.g. Cappon and Smith, 1981; Dorea et al., 1998; Burger et al., 2001; Cabanero et al. 2005, 2007; Burger 2012). In the latter studies, mercury and selenium levels are determined in muscle fillets that are eaten by people. In other studies of fish in nature, attention is directed only at selenium and mercury in edible fillets (e.g. Kaneko and Ralston, 2007; Burger and Gochfeld, 2012). Studies in whole lake ecosystems have shown that increased levels of selenium result in lower levels of mercury in fish, but again internal tissues were not specifically examined (Paulsson and Lundbert, 1989; Chen et al., 2001; Yang et al., 2008). There are few studies that have examined the levels of selenium and mercury in the brain and other internal tissues of fish to ascertain possible positive benefits of selenium on mercury toxicity.

This paper examines the levels of mercury and selenium in the brain, liver, kidney, muscle, and skin/scales of bluefish (*Pomatomus saltatrix*), to determine the selenium: mercury molar ratios in each tissue, and to consider whether either the levels or the ratio were significantly correlated with fish length. We were especially interested in the levels of mercury and selenium that penetrated the blood-brain barrier, and whether the selenium: mercury ratios in the brain were higher than in other tissues, suggesting the possibility for higher protectiveness. Bluefish were selected because they are a top-level predatory fish that are required to capture their prey, and any cognitive deficits would put them at a disadvantage (Weis and Candelmo, 2012). Further, bluefish are one of the preferred fish of recreational fishermen along the Atlantic coast, and are often frozen for consumption throughout the year (Pottern et al., 1989; Burger, 2004, 2009; Gobeille et al., 2006). Bluefish are facultative users of both estuaries and the ocean (Able, 2005). Thus the relative levels of mercury and selenium are also of interest for human consumers, as well as for a range of other top-level predators that eat small, young bluefish. Previous analysis of fillets of bluefish indicated that mercury, but not selenium, was highly correlated with size. Large bluefish had mercury levels above 0.3 ppm, and mean levels of mercury were sufficiently high to cause adverse effects in sensitive birds and mammals, including humans (Burger, 2009). However, mercury and selenium levels were only examined in white muscle. Similarly, Alexander et al. (1973) found a significant correlation between mercury levels and weight of bluefish from Long Island.

Selenium is potentially protective of the toxic effects of mercury (Satoh et al. 1985; Lindh and Johansson 1987; Berry and Ralston, 2008; Ralston 2008). Epidemiological studies with humans have documented the protective effect of selenium on mercury toxicity (Lemire et al. 2010), and Mozaffarian (2009) found that lower levels of nonfatal heart attacks are associated with higher levels of selenium. The mutual effects of selenium and mercury are currently fertile areas of research (Dang and Wang 2011). Methylmercury toxicity may be due in part to impaired selenium-dependent enzyme synthesis or activity because methylmercury is an irreversible selenoenzyme inhibitor that impairs selenoprotein form and

function (Watanabe et al. 1999; Ralston 2008, 2009; Ralston et al. 2008; Carvalho et al., 2008). Cell culture studies and animal experiments show that adverse impacts of high methylmercury exposure on selenoenzymes (particularly glutathione peroxidase and thioredoxin reductase) occur as a result of selenium-mercury interaction, which may explain oxidative damage attributable to methylmercury (Beyrouy and Chan 2006; Cabanero et al. 2007; Pinheiro et al. 2009; Ralston 2009). Mulder et al. (2011) recently showed that may disrupt selenium-dependent functions in free ranging fish, making it even more critical to examine the ratios in a range of wild fish.

Ralston and others have suggested that an excess of selenium over mercury protects against methylmercury toxicity, as measured by selenium: mercury molar ratios, and that higher ratios are more protective than lower ratios (Ralston 2008; Peterson et al. 2009a,b). They argued that the molar ratios in fillets of marine fish are generally high, counterbalancing the effect of mercury. The actual ratio that is protective is unknown, both for vertebrate classes, species, individuals, and for sensitive tissues (such as the brain). Further, there are few data that examine other tissues within a fish species to understand how the selenium and mercury levels and the molar ratio vary, especially for the brain. Since the toxic effects of methylmercury often function on cognition, the relative ratio of selenium to mercury in the brain is of interest. Thus, we particularly focused on the molar ratio for the bluefish brain.

