Mercury Advisories and Household Health Trade-offs

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ABSTRACT

We provide evidence on the controversy about the national advisory over mercury in fish. We make three contributions. First, we explore empirically the risk-benefit trade-offs consumers make when substituting between fish species with different mercury and omega-3 fatty acid concentrations. Second, we use a changes-in-changes approach to non-parametrically control for confounding factors and to recover the full response distributions. Third, we use rich household homescan panel data and species-level mercury and omega-3 loadings. The advisory induced reductions in mercury at the expense of substantial reductions in healthful omega-3's. A back of the envelope benefit transfer calculation based upon our empirical estimates and drawing from an existing public health literature finds no clear evidence for net benefits from observed advisory response.

1. Introduction

The conventional economic wisdom is that improved information enhances consumer welfare. Yet, a long-standing scientific controversy debates whether the FDA's prominent 2001 mercury advisory has improved public health (Egeland and Middaugh 1997, Cohen et al. 2005). This debate has recently taken on increased significance as the FDA and the EPA disagree over proposed advisory amendments to encourage more fish consumption (Layton 2008). The 2001 advisory instructed pregnant women and households with young children to limit fish consumption, particularly among species with high mercury concentrations. A tension arises because, as the advisory noted, a moderate amount of fish consumption provides significant health benefits to both adults and young children, particularly in the form of omega-3 fatty acids. The empirical question is whether information-induced consumer substitution patterns preserved healthful levels of omega-3s while reducing mercury exposure.

We provide the first systematic revealed preference analysis of the advisory-induced substitution patterns and the corresponding risk-benefit trade-offs that underpin the controversy. It is not clear a priori that consumers do make a nuanced adjustment. Indeed, the recent psychology and behavioral economics literature emphasizes consumers' cognitive limitations and bounded rationality (Thaler 1992, Kahneman 2003). Consumers often misestimate health risks (Viscusi 1990) and even remember warnings as recommendations (Skurnik et al. 2005, Schwartz et al. 2007). Finally, consumers face considerable uncertainty over the relevant risks and benefits.

We provide three innovations. First, previous studies (Shimshack, Ward, and Beatty 2007, Oken et al. 2003) demonstrated advisory-induced reductions in fish

consumption but failed to consider welfare-relevant substitution possibilities among different species. These studies did not have data on mercury, omega-3's, or species consumed and were thus unable to consider the risk-benefit trade-offs that consumers made. This is particularly important because risk assessments of the advisory have been primarily based upon *assumed* or *hypothesized* advisory response and substitution scenarios; our study provides evidence on the *actual* advisory response and substitution patterns. Second, our use of the quasi-experimental changes-in-changes research design non-parametrically controls for confounding factors and recovers the full response distributions for both mercury and omega-3's. Third, we use rich household homescan panel data and species-level mercury and omega-3 loadings.

Absent a randomized trial, we use Athey and Imbens's (2006) changes-in-changes model. Our treatment group is those considered *at-risk* by the advisory and our control group is households with no children or pregnant women because such households are not directed by the advisory to alter behavior. Changes-in-changes is a non-parametric extension of the traditional difference-in-differences approach to isolating causal effects. This technique identifies the entire distribution of treatment effects, rather than identifying an average treatment effect alone. Notably, the approach also allows us to distinguish the advisory impact for consumers most at-risk from the advisory impact for consumers less at-risk. This model overcomes several well known difficulties with the mean and quantile difference-in-differences methods used in previous studies.

We use rich household-level IRI homescan data. We have every packaged supermarket fish purchase from a panel of nearly 15,000 households in the year before the advisory and the two years after the advisory (2000-2002). We combine consumption

data with detailed information on more than 5,300 unique UPC-level products comprising over 50 species. We translate home fish consumption into household mercury and omega-3 intakes based on measurements reported in the scientific literature and extensive USDA testing. Our exceptionally detailed dataset permits the first assessment of household-level substitution responses to the mercury advisory.

We find that at-risk consumers significantly reduced their omega-3 intake in response to the advisory. The decline occurred everywhere along the per capita omega-3 distribution, including the lower tails. Results were driven by a broad-based decline in consumption of *all* fish. Consumers did not differentially avoid high mercury fish nor did they substitute away from high mercury species into low mercury, high omega-3 species. This is the pattern that one might expect from consumers with poor information and high uncertainty about the relevant risk-benefit trade-offs between different species of fish.

Our empirical results are a first step towards addressing the advisory controversy, but a complete picture would require a full understanding of complex relationships between diet, toxics, nutrients, and health, as well as monetized impacts of each on consumer welfare. Such a full assessment is beyond the scope of this study. However, we do combine our empirical findings with the central estimates from a prominent dose-response meta-analysis to provide a back of the envelope estimate of the relative importance of risk-benefit trade-offs. This exercise provides no clear evidence for net benefits from the actual advisory response. In contrast, had consumers responded to the advisory by eliminating high mercury fish while maintaining constant overall fish consumption, this same exercise suggests that aggregate benefits might have been large.

2. Background

Omega-3 Fatty Acids

Moderate amounts of seafood consumption provide significant health benefits, largely due to omega-3 (n-3) fatty acids. Docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) fatty acids have been linked to reductions in stroke, improvements in immune system function, and decreased coronary heart disease in adults (Kris-Etherton et al. 2002). Omega-3s are also associated with improved fetal brain development, infants' visual development, and infants' neuro-behavioral development (Lauritzen et al. 2001, Neuringer et al. 1994, Oken et al. 2005). Recent evidence even supports a link between fatty acids and fetal, infant, and childhood resistance to neurotoxins, including mercury.

Humans are unable to synthesize polyunsaturated fatty acids, so they must be obtained externally. Nearly all dietary DHA derives from fish and shellfish consumption, and seafood is the most important source of EPA as well (Mahaffey 2004). Much smaller amounts of these fatty acids are obtained from miscellaneous sources such as eggs and certain organ meats. Some plants and oil sources such as flax seeds contain alpha-linolenic acids, but these fatty acids do not share the same benefits as DHA and EPA and within-body conversion efficiencies are less than 10 percent (Holub and Holub 2004). While supplementary sources of omega-3's have proliferated in the past few years, during our sample period the omega-3 supplement market was very small. In 2002, only 2% of the US population had used fish oil supplements even once over the previous year (Kennedy 2005).

Mercury

Coal-fired electrical plants are the largest source of anthropogenic mercury. Mercury binds with sulfuric compounds in coal, and burning releases the mercury into the atmosphere. When atmospheric mercury is deposited into surface water, bacteria convert the mercury into organic methylmercury. It then enters a fish's bloodstream from water passing over gills and accumulates in the tissues. Methylmercury bio-accumulates up the food chain. Even in water where ambient mercury levels are extremely low, mercury concentrations may reach high levels in predatory species like tuna, mackerel, and shark.

