Risk-Risk Tradeoffs in Fish Consumption: Can You Have the Cake and Eat It Too?

CHRISTOPH M. RHEINBERGER [1] & JAMES K. HAMMITT [1, 2]
[1] Toulouse School of Economics (LERNA-INRA); Email: crheinbe@univ-tlse1.fr
[2] Harvard University (Center for Risk Analysis); Email: jkh@harvard.edu

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C.M. Rheinberger [1] and J.K. Hammitt [1,2]

Fish is commonly perceived as a healthy food. It is rich in proteins, vitamins A and D, selenium and iodine, and in long-chain n-3 polyunsaturated fatty acids (PUFAs). Over the past two decades, evidence for the preventive effects of PUFAs on the risks of coronary heart diseases (CHD) and strokes has grown [1-3]. PUFAs have even been found to enhance the neurodevelopment in fetuses and infants [4,5]. However, fish may also be contaminated with toxicants such as methylmercury (MeHg), polychlorinated biphenyls (PCBs), and dioxins. MeHg is well known to have detrimental effects on neurodevelopment [6,7] and may also promote the development of cardiovascular disease (CVD) [8,9]. PCBs and dioxins belong to the so-called endocrine disruptors, which are thought to inhibit the action of natural hormones, alter the normal regulatory function of the immune, nervous, and endocrine systems and may cause different forms of cancer [10].

In most aquatic systems these toxicants are present only in low concentrations, but may accumulate along the food chain and eventually reach potentially harmful concentrations in large predatory fish and marine mammals. In consequence, consumers have to make implicit risk-risk tradeoffs [11,12] when deciding whether, how much and which fish to eat. Generally speaking, consumers cannot increase their intake of PUFAs without increasing their risk of contamination. Yet, a smart species selection strategy may allow keeping the intake of contaminants at a safe level while still benefiting from the positive nutritional effects of fish. In 2001, the U.S. Food and Drug Administration (USFDA) launched a mercury advisory that informed consumers about the potential harms of fish consumption and instructed households with pregnant women and young children to eat no more than 12 ounces (340g) of fish a week and to avoid certain species with high mercury concentrations.

While this advisory had some effects on fish consumption, it is unclear whether the campaign impaired rather than improved public health because many consumers not belonging to the target group of the advisory reduced their consumption as well [13]. In this paper, we aim to shed light on how consumption behavior would have to alter in order to have the most beneficial effects on public health. We pursue a damage function approach that analyzes how changes in current consumption would affect public health in the U.S. population. In particular, we assess a number of alternative consumption scenarios (in this paper we will focus on two) and their effects on public health due to reductions or increases in the population's intake of MeHg and PUFAs using Monte Carlo simulations. We then monetize these health effects relying on benefit transfer functions to value changes in mortality, morbidity, and productivity.

[1] Toulouse School of Economics (LERNA-INRA); Email: crheinbe@univ-tlse1.fr

[2] Harvard University (Center for Risk Analysis); Email: jkh@harvard.edu

Building on previous research on the physical benefits of reducing MeHg exposure [14,15], and the economic value of those physical benefits [16,17], we provide an integrated assessment of the risk-risk tradeoffs inherent to fish consumption behavior of the U.S. population. In contrast to previous studies for the U.S. population [18] and for a population of frequent fish consumers in France [19], we express health outcomes in terms of money rather than in terms of QALYs as this may convey more information to consumers about the options they face [20]. In the next section, we describe the methods and data sources used for the physical impact modeling and the economic valuation of the relevant health endpoints. We then present preliminary results considering only effects of MeHg and PUFAs, but ignoring effects associated with PCBs and dioxins for the time being. We close with some remarks on the sensitivity of the observed health effects toward modeling assumptions and discuss the feasibility and desirability of the studied scenarios.

Methods and Data

Health effects of fish consumption are modeled as "the product of exposure to the active agent (μg/day of MeHg, mg/day of PUFA), and the dose–response relationship for that agent (incremental risk per μg/day of MeHg, reduction in risk per mg/day of PUFA)" [18:326]. Below, we first describe the consumption scenarios analyzed. We then outline the quantification of the impact of changes in fish consumption on MeHg and PUFA intake and describe the dose–response relationships between MeHg, PUFA, and different health endpoints.

Alternative consumption scenarios. In this paper, we limit the analysis to two scenarios that are of health policy relevance: (1) perfect compliance with the USFDA advisory, i.e. women younger than 45 years eat no more than 340g fish/week and no high mercury fish; everyone else does not change their consumption habits; (2) undesirable side effects of the advisory on the amount of fish consumed, i.e. while the target group adheres to the advisory, other consumers reduce their fish consumption as well, say by up to 20%. Overreaction of consumers to warnings is well-known and–as empirical evidence suggests [13]–may countermine the intended reduction in health risks.