2. Methods

Bluefish (N = 40) were collected from the New Jersey Atlantic coast in 2011, by fishing from shore or from small boats, with protocol approvals from the Rutgers University Institutional Review Board (97-017). We intentionally collected fish of different lengths to determine the effect of size (and age) on mercury and selenium levels, and on the molar ratios. Mercury levels generally increase with the size of the fish (Braune 1987). All fish were labeled by date and location, and some were frozen for later dissection. Prior to dissection, fish were weighed and their fork lengths measured. Tissues dissected included brain, liver, kidney, skin and scales, and white and red muscle. White muscle is the usual muscle in fillets. The red muscle extends along the dorsal line of the back, and is sometimes removed by fishermen because it is the “strongest-tasting” part of the fish. The white muscle samples would be considered edible fillets.

At EOHHSI a 2 g (wet weight) sample of fish tissue was digested in Ultrex ultrapure nitric acid in a microwave (CEM, MDX 2000), using a digestion protocol of three stages of ten minutes each under 50, 100, and 150 pounds per square inch (3.5, 7, and 10.6 km/cm²) at 70 % power. Digested samples were subsequently diluted to 25 ml with deionized water. All laboratory equipment and containers were washed in 10 % HNO³ solution and rinsed with deionized water prior to each use.

Total mercury was analyzed by the cold vapor technique using the Perkin Elmer FIMS-100 mercury analyzer with instrument detection levels of 0.2 ng/g, and a matrix level of quantification of 0.002 ug/g. Selenium was analyzed by the graphite furnace atomic absorption with Zeeman correction. All concentrations are expressed in parts per million on a wet weight basis.

All specimens were run in batches that included blanks, duplicate and spikes specimens. The accepted recoveries for spikes ranged from 85 % to 115%; no batches were outside of these limits. The coefficient of variation (C.V.) on replicate samples ranged from 2–9 %. A DORM-2 Certified dogfish tissue was used as the calibration verification standard for mercury, and standard reference material (SRM) 1640, “Trace Elements in Natural Water” from the National Institute of Standards and Technology (NIST) was used for Zeeman graphite furnace atomic absorption spectroscopy (selenium). Further quality control included periodic blind analysis of an aliquot from a large sample of known concentration.

The selenium:mercury molar ratio was then computed. For ease of comparison with other studies, some of which give mercury:selenium molar ratios, we also present mercury:selenium molar ratios. The selenium:mercury molar ratios were computed by two methods (both of which are used in the literature): 1) calculated on the mean mercury values, and the mean selenium values, and 2) calculated for each individual fish and then averaged to get a mean value. For the selenium:mercury molar ratio was obtained by dividing the molecular weight (200.59 for Hg and 78.9 for Se). Since we analyzed total mercury we did not convert to methylmercury (MW = 215). For fish, especially marine fish, about 90 % or more of total mercury is methylmercury (Lansens et al., 1991; Duffy et al., 1999; Scudder et al., 2009).

We used Kruskal Wallis non-parametric Oneway ANOVA X^2 values to test for differences in mercury levels, selenium levels, and the selenium:mercury molar ratios among tissues, and Kendall Tau correlations to examine the relationship between the molar ratios and mercury levels, and between the molar ratios and fish size (e.g. length) (SAS 2005). The level for significance was designated as < 0.05 . Hereafter the term selenium:mercury refers to the selenium:mercury molar ratio, even when just selenium:mercury ratio is given.

3. Results

There were significant differences in mercury and selenium levels in different tissues (Table 1). Levels of mercury were highest in the kidney, intermediate in muscle and liver, and lowest in the brain and skin/scales. Levels in the brain were an order of magnitude lower than in the kidney and muscle. Selenium levels were highest in the kidney, followed by the liver, and were lowest in the brain and white muscle. Levels of selenium and mercury were not correlated in brain, liver, or in muscle, but were significantly correlated in kidney and skin/scales (Table 1, Fig. 1). Mercury levels in muscle, kidney and skin/scales were positively correlated with length and fish weight (Table 2). Selenium levels in muscle, kidney and liver were positively correlated with fish length and weight, and selenium levels in brain and skin/scales were negatively correlated with fish length and weight.