For the general public, fish consumption is the primary source of exposure to mercury. Cooking and other forms of preparation do not mitigate exposure. Once consumed, mercury is a neurotoxin. Fetuses and nursing infants are at risk because mercury readily passes through the placenta, concentrates in umbilical tissues, and leaches into breast milk. Even modest mercury concentrations pose a risk of significant harm to the developing neurological systems of fetuses, infants, and young children (National Academies 2005). Consequences may include reduced IQ, learning and attention disorders, and generally slow intellectual and behavioral development. Severe neurological illnesses such as cerebral palsy are possible from unusually high exposure.

The 2001 FDA Commercial Fish Advisory

Until late 2000, U.S. government agencies formally maintained that mercury from fish consumption did not pose significant health threats and that benefits of seafood consumption outweighed risks. Public knowledge of mercury in commercial fish was also limited. FDA focus groups conducted in October 2000 indicated, "None of the [focus]

groups showed much interest or concern about mercury as a hazard in fish before seeing the information pieces….There was little or no awareness in any group of a hazard due to low level mercury exposure from fish consumption that was not due to a specific [localized] pollution problem." (US FDA 2000)

The FDA formally released the mercury in fish advisory on January 12, 2001. The advisory singled out infants, small children, pregnant or nursing mothers, and women who may become pregnant. The advisory named several large fish that these targeted consumers should avoid entirely. More generally, it stated that consumers should limit their consumption of all fish, including canned fish, to no more than 12 ounces per week. While the advisory focused on the risks of mercury in fish, it also stated that seafood is protein-rich, high in nutrients, and low in fat. In fact, the first line of the advisory noted that seafood "can be an important part of a balanced diet." The advisory also indicated that certain fish have lower levels of methylmercury than others and can be safely eaten frequently.

The FDA's outreach program consisted of a two-phase information campaign. Over the course of three months following the advisory, the FDA communicated its message by releasing pre-prepared newsprint and television press releases. Media kits were sent to weekly print news sources, parenting magazines, and women's health periodicals. Phase I of the information campaign also included letters to physicians and health organizations. Phase II was a methodologically similar, but less intense, "reminder" campaign conducted in 2002.

Advisory Response

This study provides the first systematic revealed preference analysis of the risk-benefit trade-offs that consumers actually made in response to the 2001 FDA Commercial Fish Advisory. In related research, Oken et al. (2003) evaluated time trends in fish consumption from April 1999 through February 2002 for women enrolled in a maternal nutrition study at a Massachusetts group practice. They gathered "semiquantitative" data on fish consumption frequency, based on patient recollections over periods from one to three months. Their analysis found evidence for reduced fish consumption after the advisory. However, without a control group it is difficult to know to what extent this reduction was due to the advisory or to confounding factors. Further, their questionnaire data lacked sufficient detail to recover mercury or omega-3 estimates or to meaningfully examine substitution across species.

Cohen et al. (2005) summarized the findings of a Harvard Center for Risk Analysis expert panel on fish consumption. The panel developed dose-response relationships for fish consumption and stroke, heart disease, and prenatal neurobehavioral development. They then applied such relationships to assumed or hypothesized changes in fish, mercury, and omega-3 consumption following a national commercial fish advisory. No actual consumption data was analyzed.[1]

Shimshack, Ward, and Beatty (2007) explored a repeated cross section of consumer expenditure survey data and found evidence for a significant reduction in canned fish consumption after the advisory. They answered questions about demographic

[1] Other related studies measured responses of recreational anglers to localized safety advisories (Belton et al. 1986, May and Burger 1996). Jakus et al. (2002) used assumptions based upon such recreational demand studies to develop health and welfare benefits estimates of a striped bass advisory to Chesapeake Bay anglers.

determinants of canned fish consumption responses to the advisory. We answer different questions: Did at-risk households reduce mercury intake in response to the advisory? Did responding households differentially avoid high mercury fish? Most desirably, did responding households substitute into low mercury, high omega-3 fish? Shimshack, Ward, and Beatty (2007) did not estimate mercury intakes, omega-3 consumption, or account for substitution possibilities and risk-benefit tradeoffs among fish species.

3. Data

Household-Based Scanner Data

Our rich and novel household-level panel data permit our fine-grained analysis of the potential substitution patterns underlying advisory-induced risk-benefit trade-offs. We analyze data from Information Resources, Inc.'s InfoScan Consumer Network database. Here, households scan universal product codes (UPCs) on purchased products from all stores upon returning home from shopping. Our sample contains data on all packaged seafood purchases for consumption within the household, including canned and shelf-stable products, refrigerated products, and frozen products. Approximately sixty percent of US seafood is consumed in the home (US National Academies 2005). We have no reason to believe that consumers' advisory response patterns would be systematically different for other sources of fish consumption, and we are unaware of any systematic data on random weight grocery purchases or out of home consumption for our sample.[2]

The use of household-based scanner data offers numerous advantages over alternative sources of consumption information. First, our dataset contains purchases of more than 5,300 distinct seafood products from a three year panel of nearly 15,000

[2] Neither IRI nor AC Nielson household-level random weight panel data includes fish for our sample period. Kuchler and Tegene (2006) find no systematic difference between fixed weight and random weight beef responses to BSE ('mad cow') announcements.

households. Second, detailed product descriptions, including species type, allow us to combine consumption data with a scientific literature and extensive USDA product testing to translate a household's consumption into its mercury and omega-3 intakes. Product information also allows us to determine how the advisory affected consumption of each fish type, allowing investigations of substitution and differential responses across species. Third, the household-level scanner data avoids the strategic bias, recall bias, and observer bias possible in common survey or diary data collection techniques. Fourth, the data are matched with a diverse set of demographic variables over a wide geographic range. Sampling weights allow us to recover a nationally representative sample.

