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ABSTRACT

The conventional economic wisdom is that improving consumer information will enhance welfare. Yet, some scientists speculate that the Food and Drug Administration's prominent mercury in fish advisory may have harmed public health. Lower mercury intakes reduce neurological toxicity risks. However, since seafood is the predominant dietary source of healthful omega-3 fatty acids, reduced fish consumption may have significant offsetting health impacts. We explore this risk trade-off using a rich panel of household-level seafood consumption data. To control for confounding factors, we use a non-parametric changes-in-changes approach. We find strong evidence that while the advisory reduced mercury loadings, it did so at the expense of substantial reductions in healthful omega-3s. We find this response pattern even for consumers with low fish consumption. Using advisory response patterns as inputs into a prominent risk assessment model, the central estimate is that net benefits from the advisory were negative.

1. Introduction

The conventional economic wisdom is that improved information enhances consumer welfare. Yet, a long-standing scientific controversy debates whether the Food and Drug Administration (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 Environmental Protection Agency disagree over advisory amendments proposed to encourage more fish consumption (Layton 2008). The 2001 advisory instructed high consuming households with pregnant women and young children to reduce fish consumption to no more than 12 ounces per week, particularly avoiding 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. In particular, since seafood is the predominant dietary source of omega-3 fatty acids, fish consumption may reduce coronary heart disease and stroke risk in adults and enhance neurological development in young children. The empirical question is whether information-induced consumer substitution patterns reduced mercury risks without increasing countervailing risks from lower omega-3s intakes.

We provide systematic analysis of the advisory-induced substitution patterns and the corresponding risk trade-offs that underpin the debate. While a fully rational, fully informed consumer would make a careful trade-off, is not necessarily clear that actual consumers do so; hence the controversy. 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), overemphasize worst case scenarios (Viscusi 1999), and even remember warnings as recommendations (Skurnik et al. 2005, Schwartz et al. 2007). Finally, consumers face considerable uncertainty over the relevant risks.

We estimate the impact of the advisory on mercury loadings and omega-3 intakes. Previous risk assessments have been primarily based upon *assumed, hypothesized, or simulated* advisory response and substitution scenarios (Jakus et al. 2002, Carrington et al. 2004, Cohen et al. 2005). In contrast, our study provides evidence on *observed* advisory response and substitution patterns.

Absent a randomized trial, we control for confounding factors like prices and advertising using Athey and Imbens's (2006) quasi-experimental changes-in-changes approach. Our treatment group is those considered potentially *at-risk* under the advisory: households with pregnant women and children under age 6. Our control group is households with no children or pregnant women because such households are never 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 allows us to distinguish the advisory impact at the upper tail of the mercury (or omega-3) distribution from the advisory impact at the lower tail of the mercury (or omega-3) distribution. The changes-in-changes model also overcomes several well known limitations of alternatives such as the mean or quantile difference-in-differences methods.

We use rich household-level consumer panel data from Information Resources, Inc (IRI). 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 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 U.S. Department of Agriculture (USDA) testing. Our exceptionally detailed dataset provides early evidence on household-level substitution responses to the mercury advisory.

We find that at-risk consumers as a whole reduced both their mercury and omega-3 intakes in response to the advisory. The omega-3 decline occurred everywhere along the distribution of per capita intakes, including the lower tails. Results were driven by a broad-based decline in consumption of *all* fish by at-risk consumers. On average, consumers did not differentially avoid high mercury fish nor did they substitute away from high mercury species into low mercury, high omega-3 species. We find this response even among college educated households, those presumably most likely to make a nuanced substitution. Moreover, we find that less educated households show no statistically significant advisory-induced reduction in mercury.

To provide evidence on the economic significance of these results, we combine our empirical findings with the central estimates from a prominent dose-response meta-analysis to provide a benefit transfer estimate of the relative importance of risk-benefit trade-offs. This exercise provides no clear evidence for net benefits from the actual advisory response; our central estimate is that net benefits from the advisory were modestly negative. 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 would have been large.

2. Background

Omega-3 Fatty Acids

Moderate amounts of seafood consumption provide significant health benefits, in part 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).

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. In other words, the advisory instructed high consuming at-risk households to reduce fish consumption.

The advisory acknowledged that seafood “can be an important part of a balanced diet.” Fish is protein-rich, high in nutrients, and low in undesirable fats. The advisory also indicated that certain fish have lower levels of methylmercury than others and can be safely eaten frequently. The FDA advised targeted women “to select a variety of other kinds of fish – including shellfish, canned fish, smaller ocean fish, or farm-raised fish.” In other words, the advisory counseled substituting from less healthful to more healthful fish.

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. News stories about the advisory generally reported both the admonishment to reduce mercury exposure and also the recommendation to selectively substitute and continue to eat more healthful seafood.

Advisory Response Literature

Several case studies have documented that public health and safety information campaigns can significantly affect household consumption behavior. Perhaps the best-known general example is information campaigns on the hazards of smoking (Hamilton 1972, Warner 1989). Jin and Leslie (2003) found that mandatory restaurant hygiene grade cards altered diners' choices and improved health outcomes. Variyam and Cawley (2006) found that updated nutrition labels reduced women's body weight and obesity probabilities. Schlenker and Villas-Boas (2009) discovered a significant reduction in beef sales after health warnings about mad cow disease. The behavioral impacts of cholesterol information have been widely studied. For example, Brown and Shrader (1990) demonstrated reduction in egg demand associated with media coverage of cholesterol risks. Studies by Chern et al (1995), for the case of fats and oils, and by Kinnucan et al (1997), for the case of meat, also found that cholesterol information caused significant reduction in foods with higher cholesterol.

Previous research on the 2001 FDA fish advisory for store-bought fish, the subject of the present paper, found that it reduced target consumers' consumption by 15 to 30 percent. Shimshack et al. (2007) analyzed the determinants of household responses to the advisory using repeated cross-sections of Consumer Expenditure Survey purchase data for aggregate canned fish and other commodities. Potential determinants included health consciousness (proxied by vegetable and tobacco purchases), information access (proxied by newspaper purchases), and demographics such as education. They found newspaper readership and education were both strongly associated with a post-advisory reduction in consumption, suggesting that access and information processing were key

drivers of consumer response. The study showed that while there was a large average response, there was considerable predictable variability in which consumers responded. Results could be used to help target outreach and information campaigns more effectively. Similarly, 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. They did find a post-advisory reduction, though without a control group it is difficult to know to what extent this reduction was due to confounding factors. Neither the Oken et al. (2003) or the Shimshack et al. (2007) studies examined mercury intakes, omega-3 consumption, or substitution possibilities and tradeoffs among fish species.