The selenium:mercury molar ratios differed significantly among tissues, regardless of the method of calculation (Table 1). However, calculating selenium:mercury molar ratios of each individual fish to get the mean ratio gives a higher ratio (i.e. more excess selenium). For all tissues, the ratio was greater than 1. The lowest ratio was in white muscle, the tissue usually eaten by humans and other top-level predators. The ratio was slightly higher in red muscle, the heavily vascularized muscle on the top of the back. The highest ratio for internal tissues was in the kidney.

Patterns of the relationship between fish length and selenium:mercury molar ratios for individual bluefish are shown in figures 2 and 3. For all tissues, correlations were stronger for fish over 50 cm in length than for smaller fish. It appears that in smaller fish mercury and selenium are not highly correlated, but in larger fish, the selenium:mercury molar ratio decreases with increasing size. The correlation was strongest for liver and brain.

4. Discussion

The main findings of this study were that: 1) There were significant differences in levels of both mercury and selenium, and in the selenium:mercury molar ratios, among tissues, 2) Mercury and selenium levels were both lowest in the brain, 3) Mercury and selenium levels were correlated only in the kidney and skin/scales, 4) Mercury levels in muscle, kidney and skin/scales were positively correlated with fish size (length), and selenium levels in muscle, kidney and liver were positively correlated with fish length, but negatively correlated with fish length in the brain, and 5) The selenium:mercury molar ratio was negatively correlated with fish length for white muscle, liver, kidney, and brain, particularly for fish over 50 cm in length. These will be discussed below, in addition to the significance of having a relatively high selenium:mercury molar ratio in the brain, and possible effects.

4.1. Tissue differences in mercury and selenium levels

Understanding the levels of selenium and mercury in different tissues ultimately requires data from several different fish species before patterns can be established. At present, few studies have examined both selenium and mercury in any tissue, and almost none have examined these ratios in multiple tissues. Thus understanding the relationship between the two, which could lead to mechanistic studies, is difficult.

Mercury and selenium levels were highest in the kidney, and lowest in the brain. However, mercury levels were similar in muscle and liver, while selenium levels were significantly higher in the liver than other tissues. The difference in selenium levels in internal tissues was greater than was the difference in mercury. Levels of selenium were nine times lower in brain than in the kidney, but were similar to levels in muscle. In contrast, mercury levels were only 6 times lower in the brain than in the kidney.

Most studies of internal tissues only examined muscle, liver and kidney (and not the brain), largely because it was thought that the blood-brain barrier prevented methylmercury from entering the brain. However, Adams et al. (2010) reported levels of mercury in muscle (0.56 ppm in one site, 0.33 in another), liver (0.63 ppm vs 0.34 ppm), kidney (1.12 ppm vs 0.44 ppm) and brain (0.24 ppm vs 0.11) for spotted seatrout (*Cynoscion nebulosus*) from South Florida. In that study, kidney levels of mercury were lower, and brain levels were higher than in the bluefish from this study. They also measured red blood (which we did not) and found that the levels in the blood (0.07 ppm and 0.02 ppm) was lower than the brain, suggesting accumulation in the brain above circulating levels. In a study from Portugal, Mieiro et al. (2012) reported mercury levels from golden grey mullet (*Liza aurata*) and European sea bass (*Dicentrarchus labras*) of 0.22 ppm and 0.4 ppm respectively in muscle, 0.45 ppm and 0.6 ppm in liver, and 0.11 ppm and 0.2 ppm in brain from the contaminated site. Thus the levels were all within the same range for muscle, liver and brain, but the

relative mercury burden in the muscle was 83 – 93 % for golden grey mullet and 63 – 96 % for European sea bass (Mieiro et al., 2012). These two studies (and the present one with bluefish) give different results for how much total mercury is entering the brain

In a laboratory exposure study with zebra-seabreams (*Diplodus cervinus*) exposed to methylmercury, Branco et al. (2011) found that the highest concentrations were in the kidney (as we found). Liver had levels 2–3 times lower than kidney, and accumulation in the brain was even lower. 28 days after exposure, levels of total mercury in the kidney were about 4 times higher than the liver, and 7–8 times lower in the brain and muscle (which had similar levels). As with many other studies of mercury, however, they did not measure selenium levels.