Sample

Our sample covers the years 2000-2002, starting one year before the January 2001 advisory and extending two years past the advisory to allow time for information dissemination and consumer adjustment. The sample of interest contains 14,821 households with less than three adults and less than three children. To prevent identification of unusually large households, IRI does not provide adequate demographic information for large households, so we omit them. To standardize comparisons across households, we scale all quantities by an adult-equivalence factor to yield per capita measures. Our method for constructing these factors follows USDA practice (Lino 2004).[3]

For every product purchased, we obtain product weight, mercury content, and total omega-3 fatty acid content. Product weight is directly provided by IRI. Mercury is

[3]To be consistent with the literature, we conduct adult-equivalence scale factors for total meat consumption. We use the 1999-2002 Consumer Expenditure Diary surveys to do so. Children under 6 consume approximately 24 percent of adult's consumption, children ages 6-11 consume 29 percent of an adult's consumption, and children ages 12-18 consume 61 percent of an adult's consumption.

constructed by matching fish species, obtained from the detailed product descriptions at the UPC level, with the scientific literature on species-specific mercury concentrations. Omega-3 content is created by matching fish species with the scientific literature on species-specific docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) concentrations. Table 1 summarizes species-specific mercury and omega-3 information.

Once we obtain volume, mercury, and omega-3 quantities for each purchased product, we sum these data to reflect total quantities for each household/year combination. We sum over full years to address seasonality.

4. Methods

Our goal is to assess the impact of the advisory on the mercury exposure, omega-3 intakes, and fish consumption of at-risk households. Since we have a panel dataset, it is perhaps tempting to assess these impacts by simply comparing at-risk households' pre-advisory mercury, omega-3, and consumption quantities with at-risk households' post-advisory mercury, omega-3, and consumption quantities. However, we can not attribute changes in consumption after the advisory to the advisory alone since there may be exogenous shocks in other determinants like prices and substitute prices.

The cleanest way to account for confounding factors would be to examine differential responses between randomly assigned control and experiment groups. While this is not possible ex-post, we mimic this structure by examining differences between households explicitly advised to limit consumption by the advisory and households not targeted by the advisory. The natural experiment afforded by this difference removes the effect of confounding factors and allows us to isolate the effect of the advisory on at-risk households. Our treatment, or quasi-experimental, group is households with pregnant

women, nursing children, or children under 6. We refer to this group as "at-risk." Our quasi-control group is households with no children or pregnant women, ie. those considered not at-risk.[4]

We use Athey and Imbens's (2006) changes-in-changes model to implement the quasi-experimental approach. Changes-in-changes is a non-parametric extension of the traditional difference-in-differences method, but instead of identifying an average treatment effect alone it identifies the entire distribution of treatment effects. Further, this model overcomes several well known difficulties with difference-in-differences such as scale-sensitivity and the possibility of negative predicted consumption.

The changes-in-changes model specifies that individuals in two groups $g \in [a,b]$ experience outcomes *y* in two periods $t \in [1,2]$. Group *a* is the control group, group *b* is the at-risk treatment group, *t=1* is the pre-treatment time period, and *t=2* is the post-treatment time period. Therefore, the treatment is observed only if *g=b* and *t=2*. In our context, the treatment is observed only for at-risk households with pregnant women and/or young children in the post-advisory period.

The model assumes that the outcome *y* for any individual in the absence of treatment depends only on an index of unobserved characteristics *u* and time *t*, which allows for time variant confounding factors. Formally,

$$y = h(u,t), \tag{4.1}$$

where *h* is assumed to be a strictly increasing function of *u*. The distribution of *u* may be different across groups, which is practically important, as assignment to the treatment and control groups depends on demographic characteristics. While the realization of *u* for any

[4] Our results are robust to a wide range of control group definitions. We choose this control to most starkly distinguish treatment households with control households.

particular individual may change over time, an identifying assumption is that the distribution of *u* within a group is unchanged over time.

The changes-in-changes economic model assumes the following. First, the treatment and control groups may have different distributions of unobservables (u) affecting consumption. Second, the distribution of unobservables within both the treatment and control groups is constant over time. Third, the mapping of unobservables to consumption outcomes is monotone and depends on period and treatment classification, but not directly on group.

Given these assumptions, the changes-in-changes model is identified. Let F_{gt} be a cumulative distribution function of outcomes *y* for group *g* in period *t*. Then the predicted counter-factual distribution for group *b* in period 2, in the absence of the treatment can be represented by:

$$\hat{F}_{b2}(y) = F_{b1}(F_{a1}^{-1}(F_{a2}(y))). \tag{4.2}$$

Note that *h(·)* from (4.1) is not directly estimated; as a non-parametric technique, changes-in-changes imposes very little structure on the problem. Accordingly, the model produces robustly consistent estimates at the usual cost of higher variance. Nonetheless, we recover statistically and economically meaningful results with this method.

The proof of (4.2) is given in Athey and Imbens (2006), and the intuition is relatively uncomplicated. Changes-in-changes assumes that the entire distribution of outcomes for the treatment group would experience the same changes over time as the distribution of outcomes for the control group in the absence of the intervention (the advisory). Once we obtain the post-treatment counterfactual distribution, the changes-in-changes estimate for any consumption quantile is the difference between the actually

observed period 2 outcome for the at-risk group and the predicted counterfactual no-advisory period 2 outcome for the at-risk group. Formally, for any quantile q, the estimated change-in-change treatment effect is:

$$\Delta^{CIC} = \hat{F}_{b2}^{-1}(q) - F_{b2}^{-1}(q). \tag{4.3}$$

Our analysis also assumes that changes in the world price for seafood are exogenous, unaffected by the FDA mercury advisory. This is a reasonable approximation, given that households targeted by the advisory are a small proportion of total world demand. According to 2001 FAO statistics, the US accounts for less than 5% of worldwide seafood consumption. Further, at-risk households accounted for less than 5% of US consumption. Our final results show about a 20% reduction in fish consumption for at-risk households. Altogether, the estimated advisory-induced response represents about a 0.05% decrease in world consumption. Demand elasticity estimates for fish vary widely by species and region, but typically exceed -0.5 in magnitude for major food species (Asche and Bjorndal 1999). Given these considerations, the impact of any endogenous price response should be insignificant in practice.

5. Results

Did at-risk households reduce mercury exposure?

Figure 1 presents the 2000 vs. 2002 changes-in-changes estimates of the advisory impact for at-risk households' total per-capita mercury intakes from fish and shellfish.[5] The figure indicates that, relative to the no-advisory baseline predicted by the changes-in-changes assumptions, the advisory induced a broad-based decline in per capita mercury consumption for the treatment group. On average, we find a 17.1 percent decline in target

[5] In addition to 2000 vs. 2002 comparisons, we repeat all analyses for 2000 vs. 2001 as well. All results are similar to those presented in sign and significance, but tend to be smaller in magnitude.

consumers' mercury exposure, with a 90 percent bootstrap confidence interval of [3.7%,32.1%].[6] We also see a particularly strong decline at the upper tail of the per-capita mercury distribution. If there were no health trade-offs from fish consumption, these results would be very promising for public health.