In short, the related empirical information campaign literature indicates that we should expect the FDA advisory to broadly affect household behavior, at least in the sense of reducing average exposure to the source of risk. However, whether the response is sufficiently nuanced to be consistent with improving public health and overall welfare is an open question. The broader advisory literature largely emphasizes cases where an advisory response is likely welfare improving because risk trade-offs are limited. We study the case where risk trade-offs are prominent and unsophisticated information responses may reduce welfare.¹ Improving welfare through an information campaign requires a judicious and measured response from consumers. And we might not expect nuanced responses. There is evidence that people may under-respond. For example, May and Burger (1996) surveyed local fishers and crabbers after a New Jersey consumption

¹ Graham and Wiener (1995) review the literature on risk vs. risk trade-offs. They note that risk decisions may produce unintended consequences and may increase net risk. The reviewed literature, however, emphasizes cases where governments or experts are the decision maker.

advisory and found that many fishers reported that they distrusted government information. On the other hand, Viscusi (1997) found that consumers tend to have an alarmist over-reaction to negative information, placing irrationally high weight on the most pessimistic sources of information. Smith et al. (1988) showed that on average consumers reacted much more strongly to negative news about milk contamination in Hawaii than to positive news that the problem was solved.

While we examine the seafood case study, our results may apply to other instances where consumers make risk-risk tradeoffs in the presence of public information. Everyday examples of such risk-risk tradeoffs for consumers abound in the news. Concern about pesticides causes some to consume fewer vegetables. Concern with tap-water drinking safety causes some to drink more soda. Concern over mildly excess body weight drives some to try fad diets. Concern over oxidants leads some to supplement with high doses of Vitamin A.

3. Data

Household-Based Scanner Data

Our rich household-level panel data permit a fine-grained analysis of the potential substitution patterns underlying advisory-induced risk-benefit trade-offs. We analyze data from Information Resources, Inc. (IRI)'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.

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 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 is a balanced panel containing 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. We retain 70 percent of households, which is approximately the proportion of households in the 2010 Census with less than three adults and less than three children. 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).²

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 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 information, omega-3 information, and a relative intake measure representing mercury concentration divided by omega-3 concentration.

Once we obtain weight, mercury, and omega-3 quantities for each purchased product, we sum these data to reflect per-capita quantities for each household/year combination. We calculate totals over full years to address seasonality. Table 2 presents summary statistics for annual per-capita fish consumption, mercury intakes, and omega-3 intakes. Since our ensuing analysis evaluates differences in behavior between an at-risk treatment group targeted by the advisory and a quasi-control group not targeted by the advisory, we present summary statistics by group. Note that fish consumption summary statistics include many households that consume no fish. Our sample mean of approximately 5.9 pounds of per-capita in-home packaged seafood consumption for 2002 is approximately 38 percent of the 2002 National Marine Fisheries Service (NMFS) estimate of total per-capita seafood consumption (in home and away from home,

²Consistent 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.

packaged and random weight). If we scale the consumption summary statistics accordingly (multiply by 1/.38), we find that approximately 5.4 percent of sample households consumed more than the 12 ounce per week FDA threshold prior to the advisory.

In 2000, at-risk households consumed approximately 22 percent less seafood by weight, 28 percent less mercury, and 37 percent fewer omega-3 fatty acids than non-target households, on average. The species composition of consumption, however, was relatively similar across the two groups. Prior to the advisory, the at-risk group's mean proportion of fish with relatively high levels of mercury and relatively low levels of omega-3s was 46.5 percent while the non-target group's proportion was 50.8 percent.³

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. Variations in fish stocks, the cost of advertising, or other typical demand or supply shifters may result in exogenous shocks to consumption determinants like prices and substitute prices.

The cleanest way to isolate the causal effects of the advisory, accounting for confounding factors, would be to examine differential responses between randomly

³ For this calculation, we define those species with mercury to omega intake ratios above 0.4 as having relatively high levels of mercury and relatively low levels of omega-3s. Alternative definitions also suggest that target and non-target groups consume similar fish compositions prior to the advisory.

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 determine the effect of the advisory on at-risk households. As previously noted, our treatment or quasi-experimental group is households with pregnant women, nursing children, or children under 6.⁴ 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 by the advisory.⁵

Some notation is helpful in presenting the estimators. Individuals in two groups $g \in [a, b]$ experience outcomes y in two periods $t \in [1, 2]$. Group a is the control group, and group b is the at-risk treatment group. Time 1 is the pre-treatment time period, and time 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 households with pregnant women and/or young children in the post-advisory period.

The traditional approach to identify the effect of the treatment on the treated is the difference-in-differences (DID) model. The core idea is that the mean of the outcomes for treatment group (b) would experience the same change from time 1 to time 2 as the mean of the outcomes for the control group (a) in the absence of the treatment. The simplest DID estimate is then $(\bar{y}_{b2} - \bar{y}_{b1}) - (\bar{y}_{a2} - \bar{y}_{a1})$ where \bar{y}_{gt} is the mean of the outcome y for

⁴ The advisory mentions “young children” without specifying ages. IRI does not provide children’s ages in years, but by broad age categories. The youngest category is 0-5 years of age. We therefore define the treatment group as households with pregnant women and children under six. This categorization is broadly consistent with risk assessments for methylmercury. See, for example, Carrington and Bolger (2002).

⁵ 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.

group g in time t . This basic approach can be slightly generalized to allow for demographic controls by casting it as a regression:

$$y = \alpha + \delta t_2 + \gamma g_b + \beta t_2 g_b + \pi X + \varepsilon, \quad (4.1)$$

where y continues to represent outcomes, t_2 is a dummy variable equal to one for the post-advisory period, g_b is a dummy variable equal to one for the treatment group, X represents a vector of demographic control variables including occupation, race, age, education, and income, and ε represents the standard idiosyncratic disturbance term. The coefficient of interest, β , represents the incremental effect of the treatment on the treatment group. Without any demographic controls, β would be identical to mechanically calculating the difference-in-differences of group-time means (\bar{y}_{gt}) as illustrated above.

The traditional DID approach to isolating causal impacts is familiar and transparent, but the identified average treatment effect may obscure important treatment effects at other points along the outcome distributions. Further, the DID model is subject to several well-known difficulties including scale-sensitivity and the possibility of negative predicted consumption. Our main analysis therefore implements Athey and Imbens's (2006) changes-in-changes (CIC) estimator. Changes-in-changes is a non-parametric generalization of the traditional difference-in-differences method, but instead of identifying an average treatment effect alone it identifies the entire distribution of treatment effects.