In contrast, Dang and Wang (2011) examined the antagonistic interactions of mercury and selenium in a marine fish and found that the interactions are dependent upon their chemical species, at high selenium concentrations in the fish diet, inorganic mercury “assimilation efficiency” was decreased, and that selenium had direct interactions with inorganic, rather than methylmercury, during dietary assimilation. These, and other experiments, suggest that the interactions between mercury and selenium is dependent upon the chemical species, as well as the fish species, but also affects the site of interaction, such as dietary assimilation, in specific organs, or on specific systems (e.g inhibition of the thioredoxin system in the brain, Branco et al., 2011). Interactions can occur in the food, the gut, the blood stream, and in organs. This illustrates the importance of both examining the levels of mercury and selenium in different tissues and of examining the physiological and toxicodynamic effects on organ, metabolic pathways, and enzyme systems (Khan and Wang, 2009), as well as for fetal or embryo development (Nishikido et al., 1987).

4.2. Selenium:mercury molar ratios

There are a growing number of studies of the selenium:mercury molar ratios in the edible muscle (white muscle) of fish, largely because of the potential protective effect of excess selenium on reduction of methylmercury toxicity for humans consuming fish (Kaneko and Ralston, 2007; Burger and Gochfeld, 2012, other references in introduction). Selenium is reported to ameliorate the toxic effects of methylmercury (Satoh et al., 1985; Raymond and Ralston, 2004; Ralston, 2008, 2009; Ralston et al., 2008). Due to the high affinity of selenium to mercury, selenium is thought to sequester methylmercury, and reduce the bioavailability in organisms (Sørmo et al., 2011). However, the results from studies are often contradictory, and suggest that the modification of selenium on mercury bioaccumulation is extremely complex (Dang and Wang, 2011). The range between the beneficial effects of selenium, and its toxicity, are narrow (ATSDR, 2003).

Human autopsy studies have identified methylmercury in the brain, particularly of fish-eating populations (Bjorkman et al., 2007), but these studies did not examine selenium levels. Rouleau et al. (1999) demonstrated that the fish blood-brain barrier is impervious to inorganic mercury in plasma. However, they did find inorganic mercury in specific areas of the brain (e.g. olfactory system, medulla of cerebellum), suggesting that waterborne inorganic mercury was taken up by water-exposed receptor cells of the sensory nerves and transferred directly toward the brain by axonal transport.

Understanding the role of selenium (including different species of selenium) on mercury toxicity has thus become an important area of research because of the potential that selenium could ameliorate the toxic effects of mercury in the muscle of top-trophic level, saltwater, predatory fish that are known to have high levels of mercury (e.g. tuna, swordfish, shark, FDA, 2001; IOM, 2006). While it is important to understand the selenium: mercury molar ratios in muscle of edible fish (in terms of human risk), it is also important to examine these ratios in different internal tissues of the fish themselves to understand the possible protection of mercury toxicity by selenium on predatory fish.

There has been the suggestion that selenium: mercury molar ratios of over 1 are protective against mercury toxicity for humans (Ralston et al., 2007; Peterson et al., 2009a,b; Sørmo et al., 2011). Kaneko and Ralston (2007) proposed a Selenium Health Benefit Value that indicates whether a given fish has sufficient selenium in relation to mercury to have a positive health benefit. While intriguing, this index is hard to interpret given the variability even with individual fish (Burger and Gochfeld, 2012). That is, while there is a mean ratio for a given species of fish from a particular location, individual fish can have very variable ratios. Thus, the practical implications of this are unclear (Watanabe, 2002). Difficulties with using either the molar ratio, or the formula are as follows: 1) the actual ratio that is protective is unclear, 2) the level that is protective for different tissues is unclear, 3) the most sensitive organ (in terms of causing adverse toxic effects) is unclear, and 4) different endpoints (neurological disruption, fetal development, visual changes, nerve damage) clearly requires detailed laboratory and epidemiological studies. Presumably there are interspecific differences in the relative protectiveness of a selenium: mercury molar ratio, and the toxicodynamics of this relationship among fish, birds and mammals may also vary. That selenium has the potential (at some levels) to protect against mercury toxicity is very clear (Eisler, 2000; Cabanero et al., 2005, 2007), making it critical to continue studies in this area. Presumably having excess selenium in the brain is variable since the central nervous system toxicity effects of mercury are well known (IOM 2006). Providing information on relative selenium: mercury molar ratios in different tissues, especially the brain in fish, birds and mammals, are important to this debate.