Did at-risk households change omega-3 intakes?

Figure 2 presents the 2000 vs. 2002 changes-in-changes estimates of the advisory impact for at-risk households' total per-capita omega-3 intakes from fish and shellfish. The figure indicates that, relative to the no-advisory baseline predicted by the changes-in-changes assumptions, the advisory induced a broad-based decline in per capita DHA and EPA omega-3 consumption for the treatment group. On average, we find a 21.4 percent decline in target consumers' omega-3 intakes, with a 90 percent confidence interval of [10.2%, 34.5%]. We also see that this decline occurs everywhere along the per capita omega-3 distribution, including the lower tails. The mercury reduction therefore created a substantial trade-off of omega-3 reductions.[7,8]

How did at-risk households reduce fish consumption?

Figures 1 and 2 document that both mercury and omega-3 intakes by at-risk households fell in response to the advisory. This would be consistent with a broad-based decline in fish consumption. Figure 3 presents the 2000 vs. 2002 changes-in-changes estimates of the advisory impact for at-risk households' total per-capita fish and shellfish

[6] The bootstrap procedure re-samples households in order to preserve the panel structure. Reported intervals are based on the bootstrap percentile method with 10,000 replications.

[7] As previously noted, the omega-3 supplement market was insignificant over the sample period. Further, growth in fish oil supplement markets was modest over the same period. The market growth between 2000 and 2002 was approximately $20 million. For comparison, the market growth between 2003 and 2005 was greater than $240 million. It is therefore unlikely that omega-3 supplementation meaningfully alters this conclusion.

[8] It is worth noting that the counterfactual distribution exhibits second order stochastic dominance over the actually observed response distribution. Consequently, under any non-decreasing and concave omega-3 benefit function, expected benefits from omega-3's are reduced by the advisory response.

consumption. The figure indicates that, indeed, the advisory induced a broad-based decline in per capita fish consumption for the treatment group. Baseline per-capita consumption for at-risk households is 5.15 pounds, or 23.3 meals, per year. On average, we find a 21.1 percent decline in at-risk consumers' aggregate fish consumption, with a 90 percent confidence interval of [11.0%, 34.2%]. We also see that this decline occurs everywhere along the per capita volume distribution, including the lower tails.

Did at-risk households differentially avoid high-mercury fish?

Table 2 presents the 2000 vs. 2002 mean changes-in-changes estimates of the advisory impact for at-risk households' per-capita consumption of commonly consumed seafood items. The table indicates that consumption declines for every major commonly consumed fish and shellfish type analyzed. After accounting for confounding factors, at-risk households' white tuna, light tuna, and pollack consumption fell 14.0 percent, 19.5 percent, and 17.9 percent relative to the no-advisory baseline predicted by the changes-in-changes assumptions. At-risk households' shrimp and salmon consumption volume fell 17.5 percent and 27.9 percent respectively.

None of the category-specific consumption reductions are statistically different from one another. We find no evidence for differential avoidance of high mercury fish. Consumption reductions include low mercury, high omega-3 seafoods like shrimp and salmon. White tuna, the commonly consumed species with the highest mercury concentration, fell the least in percentage terms. It does not appear that at-risk households responded to the advisory in a nuanced fashion that recognized trade-offs.

Is the estimation method appropriate?

In principle, it is possible to test and reject the CIC model. In particular, the CIC method should accurately predict actual behavior for time periods when there is no treatment. Unfortunately, no data for our scanner panel exist prior to 2000. However, we gathered repeated cross-section data on canned fish consumption from the Consumer Expenditure Survey (CEX) for multiple years before the advisory. Using demographic definitions of control and treatment groups similar to those in our main analysis, we calculate the CIC mean fish consumption predictions and confidence intervals for the pre-advisory year pairs 1996/97, 1997/98, 1998/99, and 1999/2000. In each case, the actual mean canned fish consumption was within the 90% confidence interval of the CIC prediction, as one would expect absent a treatment. We also applied the CIC method to the CEX data for 2000/01, the period of the advisory. Consistent with the results of our scanner-data analysis, actual mean consumption was statistically below the CIC prediction after the treatment. In short, we reject the CIC predictions only when we have prior reason to believe they should be rejected.

Are key results robust to an alternative estimation method?

We replicated the key analysis with the traditional difference-in-differences research design and find qualitatively similar results. Recall that CIC mean declines for per capita mercury, omega-3s, and volume consumption are 17.1, 21.4, and 21.1 percent, respectively. DID mean declines for per capita mercury, omega-3s, and volume consumption changes are 21.6, 30.0, and 24.5, respectively. 90 percent confidence intervals are [6.7%, 21.6%], [16.0%, 43.8%], [13.2%, 35.7%]. In sum, DID mean

estimates are statistically similar and consistently larger in magnitude. CIC results are conservative relative to DID results.

6. Discussion

Our empirical results show that at-risk consumers' omega-3 intakes from this food source fell 21.4 percent while their mercury intakes fell 17.1 percent, on average, in response to the advisory. The existing risk assessment literature primarily relies upon *assumed* or *hypothesized* advisory response and substitution scenarios. While one should be mindful of the previously discussed caveats to our analysis, this study fills a key gap by providing estimates of the *actual* impact of the advisory on mercury and omega-3 intakes.

Given considerable uncertainties in the relationships between diet, toxics, nutrients, and health, addressing the full complexity of advisory-induced welfare impacts is beyond the scope of this study. Indeed, there is tremendous controversy over these relationships in the epidemiological literature and within regulatory agencies. However, in order to provide a rough guide to the economic significance of our findings, we combine our central empirical behavioral estimates with the central estimates from a prominent dose-response meta-analysis of mercury and omega-3 impacts on childhood IQ and fish consumption impacts on adult CHD and stroke mortality (Cohen et al. 2005). We monetize impacts using standard Environmental Protection Agency benefit transfer figures of $13084 per IQ point and $7.52 million for the value of statistical life (USEPA 2000a, EPA2000b).[9] Our back of the envelope exercise provides a central estimate of -$30 per at-risk household net benefits.[10] While estimates from a full range of parameter

[9] $13084 = $8346 1990USD in 2007USD and $7.52 million = $4.8 million 1990USD in 2007USD.
[10] Details of all calculations are in the appendix.

values would vary on both sides of the central tendency, we can at least conclude that there is no *clear* evidence for significant net benefits.