The intuition behind CIC is relatively uncomplicated, and it parallels DID. The core idea is 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). Let F_{gt} be the cumulative distribution function of outcomes y for group g in period t . Athey and Imbens (2006) show that the predicted counter-factual distribution for the treatment group (b) in the post-advisory period (2), in the absence of the treatment can be represented by:

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

The CIC 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:

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

Athey and Imbens (2006) prove (4.2) and (4.3) based on a simple stylized model. The CIC model specifies that the outcome y for any individual *in the absence of treatment* depends only on an index of unobserved characteristics and preferences u and time t , which allows for time variant confounding factors. Formally,

$$y = h(u, t). \quad (4.4)$$

In our context, one might think of u as capturing preferences about risk and think of h as a function that translates preferences into behavior conditional on information. In this case, (4.4) can be interpreted as the natural restriction that the same preferences lead to the same behavior, absent a treatment. The translation may change for the treatment group after the advisory is announced, however, because the advisory embeds new information about risk.

The CIC model further assumes that h is a strictly increasing function of u . Monotonicity of h in u imposes a natural ordering that allows an interpretation of higher

u as more intense preferences. 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. However, while the realization of u for any particular individual may change over time, an identifying assumption of the CIC model is that the distribution of u within a group is unchanged over time. Without this assumption, the model would have no refutable implications, since one can always ascribe changing behavior to arbitrarily changing preferences (Stigler and Becker 1977).⁶

Our changes-in-changes approach does not model prices directly, instead it treats prices as confounding factors. We “sweep out” price effects for the treatment group using observed consumption changes in a control group that faces the same movements in prices and other confounders over time. The underlying intuition is that a treatment group member with latent variable u would experience the same changes over time as a control group member with the same latent variable u in the absence of the intervention. This reduced form approach is useful in our context because it would be impractical to estimate a structural demand model accounting for prices of thousands of products and dozens of species. Further, changes-in-changes simultaneously picks up other confounders like advertising.

5. Results

Did at-risk households reduce mercury exposure?

Columns 1 and 2 of Table 3 present the 2000 vs. 2002 differences-in-differences estimate of the advisory impact for at-risk households’ total per-capita mercury intakes

⁶ A final technical assumption of the CIC model is that the support of the control group distribution of u encompasses the support of the treatment group distribution of u . Since the mapping from u to y is strictly monotone, we can confirm this assumption for all applications by confirming that the support of the control group distribution of y encompasses the support of the treatment group distribution of y .

from fish and shellfish.⁷ Figure 1 presents the corresponding changes-in-changes estimates. The table and the figure indicate that, relative to the no-advisory baseline predicted by the DID and CIC assumptions, the advisory induced a broad-based decline in per capita mercury consumption for the treatment group. The DID coefficient of -47.1 is significant at the 5 percent level, and represents a 20.7 percent decline in target consumers' mercury exposure relative to their predicted counterfactual consumption. The mean of the CIC estimates represents a 17.1 percent decline in target consumers' mercury exposure, with a 90 percent bootstrap confidence interval of [3.7%, 32.1%].⁸ We see a particularly strong decline at the upper tail of the per-capita mercury distribution. If there were no risk trade-offs from reduced fish consumption, these results would be promising for public health.

Did at-risk households change omega-3 intakes?

Columns 3 and 4 of Table 3 present the 2000 vs. 2002 differences-in-differences estimate of the advisory impact for at-risk households' total per-capita omega-3 intakes from fish and shellfish. Figure 2 presents the corresponding changes-in-changes estimates. The table and the figure indicate that, relative to the no-advisory baseline predicted by the DID and CIC assumptions, the advisory induced a broad-based decline in per capita DHA and EPA omega-3 consumption for the treatment group. The DID coefficient of -3.07 is significant at the 5 percent level, and represents a 27.6 percent decline in target consumers' omega-3 intakes relative to their predicted counterfactual consumption. The mean of the CIC estimates represents a 21.4 percent decline in target

⁷ 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.

⁸ 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.

consumers' omega-3 intakes, with a 90 percent bootstrap confidence interval of [10.2%, 34.5%]. We 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.⁹

How did at-risk households reduce fish consumption?

So far, DID and CIC results 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. Columns 5 and 6 of Table 3 present the 2000 vs. 2002 differences-in-differences estimate of the advisory impact for at-risk households' total per-capita fish and shellfish consumption. Figure 3 presents the corresponding changes-in-changes estimates. The table and the figure indicate that, indeed, the advisory induced a decline in aggregate per capita fish consumption for the treatment group. The DID coefficient of -1.26 is significant at the 5 percent level, and represents a 23.7 percent decline in target consumers' aggregate fish consumption. The mean of the CIC estimates represents a 21.1 percent decline in at-risk consumers' aggregate fish consumption, with a 90 percent confidence interval of [11.0%, 34.2%].

Figure 3 reveals that the per-capita decline in fish and shellfish consumption occurs everywhere along distribution, including the upper tail. We find an approximately 50 percent decline in the number of target consumers consuming above the 12 ounce per person per week FDA threshold. However, we also find that the decline in fish and shellfish consumption includes the lower tail of the distribution. We find a nearly 60 percent *increase* in the number of target consumers with no significant fish and shellfish

⁹ 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.¹⁰

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

Table 4 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. DID estimates are broadly similar to CIC mean estimates. 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 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.

Does response vary by education level?

The two key components to FDA recommendations were that consumers should reduce consumption if they exceed the recommended limit and that consumers should substitute into more healthful fish. Aggregate results so far do not show the latter part of this pattern. However, since more educated households may be better equipped to

¹⁰ As noted previously, we scale our in-home packaged totals by 1/0.38 to estimate total per capita seafood consumption (in home and away from home, packaged and random weight). Following the epidemiological literature, no significant consumption is defined as less than one 100g serving per person per month.

understand and appropriately implement the advisory language, we repeat the previous analyses disaggregated by education level. We consider an educated group consisting of households with at least one college educated member and a less educated group consisting of households with no college educated members.¹¹

Figure 4 presents the estimates of the advisory impact broken down by education level and Table 5 presents the corresponding CIC mean point estimates and confidence intervals. DID estimates are broadly similar to CIC mean estimates. The upper-left panel in Figure 4 shows a significant decrease in mercury intakes for educated households, especially at the upper tail, with a 27.8 percent mean decline. However, the upper-right panel shows a corresponding decrease in omega-3 intakes for these households, with a 22.9 percent decline. The lower-left panel shows a weak and statistically insignificant decrease in mercury intakes for the less educated households, and we find particularly little evidence for a reduction in the upper tail. On average, we find a trivial 0.003 percent mean decline in less educated consumers' mercury intakes. In contrast, the lower-right panel shows a broad and statistically significant decline in omega-3 intakes for the less educated group, including the lower tail, with a 19.6 percent decline.