There were significant differences in the selenium: mercury molar ratios in the different tissues of bluefish in this study; the highest internal tissue ratios were in the kidney, and the lowest ratios were in the muscle. The brain selenium:mercury molar ratios was three times higher than in the white muscle, suggesting that selenium could be protective in the brain of fish, and that proportionately more selenium is entering the brain than is mercury. These findings suggest that it is important to examine these differences in fish with even higher levels of mercury in internal tissues.

4.3 Geographical and temporal comparisons of mercury and selenium levels

While not the main focus of the paper, it is important to track geographical and temporal differences in mercury and selenium levels because of the importance of bluefish for human consumers, as well as their predatory role in marine ecosystems. Since many marine food webs are being “fished down”, with removal of larger members of fish species, with consequences for fish populations (Pauly et al., 1998), it is important to understand any role

contaminants might play in fish population health and well-being. Comparative data are available only for muscle tissue. The fish from this study were collected in 2011, and mean mercury levels were 0.37 ppm in white muscle, and selenium levels were 0.50 ppm.

Mercury and selenium in bluefish muscle were examined from the same sites from 2005 to 2008 (Burger, 2009). Comparing results from the present study to the previous study indicates that: 1) mean levels were 0.32 ppm for mercury (similar or lower than the earlier study) and 0.50 ppm for selenium in the present study (higher than the previous study), 2) bluefish in the present study averaged 53.2 cm in fork length (compared to 57 cm for the previous study), 3) in both studies size was correlated with mercury but not selenium, and 4) the selenium mercury ratio in the present study for white muscle was 3.42 compared to 2.65 for the previous study. The bluefish collected in 2006 were similar in size to those collected in the present study (2011); they averaged 50.2 ± 2.4 cm compared to 53.2 ± 2.65 cm in the present study. The mean mercury levels in the present study were similar to the earlier study (0.37 ppm compared to 0.34 ppm in 2006). However, the mean selenium levels were not (0.50 ppm compared to 0.36 ppm in 2006). Thus the cause of the higher molar ratio in 2011 compared to previous data is due to increases in selenium in muscle, and not to size. The causes of increased selenium uptake are unclear.

Levels of mercury and selenium were also determined in bluefish collected from stores and fish markets in 2002–3 in New Jersey (Burger and Gochfeld, 2005). Blue fish in these markets come directly from New Jersey fisheries, or from nearby New York City fish markets. The mean levels for bluefish fillets was 0.26 ± 0.02 ppm (the small variance presumably reflecting similar sized-fish), and selenium averaged 0.51 ppm. While the mercury levels were lower in the commercial fish, the selenium levels were similar to those reported from 2011 (the present study).

The FDA (2012) also publishes mercury levels in fish on their website. They report mean levels of mercury in bluefish of 0.368 ppm (standard deviation of 0.221 ppm) for 94 samples taken from 1990 to 2010. These levels were slightly higher than the levels reported in the present study, but the samples were taken over a longer time period. Other than the four fish for which the FDA has issued consumption advisories, bluefish has the highest mean reported levels (FDA, 2012).

Taken altogether, the New Jersey data indicate some variation from year to year in both mercury and selenium levels, with associated differences in the selenium molar ratio. Thus the selenium molar ratio can vary, presumably resulting in more or less protectiveness in terms of selenium's ability to protect against mercury toxicity. Compared to other predatory fish in the same New Jersey waters, mercury levels in bluefish were lower than shortfin mako shark (*Isurus ocyrinchus*, 1.83 ppm), Bluefin tuna (*Thunnus thynnus*, 0.52 ppm) and striped bass (*Morone saxatilis*, 0.39 ppm), but higher than several other species (Burger and Gochfeld, 2011).