For comparison, we calculate results for one of Cohen et al. (2005)'s hypothetical substitution scenarios, an idealized best case. In this scenario, at-risk households eliminated consumption of fish containing high or medium concentrations of mercury while holding overall fish consumption constant. That scenario assumes that mercury falls by 47.2 percent and omega-3s increase by 14.3 percent. For this idealized hypothetical case, the same exercise now produces a point estimate of $587 per at-risk household net benefits, or approximately $9.5 billion for nation. In short, while we find no clear evidence for net benefits from actual advisory response, we do find evidence a more nuanced response could have generated economically meaningful net benefits.[11]

Our results reinforce the importance of carefully crafting information policies. The classical economic belief that information provision improves consumer decision-making relies upon assumptions about how consumers understand and process information. Consumers responded coarsely to the FDA mercury advisory by broadly reducing consumption without a clear recognition of varying risk-benefit trade-offs across species. Addressing the psychological and behavioral realities of consumer responses to risk information and uncertainty is essential for advisory design and dissemination.

[11] Our focus is on health; these calculations do not consider potential utility losses due to taste for fish.

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Table 1. Mercury and Omega-3 Concentrations by Species

Seafood	Mercury (in µg/g)	Omega-3 (in g/100g)	Original Hg Source	Omega-3 Source
Anchovy	.043	2.05	NMFS (1978)	USDA (2006)
Catfish	.049	0.17	FDA 1990-2004	Ackman (2000)
Clams	.001*	0.35	FDA 1990-2002	Ackman (2000)
Cod	.095	0.31	FDA 1990-2004	Ackman (2000)
Crab	.060	0.36	FDA 1990-2004	Ackman (2000)
Crayfish	.033	0.17	FDA 2002-2004	USDA (2006)
Croaker	.180	0.24	FDA 1990-2003	Ackman (2000)
Eel	.213	0.96	EPA (1997)	Ackman (2000)
Flounder	.045	0.12	FDA 1990-2004	Ackman (2000)
Grouper	.465	0.30	FDA 2002-2004	Ackman (2000)
Haddock	.031	0.21	FDA 1990-2002	Ackman (2000)
Halibut	.252	0.38	FDA 1990-2004	Ackman (2000)
Herring	.044	2.34	NMFS (1978)	Ackman (2000)
Jack Mackerel	.267	1.23	EPA (1997)	USDA (2006)
Lobster	.240	0.36	FDA 1991-2004	Ackman (2000)
Mackerel	.069	2.29	NMFS (1978)	Ackman (2000)
Mahi	.144	0.22	EPA (1997)	Ackman (2000)
Mullet	.046	0.33	NMFS (1978)	USDA (2006)
Mussel	.030	0.79	Sunderland (2007)	USDA (2006)
Octopus	.029	0.16	EPA (1997)	USDA (2006)
Oyster	.013	0.19	FDA 1990-2004	Ackman (2000)
Perch	.140	0.32	FDA 1990-2002	USDA (2006)
Pike	.310	0.14	EPA (1997)	USDA (2006)
Pollack	.041	0.26	FDA 1990-2004	Ackman (2000)
Redfish	.001*	0.21	FDA 1990-2002	Ackman (2000)
Roughy	.554	0.04	FDA 1990-2004	USDA (2006)
Salmon, atlantic	.014	2.00	FDA 1990-2002	USDA (2006)
Salmon, canned	.001*	1.35	FDA 1990-2002	USDA (2006)
Salmon, chinook	.014	1.74	FDA 1990-2002	USDA (2006)
Salmon, chum	.014	0.81	FDA 1990-2002	USDA (2006)
Salmon, pink	.014	1.29	FDA 1990-2002	USDA (2006)
Salmon, sockeye	.014	1.23	FDA 1990-2002	USDA (2006)
Salmon, unknown	.014	1.46	FDA 1990-2002	USDA (2006)
Sardines	.016	0.98	FDA 2002-2004	USDA (2006)
Scallop	.050	0.37	NMFS (1978)	Ackman (2000)
Seabass	.219	0.15	FDA 1990-2004	Ackman (2000)
Shrimp	.001*	0.44	FDA 1990-2002	Ackman (2000)
Smelt	.108	0.89	FDA 1990-2002	USDA (2006)
Snapper	.189	0.26	FDA 2002-2004	Ackman (2000)
Sole	.045	0.19	FDA 1990-2004	Ackman (2000)
Squid	.070	0.54	NMFS (1978)	USDA (2006)
Sturgeon	.235	1.97	EPA (1997)	Ackman (2000)
Swordfish	.976	0.58	FDA 1990-2004	Ackman (2000)
Tilapia	.010	0.14	FDA 1990-2002	USDA (2006)
Trout	.072	0.62	FDA 2002-2004	Ackman (2000)
Tuna	.383	0.30	FDA 2002-2004	USDA (2006)
Tuna, canned	.118	0.20	FDA 2002-2004	USDA (2006)
Tuna, canned light	.118	0.20	FDA 2002-2004	USDA (2006)
Tuna, canned light oil	.118	0.13	FDA 2002-2004	USDA (2006)
Tuna, canned light water	.118	0.27	FDA 2002-2004	USDA (2006)
Tuna, canned oil	.118	0.13	FDA 2002-2004	USDA (2006)
Tuna, canned water	.118	0.27	FDA 2002-2004	USDA (2006)
Tuna, canned white	.353	0.56	FDA 2002-2004	USDA (2006)
Tuna, canned white oil	.353	0.25	FDA 2002-2004	USDA (2006)
Tuna, canned white water	.353	0.86	FDA 2002-2004	USDA (2006)
Tuna, white (albacore)	.357	0.81	FDA 2002-2004	Ackman (2000)
Turbot	.100	0.62	EPA (1997)	Ackman (2000)
Whitefish	.069	0.21	FDA 2002-2004	USDA (2006)
Whiting	.001*	0.24	FDA 1990-2002	Ackman (2000)

* indicates mercury concentrations were below detection levels. Table I is similar to Tables 2 and 3 in Maheffey (2004). Date ranges indicate that the FDA conducted its test of this species over these years, as reported by USHHS and the US EPA, "Mercury Levels in Commercial Fish and Shellfish," February 2006. Accessible online at www.cfsan.fda.gov/~frf/sea-mehg.html .