Educated people had an overall reaction that is broadly consistent with the advisory recommendation to limit consumption at the upper tail of the distribution, and we see this result in the lower left panel of Figure 4. However, the middle left panel shows that their response still resulted in omega-3 losses that in principle could have been avoided with sensible substitution between species. For less educated households, we

¹¹ On average, less educated households consume slightly more seafood than more educated households. For example, in 2000, educated at-risk households consumed 3.98 pounds per person per year while less educated at-risk households consumed 4.62 pounds per person per year. Educated non-target households consumed 5.17 pounds per person per year while less educated at-risk households consumed 5.64 pounds per person per year.

find no responses that were consistent with the advisory language in either dimension. We find no strong evidence that less educated at-risk households reduced their mercury intakes or substituted between species sensibly. However, we do find evidence that their omega-3 intake was reduced, even at the lower tail.

6. Robustness

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 demographic controls?

Even numbered columns in Table 3 show that difference-in-differences regression results are not sensitive to inclusion of demographic variables in the model. Estimated

coefficients on time, treatment, and the key DID coefficient are largely unaffected by controls for occupation, race, age, education, or income. Athey and Imbens show that demographic variables can also be incorporated into the CIC approach. Results for this procedure were qualitatively similar to the main CIC results. Recall that our presented CIC mean declines for per capita mercury, omega-3s, and fish consumption are 17.1, 21.4, and 21.1 percent, respectively. The demographic-adjusted CIC means are 18.8, 30.2, and 23.2 percent, respectively, and all remain statistically different from zero.

How do basic response magnitudes compare to previous studies?

Relative to no-advisory baselines predicted by the CIC and DID assumptions, respectively, the advisory induced a 21.1 to 23.7 percent average decline in per capita fish and shellfish consumption among households with pregnant women and/or young children. Shimshack, Ward, and Beatty (2007) reported the same advisory resulted in a 21.8 percent net drop in canned fish expenditures among households with young children. A survey by Oken et al. (2003) found that pregnant women self-reported reductions of 27.6 percent for canned tuna and 16.9 for a composite of various fish. Those studies do not estimate advisory impacts on mercury intakes, omega-3 intakes, or fish substitution, but their results do support the plausibility of our empirical estimates.

Are the data sources sufficiently comprehensive?

A limitation of this study is that we do not observe all potential sources of mercury or omega-3s. We are unaware of any systematic data on random weight grocery seafood purchases or out of home seafood consumption for our sample period. Consequently, we can only analyze the evidence for in-home packaged fish consumption. We would be surprised to see a completely different substitution pattern for packaged in-

home and other fish consumption.¹² However, when extrapolating from our dataset to total consumption, we used a fixed proportionality factor based on available data. This extrapolation should be interpreted with caution, since dining out may be correlated with factors that predict advisory response. Similarly, we do not have data on omega-3 supplements. 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). The market growth between sample years 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 data on omega-3 supplementation would significantly alter our conclusions.

Are seafood prices endogenous?

A possible concern is that the advisory itself may have influenced aggregate prices. We assume that changes in the world price for seafood are exogenous, unaffected by the FDA mercury advisory. We believe 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

¹² Kuchler and Tegene (2006) find no systematic difference between fixed weight and random weight beef responses to BSE ('mad cow') announcements.

(Asche and Bjorndal 1999). Given these quantities and elasticities, any endogenous advisory induced price drop would be likely less than 0.1% and insignificant in practice.

A related concern is that the advisory may have caused manufacturers to tailor price discounts and promotions to distinct populations that were differentially affected by the advisory. However, we test this conjecture using prices, coupons, and in-store promotions from our IRI dataset, and we find no evidence of this behavior. We select a common market basket based on the purchases of the at-risk group in 2000. We then apply Laspeyres-type indexes to see if target and non-target populations paid different prices, exhibited differential coupon usage, or encountered more in-store promotions. We find that the expenditures required for the target group to purchase the market basket were within 1% of the expenditures required for the non-target group to purchase the market basket for both 2000 and 2002. It follows that expenditures have changed in approximately the same way over time for both groups. Relative trends for coupons and promotions were also similar across the two groups. Finally, we repeat the same analyses for high-mercury fish and still find no significant differences.¹³

7. Discussion

The existing risk assessment literature primarily relies upon assumed or hypothesized advisory response and substitution scenarios. This study fills a key gap by providing estimates of the observed impact of the advisory on mercury and omega-3 intakes. Our empirical findings show that mercury intakes fell 17.1 percent in response to

¹³ By a Laspeyres-type index, we mean $\sum_i p_{i2}q_{i1} / \sum_i p_{i1}q_{i1}$, where p_{it} is measured by the sample average of prices paid (or 0/1 coupon usage or 0/1 promotions) for product i in time t by a particular group. The 2002 price index (using 2000 as a baseline) for the target group was 0.793 and the 2002 price index (using 2000 as a baseline) for the non-target group was 0.784. The 2002 Laspeyres-type index for coupon use was 0.403 for the target group and 0.409 for the non-target group. The 2002 Laspeyres-type index for the promotions was 0.771 for the target group and 0.778 for the non-target group.

the advisory on average, and this reduction was concentrated among college educated, high consuming households. It appears that the FDA mercury advisory was successful, for at least some groups, in reducing overall mercury exposure. However, at-risk consumers' omega-3 intakes from this food source also fell 21.4 percent, and this reduction occurred across the board including the low end of the omega-3 distribution. It appears that the recommendation to continue consuming healthful levels of seafood and to substitute towards lower mercury fish was not heeded on average.

What can we say about welfare? In order to provide a guide to the economic significance of our findings, we combine our empirical behavioral estimates with the epidemiological risk analysis of Cohen et al. (2005), which is based on a meta-analysis of mercury, omega-3, and fish consumption impacts on childhood IQ, adult heart disease mortality, and adult stroke mortality. We monetize impacts using standard Environmental Protection Agency benefit transfer figures of \$13,084 (2007 USD) per IQ point and \$7.52 million (2007 USD) for the value of statistical life (USEPA 2000a, EPA2000b).¹⁴ Table 6 presents the estimated benefits from the advisory response by category of health impact. Our central estimate of net benefits is *negative* \$30 per at-risk household.¹⁵ That is, our best estimates suggest that the advisory harmed net welfare, on average, even without considering potential utility losses due to tastes for fish and shellfish. At a minimum, we find no clear evidence for net benefits from the large-scale public advisory.

¹⁴ \$13084 = \$8346 1990USD in 2007USD and \$7.52 million = \$4.8 million 1990USD in 2007USD.

¹⁵ Details of all calculations are in the appendix. We are not epidemiologists, so we simply repeat the calculations in the appendix of the Cohen et al. risk analysis, but using our empirical estimates as inputs. This exercise is clearly imperfect, but similar calculations using *hypothesized* data as inputs are frequently cited to influence public policy. See, for example, U.S. National Academies (2005).