In nearby Long Island, methylmercury levels in 1 year old bluefish averaged 0.14 ppm, and averaged in 0.23 ppm in 2-year olds, which are within the range reported for New Jersey for this size range (Hammerschmidt and Fitzgerald, 2006). In the early 1970s, level in bluefish

from Long Island were as high as 1.1 ppm, with most ranging from 0.2 to 0.6 ppm (Alexander et al., 1973), which were higher than current levels. Thus, levels of mercury in bluefish appear to be declining (assuming fish sizes were similar), and are currently similar in New York and New Jersey.

4.4 Toxicological implications

Recent studies with the toxicological effects of mercury in fish (summarized in Sandheinrich and Wiener, 2011) indicate that toxic effects occur at levels of mercury in muscle that are well below 1 ppm, at least for some sensitive fish species, and may range from between 0.5 ppm and 1.2 ppm (wet weight). Biochemistry, gene expression, behavior, reproductive, and some histochemistry and growth effects occur at levels below 0.5 ppm, as determined from a review of laboratory studies (Sandheinrich and Wiener, 2011). Presumably there will be species effects, given the recent work by Heinz et al (2009) with avian embryos where even closely-related species differed in sensitivity. Partly this sensitivity may relate to freshwater vs marine species; marine species have evolved with higher mercury levels than freshwater fish due to ambient mercury in marine environments (Fitzgerald and Lamborg, 2003).

The variability in selenium: mercury molar ratios in ocean fish is great, with means ranging from 0.32 for mako shark to 14.12 in yellowfin tuna (*Thunnus albacares*, Kaneko and Ralston, 2007) from the Central North Pacific, from 0.64 for mako shark to 12.5 in canned tuna (Cappon and Smith, 1981, and from 0.34 in mako shark to 61 in menhaden (*Brevoortia tyrannus*, Burger and Gochfeld, 2012) in New Jersey. Some of this variability may be due to habitat use. The fish in the Kaneko and Ralston (2007) study were largely open ocean, while those in the New Jersey study were largely estuarine and near shore. While these variations are of interest to human consumers because of their potential protection against mercury toxicity, for the fish, the importance of the ratios is less clear, and it may be that the ratio in the blood, brain and liver are far more important. In the bluefish in this study, the ratios for brain, kidney and liver were higher than for muscle (the tissue usually examined in fish), suggesting greater protection.

4.5. Human health implications

In the present study, the selenium: mercury molar ratios declined with size of the fish, particularly for fish over 50 cm long. It has been known for some time that mercury increases with fish size (e.g. Lange et al. 1994; Green and Knutzen, 2003; Burger, 2009; Gewurtz et al., 2011). For the fish, this means that as fish get larger, they have less selenium available to protect against mercury toxicity. Further, the decline was most significant in liver and brain, the two tissues where having sufficient selenium might be most important (Sanheinrich and Wiener, 2011). Examinations of molar ratios should include reports of variability. When this variability is examined (Burger and Gochfeld, 2012), two critical aspects are clear: 1) the mean selenium:mercury molar ratio declined with mean size of the fish species, and 2) the selenium:mercury molar ratio declines with fish size within fish species. Both aspects suggest that the larger, predatory, open ocean fish (shark, tuna, swordfish), and the largest individuals of each species, have the lowest selenium: mercury molar ratio. This in turn suggests that the large predatory fish may not provide the best selenium protection against mercury toxicity for human consumers, compared with the

smaller, estuarine fish. Further, the smaller fish of a given species provide higher selenium:mercury molar ratios, suggesting that the protective benefits for human consumers reside with the smaller fish that reside partly in estuaries (rather than the large, predatory fish of the open ocean).

The variability found in the present study, with one species of fish, indicate that variations in mercury levels with size of the fish, with associated differences in the selenium: mercury molar ratio, may make it difficult to use this ratio in risk assessment, risk management, and risk communication at this time. Most state agencies (and others) devising fish consumption advisories do not have the data on mercury levels in individual fish, nor do they have selenium levels in individual fish (both necessary to compute selenium: mercury molar ratios for individual fish). Using mean values does not provide an indication of the variation of selenium: mercury molar ratios among individual fish of the same species. Since the ratios vary so greatly among individuals of the same species, it is impossible for a risk assessor to predict whether a pregnant women would eat several meals with a low selenium: mercury ratio, it is difficult to write advisories that are protective. Thus, at this time, it is unclear how to use selenium: mercury molar ratios in risk management.