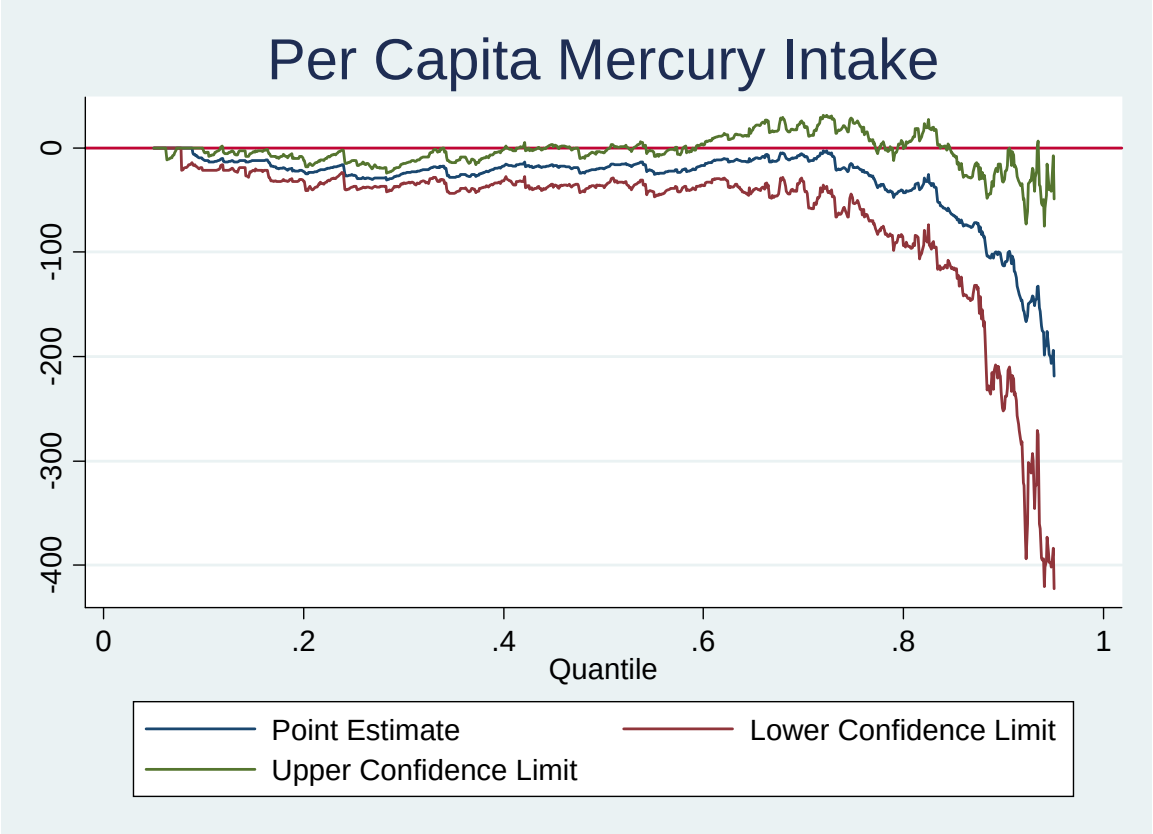

Figure 1. The graph presents the changes-in-changes estimates of the advisory impact on the per capita mercury intake of the at-risk group. For each quantile along the distribution of at-risk consumers' mercury intake distribution, the point estimate is the advisory-induced change in micrograms of mercury intake. The upper and lower lines represent the bootstrapped 90 percent confidence interval for each estimate. For example, the quantity associated with the 90th quantile fell 113.36 micrograms with a confidence interval of [-251.97,-33.14].

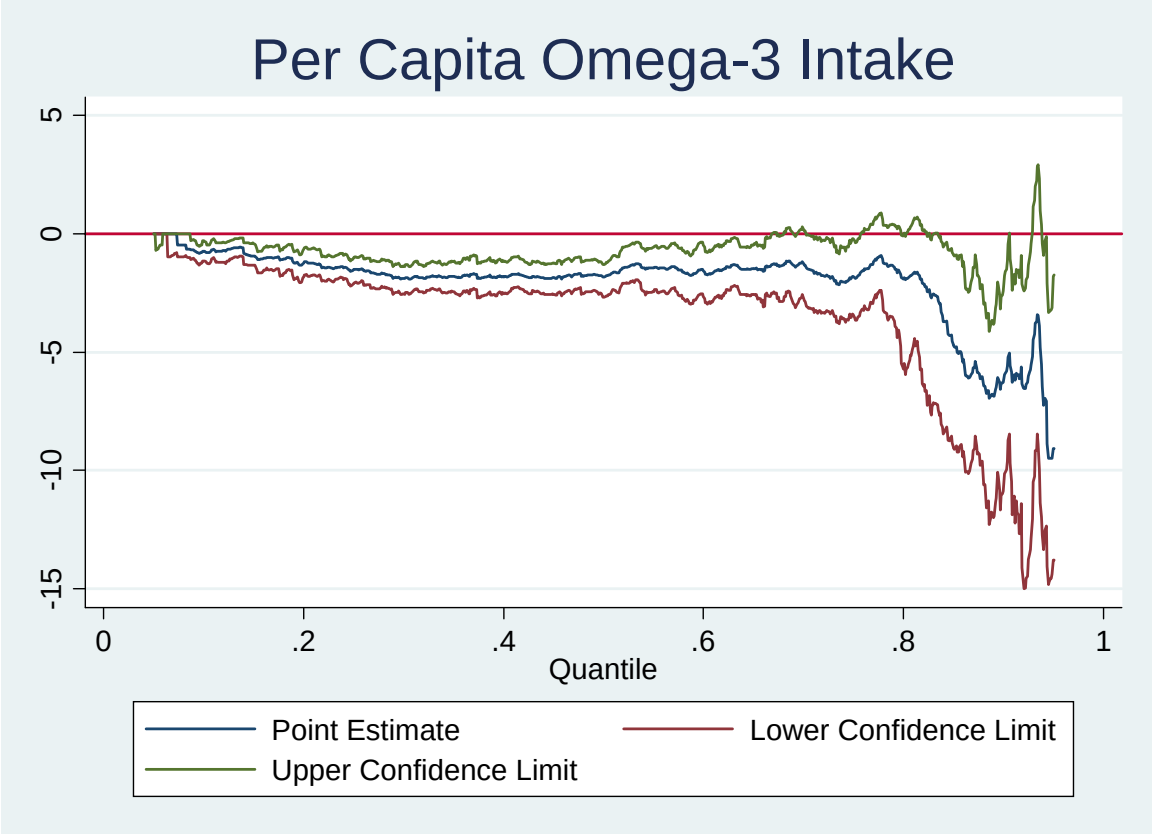

Figure 2. The graph presents the changes-in-changes estimates of the advisory impact on the per capita omega-3 (DHA+EPA) intake of the at-risk group. For each quantile along the distribution of at-risk consumers' omega-3 intake distribution, the point estimate is the advisory-induced change in grams of omega-3 (DHA+EPA) intake. The upper and lower lines represent the bootstrapped 90 percent confidence interval for each estimate. For example, the quantity associated with the 20th quantile fell 1.24 grams with a confidence interval of [-1.82,-0.67].