While the advisory likely resulted in a modest decrease in average welfare, the FDA was in a sense successful, because it reduced the number of at-risk households consuming above the FDA threshold by approximately 50 percent. Indeed, this may have the Agency's primary goal. However, reducing fish consumption below a threshold is a coarse goal, not necessarily closely linked to overall public health and welfare. Cohen et al. (2005) also present an idealized substitution scenario, in which 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 counterfactual idealized scenario, the point estimate of net benefits would be *positive* \$587 per at risk household, as indicated in Table 7. In short, while we find no clear evidence for net benefits from actual advisory response, a more nuanced substitution response would likely have generated large net benefits.

What else can be said about the 2001 FDA advisory? The advisory reduced risks from mercury. While the advisory was short at less than 1 page, it did have simple and direct language regarding mercury risks. The advisory specifically counseled pregnant women, nursing mothers, and young children to avoid larger fish, including four specific species. The advisory suggested that consuming more than 12 ounces of fish per week may be unsafe for at-risk individuals. An additional possibility is that consumers simply reacted strongly to negative information, a likelihood that Smith et al. (1988) and Viscusi (1997) have demonstrated in other contexts. The 2001 seafood advisory failed to trigger nuanced substitution that would have minimized risk tradeoffs for all households and protected public health for those households not initially at-risk. These failures may have

resulted from coarse information and broad behavioral guidance. The advisory did not state that there may be risks to reducing overall fish and shellfish consumption. The advisory recommended that consumers select “a variety of other kinds of fish,” without noting what particular species would be preferable. An additional possibility is that consumers’ cognitive biases prevent nuanced responses in the presence of risk-risk trade-offs.

What have we learned about health information policies more broadly? Notably, great care is required when using advisories and information campaigns to address cases where risk-risk trade-offs are important. A growing literature demonstrates that advisories and information campaigns influence household consumption behavior, but behavioral changes may not enhance welfare in cases where consumers must carefully balance competing risks. At a minimum, our results suggest that public agencies considering information policies must consider risk systematically, not one risk at a time. In cases with the potential for risk-risk trade-offs, organizations should take into account emerging strategies for communicating comparative risks (see, for example Hoffstetter et al. 2002, Connelly and Knuth 1998, and Waters et al. 2006).

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Table 1. Mercury and Omega-3 Concentrations by Species

Seafood	Mercury (in µg/g)	Omega-3 (in g/100g)	Relative Intake (mercury/omega-3)	Original Hg Source	Omega-3 Source
Anchovy	.043	2.05	0.021	NMFS (1978)	USDA (2006)
Catfish	.049	0.17	0.288	FDA 1990-2004	Ackman (2000)
Clams	.001*	0.35	0.003	FDA 1990-2002	Ackman (2000)
Cod	.095	0.31	0.306	FDA 1990-2004	Ackman (2000)
Crab	.060	0.36	0.167	FDA 1990-2004	Ackman (2000)
Crayfish	.033	0.17	0.194	FDA 2002-2004	USDA (2006)
Croaker	.180	0.24	0.750	FDA 1990-2003	Ackman (2000)
Eel	.213	0.96	0.222	EPA (1997)	Ackman (2000)
Flounder	.045	0.12	0.375	FDA 1990-2004	Ackman (2000)
Grouper	.465	0.30	1.550	FDA 2002-2004	Ackman (2000)
Haddock	.031	0.21	0.148	FDA 1990-2002	Ackman (2000)
Halibut	.252	0.38	0.663	FDA 1990-2004	Ackman (2000)
Herring	.044	2.34	0.019	NMFS (1978)	Ackman (2000)
Jack Mackerel	.267	1.23	0.217	EPA (1997)	USDA (2006)
Lobster	.240	0.36	0.667	FDA 1991-2004	Ackman (2000)
Mackerel	.069	2.29	0.030	NMFS (1978)	Ackman (2000)
Mahi	.144	0.22	0.655	EPA (1997)	Ackman (2000)
Mullet	.046	0.33	0.139	NMFS (1978)	USDA (2006)
Mussel	.030	0.79	0.038	Sunderland (2007)	USDA (2006)
Octopus	.029	0.16	0.181	EPA (1997)	USDA (2006)
Oyster	.013	0.19	0.068	FDA 1990-2004	Ackman (2000)
Perch	.140	0.32	0.438	FDA 1990-2002	USDA (2006)
Pike	.310	0.14	2.214	EPA (1997)	USDA (2006)
Pollack	.041	0.26	0.158	FDA 1990-2004	Ackman (2000)
Redfish	.001*	0.21	0.005	FDA 1990-2002	Ackman (2000)
Roughy	.554	0.04	13.85	FDA 1990-2004	USDA (2006)
Salmon, atlantic	.014	2.00	0.007	FDA 1990-2002	USDA (2006)
Salmon, canned	.001*	1.35	0.001	FDA 1990-2002	USDA (2006)
Salmon, chinook	.014	1.74	0.008	FDA 1990-2002	USDA (2006)
Salmon, chum	.014	0.81	0.017	FDA 1990-2002	USDA (2006)
Salmon, pink	.014	1.29	0.011	FDA 1990-2002	USDA (2006)
Salmon, sockeye	.014	1.23	0.011	FDA 1990-2002	USDA (2006)
Salmon, unknown	.014	1.46	0.010	FDA 1990-2002	USDA (2006)
Sardines	.016	0.98	0.016	FDA 2002-2004	USDA (2006)
Scallop	.050	0.37	0.135	NMFS (1978)	Ackman (2000)
Seabass	.219	0.15	1.460	FDA 1990-2004	Ackman (2000)
Shrimp	.001*	0.44	0.002	FDA 1990-2002	Ackman (2000)
Smelt	.108	0.89	0.121	FDA 1990-2002	USDA (2006)
Snapper	.189	0.26	0.727	FDA 2002-2004	Ackman (2000)
Sole	.045	0.19	0.237	FDA 1990-2004	Ackman (2000)
Squid	.070	0.54	0.130	NMFS (1978)	USDA (2006)
Sturgeon	.235	1.97	0.119	EPA (1997)	Ackman (2000)
Swordfish	.976	0.58	1.683	FDA 1990-2004	Ackman (2000)
Tilapia	.010	0.14	0.071	FDA 1990-2002	USDA (2006)
Trout	.072	0.62	0.116	FDA 2002-2004	Ackman (2000)
Tuna	.383	0.30	1.277	FDA 2002-2004	USDA (2006)
Tuna, canned	.118	0.20	0.590	FDA 2002-2004	USDA (2006)
Tuna, canned light	.118	0.20	0.590	FDA 2002-2004	USDA (2006)
Tuna, canned light oil	.118	0.13	0.908	FDA 2002-2004	USDA (2006)
Tuna, canned light water	.118	0.27	0.437	FDA 2002-2004	USDA (2006)
Tuna, canned oil	.118	0.13	0.908	FDA 2002-2004	USDA (2006)
Tuna, canned water	.118	0.27	0.437	FDA 2002-2004	USDA (2006)
Tuna, canned white	.353	0.56	0.630	FDA 2002-2004	USDA (2006)
Tuna, canned white oil	.353	0.25	1.412	FDA 2002-2004	USDA (2006)
Tuna, canned white water	.353	0.86	0.410	FDA 2002-2004	USDA (2006)
Tuna, white (albacore)	.357	0.81	0.441	FDA 2002-2004	Ackman (2000)
Turbot	.100	0.62	0.161	EPA (1997)	Ackman (2000)
Whitefish	.069	0.21	0.329	FDA 2002-2004	USDA (2006)
Whiting	.001*	0.24	0.004	FDA 1990-2002	Ackman (2000)

Notes: A * indicates that mercury concentrations were below detection levels. Table 1 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/~fr/sea-mehg.html.