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Figure 1.
Relationship between selenium and mercury (ppm, wet weight) for white muscle, brain, kidney and liver of bluefish from New Jersey.

Figure 2.
 Relationship of selenium: mercury molar ratio to fork length for white muscle, red muscle, liver and kidney of bluefish collected from New Jersey

Figure 3.

Relationship of selenium: mercury molar ratio to fork length for brain and skin/scales of bluefish collected from New Jersey

Table 1

Total Mercury and Selenium levels (ppm, wet weight)(ug/g) in Bluefish collected from New Jersey. Given are arithmetic means $\pm$ SE, standard error and non-parametric oneway analysis of variance using the Kruskal-Wallis Chi-square (p).

Tissue	n	Mercury Mean $\pm$ SE	Selenium Mean $\pm$ SE	Hg-Se Correlation tau (p)	Hg nmol/g wet wt.	Se nmol/g wet wt.	Se:Hg ^a	Se:Hg ^b	Hg:Se
White Muscle	40	0.32 $\pm$ 0.02	0.37 $\pm$ 0.02	0.1 (NS)	1.59	4.67	2.93	3.22	0.34
Red Muscle	40	0.37 $\pm$ 0.09	0.50 $\pm$ 0.04	0.03 (NS)	1.86	6.36	3.42	5.08	0.29
Brain	40	0.09 $\pm$ 0.01	0.36 $\pm$ 0.03	0.17 (NS)	0.46	4.55	9.87	11.4	0.10
Kidney	40	0.57 $\pm$ 0.09	3.03 $\pm$ 0.17	0.49 (<0.0001)	2.84	38.3	13.5	20.0	0.07
Liver	40	0.38 $\pm$ 0.06	1.94 $\pm$ 0.08	0.17 (NS)	1.91	24.5	12.8	18.7	0.08
Skin/Scales	15	0.05 $\pm$ 0.01	0.71 $\pm$ 0.06	-0.39 (0.05)	0.26	8.99	34.7	148	0.03
Kruskal Wallis χ^2		98.9 (<0.0001)	152 (<0.0001)				125 (<0.0001)		

^aThe Se/Hg molar ratios are calculated on unrounded mean Hg and Se values.

^bThe Se/Hg molar ratios are calculated on individual samples.

Table 2

Correlation among tissues for mercury and selenium levels in Bluefish. Given are Kendall tau correlations with mercury above the diagonal and selenium below. n=40 for all tissues except skin & scales where n=15.

	Length	Weight	White Muscle	Red Muscle	Brain	Kidney	Liver	Skin & Scales
Length	*****	0.86 (<0.0001)	0.43 (0.0001)	0.24 (0.03)	0.02 NS	0.56 (<0.0001)	0.17 NS	0.43 (0.03)
Weight	0.86 (<0.0001)	*****	0.31 (0.005)	0.16 NS	-0.06 NS	0.51 (<0.0001)	0.10 NS	0.46 (0.02)
White Muscle	0.29 (0.01)	0.32 (0.003)	*****	0.48 (<0.0001)	0.33 (0.003)	0.46 (<0.0001)	0.48 (<0.0001)	0.18 NS
Red Muscle	0.37 (0.001)	0.36 (0.001)	0.21 (0.05)	*****	0.44 (<0.0001)	0.33 (0.003)	0.57 (<0.0001)	0.26 NS
Brain	-0.31 (0.006)	-0.38 (0.0007)	0.22 NS	-0.08 NS	*****	0.23 (0.05)	0.49 (<0.0001)	0.13 NS
Kidney	0.41 (0.0002)	0.41 (0.0003)	0.30 (0.009)	0.34 (0.002)	-0.09 NS	*****	0.37 (0.0009)	0.07 NS
Liver	0.25 (0.03)	0.21 (0.07)	0.19 (0.09)	0.22 (0.05)	0.20 (0.07)	0.42 (0.0002)	*****	0.24 NS
Skin & Scales	-0.21 NS	-0.28 NS	-0.08 NS	0.26 NS	0.21 NS	0.14 NS	0.04 NS	*****