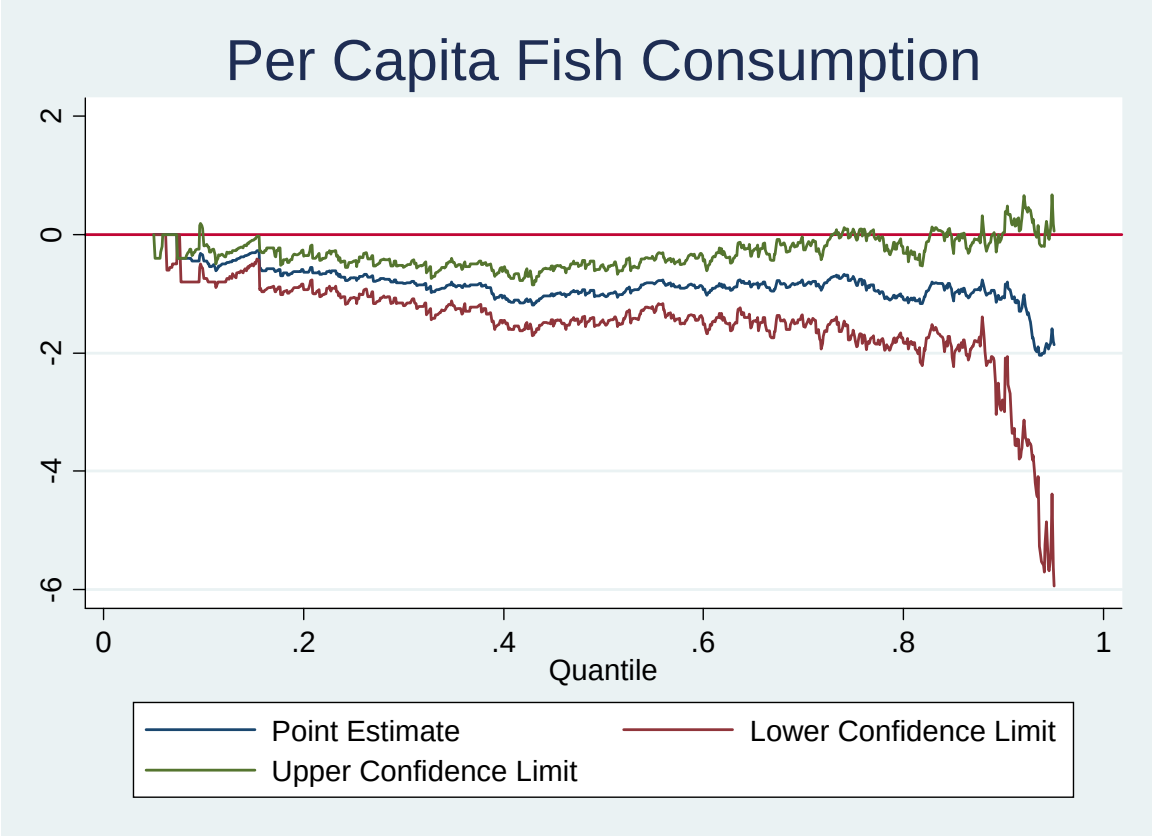

Figure 3. The graph presents the changes-in-changes estimates of the advisory impact on the per capita fish consumption of the at-risk group. For each quantile along the distribution of at-risk consumers' fish weight distribution, the point estimate is the advisory-induced change in pounds of fish consumption. The upper and lower lines represent the bootstrapped 90 percent confidence interval for each estimate. For example, the quantity associated with the 40^{th} quantile fell 1.04 pounds with a confidence interval of [-1.45,-0.56].

Table 2. Mean Per-Capita Seafood Consumption Change (in pounds) by Category

Seafood Type	Mean Changes-in-Changes Point Estimate	Percent Change for Point Estimate	90% CI, expressed as percent change
White Tuna	-0.07	-14.0%	[-36.5%, +12.5%]
Light Tuna	-0.31	-19.5%	[-46.1%, -0.4%]
Pollack	-0.19	-17.9%	[-36.5%, -0.8%]
Shrimp	-0.11	-17.5%	[-39.1%, +0.8%]
Salmon	-0.09	-27.9%	[-65.9%, +3.8%]

Appendix: Health Interpretation Calculations

As summarized in Cohen et al.'s (2005) meta-analysis, the epidemiology literature indicates: (1) 1 microgram of mercury per person per day approximately translates into mercury concentrations of 0.17 micrograms per gram of hair, (2) 1 additional microgram of mercury per gram of hair is approximately equivalent to a loss of 0.7 IQ points, (3) 1 additional gram per person per day of (DHA) omega-3 fatty acids is approximately equivalent to a gain of 1.3 IQ points, (4) the change in relative CHD risk for adults that stop consuming fish is +0.17, (5) the change in relative stroke risk for adults that stop consuming fish is +0.12, (6) the change in relative CHD risk for adults per additional fish serving per week is -0.039, and (7) the change in relative stroke risk for adults per additional fish serving per week is -0.02. Our baseline counterfactual consumption means are: (1) 217.8 micrograms of mercury per person per year, (2) 5.147 pounds of fish per person per year, and (3)10.24 grams of omega-3s per person per year.

Table A1. Health Impacts of the Observed Advisory Response

Health Effect	Risk relationship (central estimate)	Empirical Change (central estimate)	Health Impacts (central estimate)	Valuation per at-risk household
Hg exposure & IQ	-0.00033 IQ pts per ug/year total hg	-37.2 ug/year	+.012 IQ pts/child	+$181
N-3 intake & IQ	+0.0036 IQ pts per g/year total DHA intake	-2.19 g/year	-.008 IQ pts/child	-$120
Fish consumption & CHD and stroke mortality	+6.18 deaths per 100000 adults that stop consuming significant quantities of fish	+8.1 percent households consuming no fish	+.50 CHD deaths/100000 adults	-$72
Fish consumption & CHD and stroke mortality	-0.026 deaths per 100000 adults per additional fish serving/year	-4.9 meals per year	+.13 CHD deaths/100000 adults	-$19
Net health effects per at-risk household				-$30

Notes: Following Cohen et al. (2005), no significant fish consumption is defined as <1 serving/month.

Table A1, Row 1: The risk relationship is obtained as follows: 1 μg mercury per gram of hair is equivalent to 0.7 IQ points lost, so 1 microgram of mercury per person per day is equivalent to 0.17*0.7 IQ points lost. 1 microgram of mercury per person per year is therefore equivalent to (0.17*0.7)/365 days = 0.00033.