Table 2. Summary Statistics

	2000	2002
<u>Per Capita Fish Consumption (lbs.)</u>		
Target	4.25	4.06
Non-Target	5.46	6.53
<u>Per Capita Mercury Intake (µg/g)</u>		
Target	186.8	180.7
Non-Target	258.3	299.3
<u>Per Capita Omega-3 Intake (g/100g)</u>		
Target	8.13	8.04
Non-Target	12.84	15.82

Table 3. Difference-in-Differences Results

	[1] Mercury	[2] Mercury	[3] Omega	[4] Omega	[5] Fish	[6] Fish
Constant	258.30* (7.07)	180.15* (26.52)	12.84* (0.362)	7.65* (1.16)	5.46* (0.115)	5.13* (0.416)
Targeted HH? “Treatment”	-71.53* (14.90)	-42.46* (16.21)	-4.71* (0.682)	-0.58 (0.694)	-1.21* (0.293)	-0.38 (0.320)
Post Advisory? “Time”	41.04* (9.57)	41.72* (9.55)	2.99* (0.490)	2.92* (0.488)	1.07* (0.158)	1.07* (0.158)
Treatment * Time “DID”	-47.12* (19.85)	-43.98* (19.92)	-3.07* (0.863)	-2.96* (0.867)	-1.26* (0.351)	-1.19* (0.349)
Occupation = 0/1 White Collar?	-	18.33 (11.12)	-	-1.35* (0.528)	-	-0.20 (0.173)
Race = 0/1 White?	-	-7.87 (14.63)	-	-2.66* (0.780)	-	-1.09* (0.268)
Age? 1-4 Categorical Variable	-	21.42* (5.17)	-	2.32* (0.217)	-	0.44* (0.085)
Education = 0/1 College?	-	15.68 (11.06)	-	1.26* (0.524)	-	-0.06 (0.164)
Income = 0/1 Income > \$65K?	-	-14.94 (10.43)	-	-0.45 (0.532)	-	-0.42* (0.169)

Notes: Table 3 presents the DID regression results with and without demographic controls for mercury, omega-3's, and fish consumption. The row displaying the coefficient of primary interest, the DID treatment estimate, is presented in bold. Robust standard errors appear in parentheses. A * superscript indicates significance at the 5 percent level or less. Our dataset does not contain continuous demographics, so we model demographics using categorical variables.

Table 4. 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%]

**Table 5. Changes-in-Changes Percent Impact on Mercury and Omega-3 Intakes:
By Education Status**

At-Risk Demographic	Mercury Estimated Percent Change	Mercury 90% CI	Omega-3 Estimated Percent Change	Omega-3 90% CI
All	-17.1%	[-32.1%, -3.7%]	-21.4%	[-34.5%, -10.2%]
College Educated	-27.8%	[-49.5%, -11.1%]	-22.9%	[-42.6%, -7.8%]
Less Educated	-0.00%	[-23.1%, +18.0%]	-19.6%	[-36.3%, -3.7%]

Table 6. 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
Mercury exposure & IQ	-0.00033 IQ pts per ug/year total mercury	-37.2 ug/year	+0.12 IQ pts/child	+\$181
Omega-3 intake & IQ	+0.0036 IQ pts per g/year total DHA intake	-2.19 g/year	-0.008 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	+0.50 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	+0.13 CHD deaths/100000 adults	-\$19
Net health effects per at-risk household				-\$30

Notes: Health effects and risk relationships are taken from Cohen et al. (2005)'s risk assessment model. Empirical changes are derived from this paper's central estimates. Following Cohen et al. (2005), no significant fish consumption is defined as <1 serving/month.

Table 7. 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
Mercury exposure & IQ	-0.00033 IQ pts per ug/year total hg	-102.8 ug/year	+0.034 IQ pts/child	+\$512
Omega-3 intake & IQ	+0.0036 IQ pts per g/year total DHA intake	+1.46 g/year	+0.005 IQ pts/child	+\$75
Net health effects per at-risk household				+\$587

Notes: Health effects and risk relationships are taken from Cohen et al. (2005)'s risk assessment model. Empirical changes are derived by applying Cohen et al. (2005)'s idealized substitution scenarios to the baseline consumption levels in our dataset.

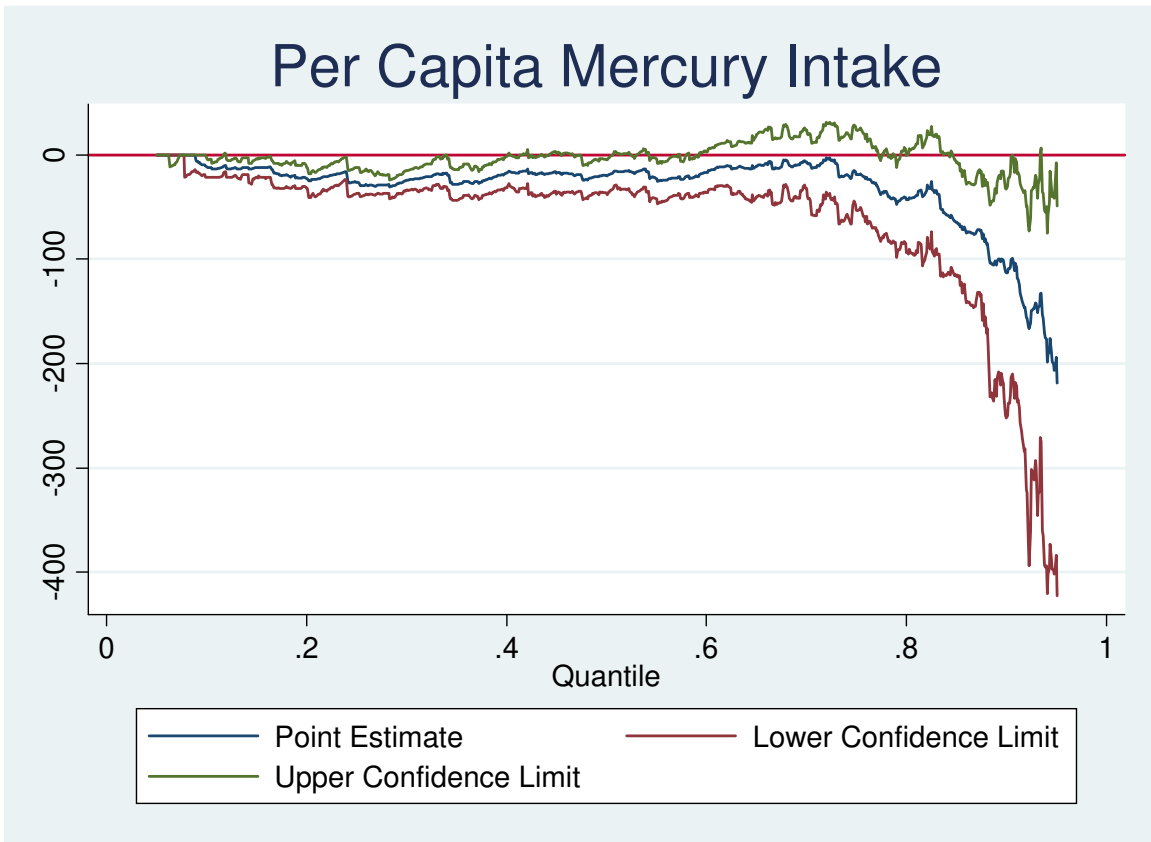


Figure 1. The graph presents the changes-in-changes estimates of the advisory impact on the per capita mercury intake of the at-risk group. For each quantile along the distribution of at-risk consumers' mercury intake distribution, the point estimate is the advisory-induced change in micrograms of mercury 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].

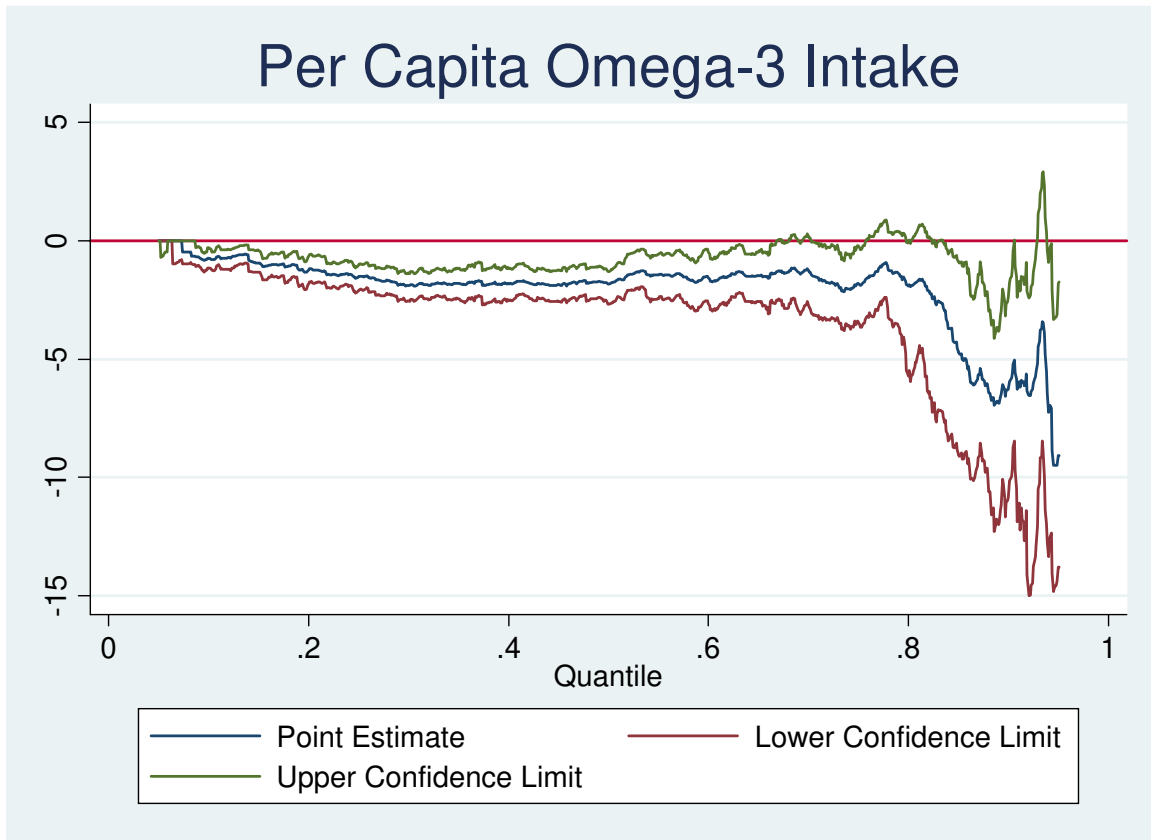


Figure 2. The graph presents the changes-in-changes estimates of the advisory impact on the per capita omega-3 (DHA+EPA) intake of the at-risk group. For each quantile along the distribution of at-risk consumers' omega-3 intake distribution, the point estimate is the advisory-induced change in grams 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].