The CIC mean change in mercury exposure is obtained by multiplying the empirically observed 17.1 percent fall by the baseline mercury consumption mean of 217.8 micrograms per person per year. 0.171*217.8 = 37.2.

Health impacts are obtained by multiplying the implicit dose-response relationship (0.00033) times the observed annual per capita mercury consumption change (37.2). 0.00033*37.2 = 0.012.

Valuation applies a central IQ estimate of $13,084 per point to at-risk households that average 1.15 young children per HH. 0.012*13084*1.15 = 181.

Table A1, Row 2: The risk relationship is obtained as follows: 1 g of omega-3 per person per day is equivalent to 1.3 IQ points gained, so 1 g of omega-3 per person per year is equivalent to 1.3/365 days = 0.0036.

The CIC mean change in omega-3 exposure is obtained by multiplying the empirically observed 21.4 percent fall by the baseline omega-3 consumption mean of 10.24 grams per person per year. 0.214*10.24 = 2.19.

Health impacts are obtained by multiplying the implicit dose-response relationship (0.0036) times the observed annual per capita omega-3 consumption change (2.19). 0.0036*2.19 = 0.008.

Valuation applies a central IQ estimate of $13084 per point to at-risk households that average 1.15 young children per HH. 0.008*13084*1.15 = 120.

Table A1, Row 3: The change in relative CHD risk for adults that stop consuming significant quantities of fish is 0.17 and the change in relative stroke risk for adults that stop consuming significant quantities of fish is 0.12. Significant consumption is defined to be at least one 100g serving per person per month, equivalent to 2.65 pounds per year. Table 10 of Arias et al. (2003) reports that for ages 15-54, the weighted average CHD death rate is 32.45 deaths per 100000 adults and the weighted average stroke death rate is 5.54 deaths per 100000 adults. Therefore, the risk relationship is (0.17*32.45) + (0.12*5.54) = 6.18 deaths/100000 adults that stop consuming significant quantities of fish.

Our 2002 observed average consumption per person per year for packaged fish is 38% of 2002 National Marine Fisheries Services estimates of per capita consumption (5.93 pounds per person per year relative to 15.6 pounds per person per year). Therefore, we assume less than 1.0 pounds per person per year (.38*2.65) in our dataset is not significant consumption. CIC results reveal an 8.1 percent increase in the number of households consuming less than 1.0 per person per year.

Health impacts are obtained by multiplying the implicit dose-response relationship (6.18 deaths) times the observed reduction in the number of households consuming significant quantities of fish (.081). 0.081*6.18 = 0.50.

Valuation applies a central value of a statistical life (VSL) estimate of $7.52 million to at-risk households that average 1.92 adults per household.(0.50/100,000)*7,520,000*1.92 = 72.

Table A1, Row 4: The change in relative CHD risk for adults per additional fish serving per week is 0.039 and the change in relative stroke risk for adults per additional fish serving per week is 0.02. Table 10 of Arias et al. (2003) reports that for ages 15-54, the weighted average CHD death rate is 32.45 deaths per 100000 adults and the weighted average stroke death rate is 5.54 deaths per 100000 adults. Therefore, the risk relationship is (0.039*32.45) + (0.02*5.54) = 1.376 deaths/100000 adults per additional fish serving per week or 0.026 deaths/100000 adults per additional fish serving per year.

The CIC mean change in fish consumption per person per year is obtained by multiplying the empirically observed 21 percent fall by the baseline consumption mean of 5.147 pounds. 0.21*5.147 = 1.09. 1.09 pounds or 494 grams per year, equivalent to 4.9 meals per year.

Health impacts are obtained by multiplying the implicit dose-response relationship (0.026 deaths) times the observed annual per capita consumption change (4.9). 0.026*4.9 = 0.13.

Valuation applies a central value of statistical life (VSL) estimate of $7.52 million to at-risk households that average 1.92 adults per household. (0.13/100,000)*7,520,000*1.92 = 19.

Table A2. Health Impacts of a Pure Substitution Advisory Response

Health Effect	Risk relationship (central estimate)	Empirical Change (central estimate)	Health Impacts (central estimate)	Valuation per at-risk household
Hg exposure & IQ	-0.00033 IQ pts per ug/year total hg	-102.8 ug/year	+.034 IQ pts/child	+$512
N-3 intake & IQ	+0.0036 IQ pts per g/year total DHA intake	+1.46 g/year	+.005 IQ pts/child	+$75
Net health effects per at-risk household				+$587

Table A2, Row 1: The risk relationship is obtained as follows: 1 μg mercury per gram of hair is equivalent to 0.7 IQ points lost, so 1 microgram of mercury per person per day is equivalent to 0.7*0.17 IQ points lost. 1 microgram of mercury per person per year is therefore equivalent to (0.7*0.17)/365 days = 0.00033.

In Cohen et al. (2005), the ideal substitution scenario assumes a 47.2 percent fall in mercury consumption per person per year. Applying this change to the baseline mercury consumption mean of 217.8 yields a change of 102.8 micrograms per year (.472*217.8).

Health impacts are obtained by multiplying the implicit dose-response relationship (0.00033) times the assumed annual per capita mercury consumption change (102.8). 0.00033*102.8 = 0.034.

Valuation applies a central IQ estimate of $13084 per point to at-risk households that average 1.15 young children per HH. 0.034*13084*1.15 = 512.

Table A2, Row 2: The risk relationship is obtained as follows: 1 g of omega-3 per person per day is equivalent to 1.3 IQ points gained, so 1 g of omega-3 per person per year is equivalent to 1.3/365 days = 0.0036.

In Cohen et al. (2005), the ideal substitution scenario assumes a 14.3 percent increase in omega-3 consumption per person per year. Applying this change to the baseline omega-3 consumption mean of 10.24 yields a change of 1.46 grams per year (.143*10.24).

Health impacts are obtained by multiplying the implicit dose-response relationship (0.0036) times the hypothetical annual per capita omega-3 consumption change (1.46). 0.0036*1.46 = 0.005.

Valuation applies a central IQ estimate of $13084 per point to at-risk households that average 1.15 young children per HH. 0.005*13084*1.15 = 75.

National Calculations: The US census bureau's current population survey reports 16.14 million households with children ages 0-5 in the US population. Our central estimate of household benefits per at-risk household for the pure substitution scenario is $587. 587*16,140,000 = 9.5 billion.

Additional Reference:

Arias et al. "Deaths: Final Data for 2001," *National Vital Statistics Report,* 52(3), 1-116, 2003.