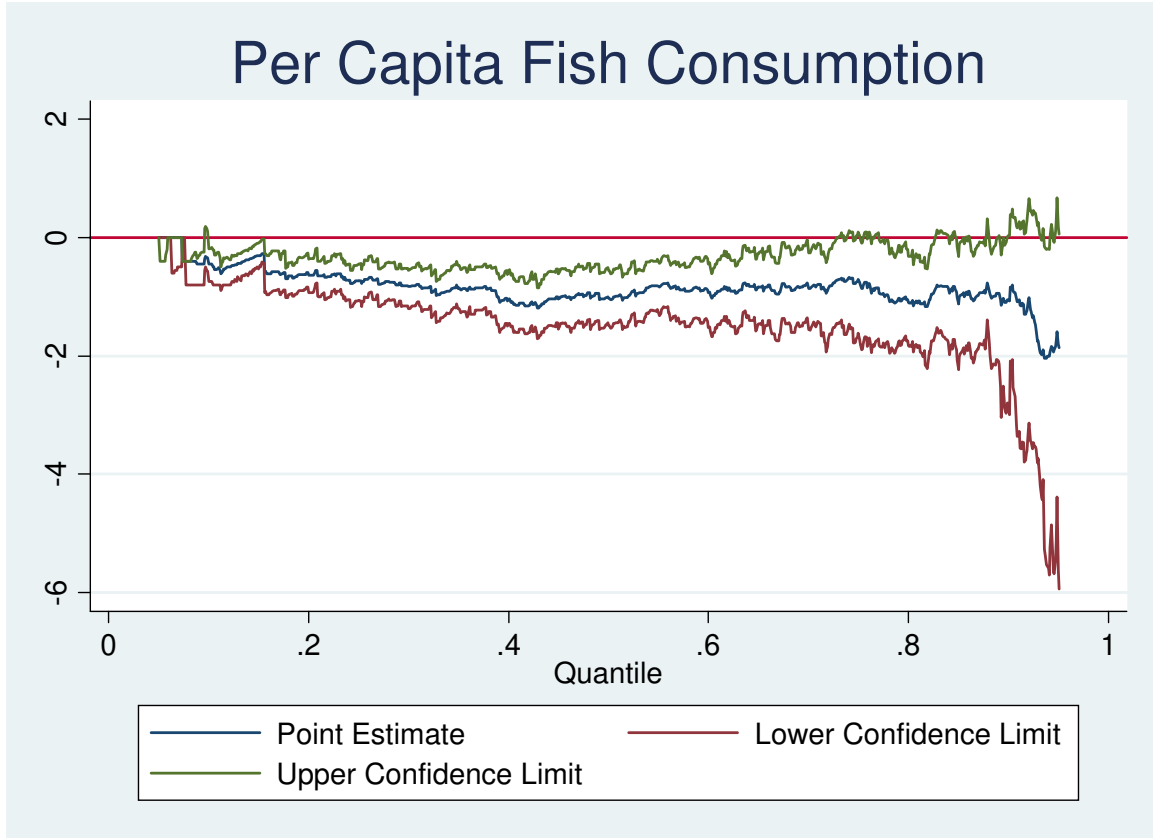


Figure 3. The graph presents the changes-in-changes estimates of the advisory impact on the per capita fish consumption of the at-risk group. For each quantile along the distribution of at-risk consumers' fish weight distribution, the point estimate is the advisory-induced change in pounds of fish consumption.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

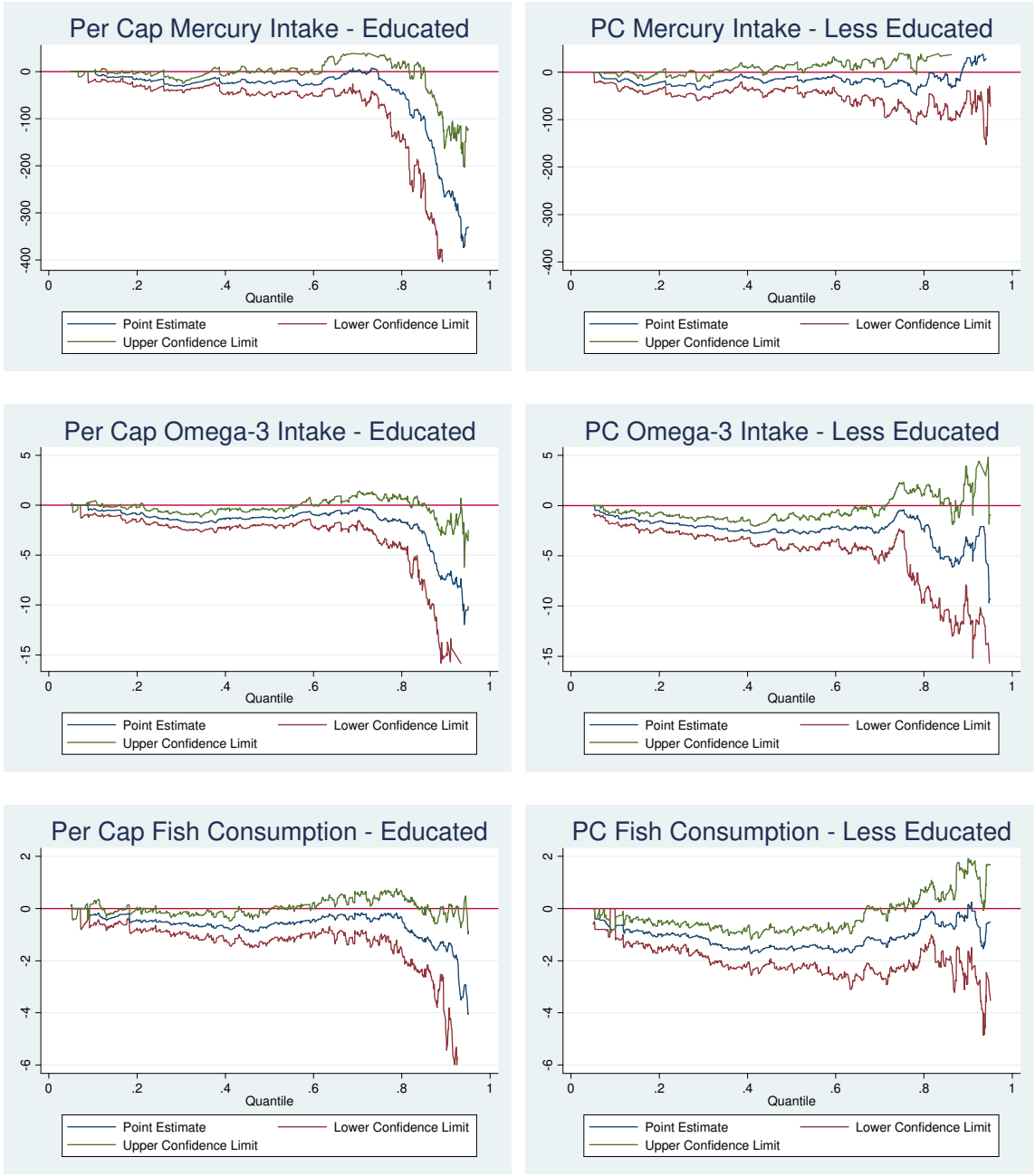


Figure 4. These graphs present changes-in-changes estimates of the advisory impact on per capita mercury intakes, per capita omega-3 (DHA+EPA) intakes, and per capita fish consumption of the at-risk group by education status. “Educated” households have an adult with a college degree; “less educated” households do not.

Online 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 6, 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 $(0.17 \times 0.7) / 365 \text{ days} = 0.00033$.

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. $0.171 \times 217.8 = 37.2$.

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

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

Table 6, 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 $1.3 / 365 \text{ days} = 0.0036$.

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. $0.214 \times 10.24 = 2.19$.

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). $0.0036 \times 2.19 = 0.008$.

Valuation applies a central IQ estimate of \$13,084 per point to at-risk households that average 1.15 young children per HH. $0.008 \times 13084 \times 1.15 = 120$.

Table 6, 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 6, 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 7, 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 $(0.7 \times 0.17) / 365 \text{ days} = 0.00033$.

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 ($.472 \times 217.8$).

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

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

Table 7, 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 $1.3 / 365 \text{ days} = 0.0036$.

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 ($.143 \times 10.24$).

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). $0.0036 \times 1.46 = 0.005$.

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

Additional Reference:

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