Fish consumption model. We extend the fish consumption model developed by Carrington et al. [14,15] to quantify the impact of changes in fish consumption behavior on MeHg and PUFA intake. The model draws on food survey data from the 2007-08 U.S. National Health and Nutrition Examination Survey, NHANES [21], to estimate consumption rates for some 40 types of fish covering more than 95% of the U.S. seafood market supply. (NHANES contains choices over 26 types of fish and aggregates other types into two broad categories, other fish and other shellfish). Carrington et al. [14,15] developed frequency distributions for MeHg concentrations in fish based on USFDA surveillance data [22] and information from the National Marine Fisheries Service [23]. We used an updated version of these data as well as data from the USDA [24] and a meta-database of nutrients in fish [25] to construct frequency distributions of MeHg (in μg/g fresh weight) and PUFA (in mg/g fresh weight) concentrations [Table 1].

These PUFA and MeHg concentration distributions served as input into a Monte Carlo simulation of the market basket available to U.S. consumers. We took 10,000 draws from the concentration distributions for each fish species and multiplied them by observed patterns of consumption by fish type that we sampled randomly from the NHANES consumer survey. This way, we capture both the selection frequency of each type of fish and correlations among fish types consumed. We assumed a triangular distribution function to account for variations in serving size, implying average serving sizes of fish ranging from 10g to 200g with a mode of 50g per meal [26]. By summing up over types of fish, we obtained the monthly MeHg and PUFA intake from fish consumption of each of the 10,000 stylist consumers.

Health effects valuation model. This model quantifies the welfare implications, W, of the health endpoints expected from changes in fish consumption behavior. These health effects can be broadly summarized as the net present value of changes in neurotoxicity, V_N, and the net present value of changes in cardiovascular events, V_C, including fatal and nonfatal heart attacks and strokes. For each of the consumption scenarios including the baseline, we estimate the present value of the discounted streams of health effects generated by reduction, increase or selection of fish consumption.

Both neurotoxicity and cardiovascular effects are multifaceted, and interdependences between PUFA and MeHg intake may confound the impact analysis. In line with earlier research [14,16,18], we use proxies to quantify the most dominant consequences. For neurotoxicity, we focus on unborn children as few infants eat fish in amounts sufficiently large to be harmful. Information on the relationship between maternal PUFA intake during pregnancy and cognitive development in the fetus is limited [5]. Following Cohen et al. [18], we assume that the PUFA to IQ point relationships observed in toddlers can be used to approximate the cognitive benefits of maternal PUFA intake on neurodevelopment. We estimate the present value of future earnings expected from IQ increases (decreases) in an annual birth cohort as:

$$V_N = \Delta E \cdot B, \tag{1}$$

where ΔE denotes the expected gain (loss) in the present value of lifetime earnings L per IQ point gained (lost) and B is the annual number of births in the population. Since IQ changes in this context are rather small, the dose–response relationships between a child's future earnings, IQ, and maternal intake of PUFA (ΔP), docosahexaenoic acid (ΔP_{DHA}), hair MeHg (ΔM_H), blood MeHg (ΔM_B), and MeHg (ΔM) are assumed to be linear with slope parameters η, ι, θ, γ, λ, and β, respectively:

$$\Delta E = \eta \cdot L \cdot \Delta IQ; \Delta IQ = \gamma \cdot \Delta M_H - \iota \cdot \Delta P; \Delta P = \theta \cdot \Delta P_{DHA}; \Delta M_H = \lambda \cdot \Delta M_B; \Delta M_B = \beta \cdot \Delta M,$$

and upon inserting into Eq.(1): $V_N = \eta \cdot L \cdot (\gamma \cdot \lambda \cdot \beta \cdot \Delta M - \iota \cdot \theta \cdot \Delta P) \cdot B.$ (2)

We assume that cardiovascular deaths are prevalent only among individuals aged 40 years or more. The change in CVD death risk is denoted by ΔD_G (deaths/year among individuals of gender G) and the change in nonfatal heart attacks and strokes by ΔH_G (cases/year among individuals of gender

G). The economic value of reducing (increasing) fatal and nonfatal consequences of fish consumption on cardiovascular health is measured in monetary terms as:

$$V_C = \int_0^{\tau} \sum_G N_G \cdot (VSL \cdot \Delta N_G + VHA \cdot \Delta H_G) \exp(-\rho\tau)\, d\tau. \quad (3)$$

Eq.(3) draws on the value of statistical life [27], *VSL* ($/death), and the value per avoided nonfatal heart attack [28], *VHA* ($/case), to value changes in CVD death risk and in the risk of nonfatal heart attacks and strokes. Further, we assume that consumption behavior today affects CVD health only in the future. To motivate this assumption, we refer to studies in ex-smokers suggesting that their risk of heart attacks declines to those of never smokers between two and ten years after quitting smoking [29,30]. Similarly, the risk of heart attacks to new smokers does not rise instantaneously. In Eq. (3), we consider this lag effect, τ (in years), expressing the time until changes in MeHg and PUFA intake have an impact on the risk of heart attacks. Since the length of this lag is inherently uncertain, we allow τ to randomly vary over the cohort and discount the monetized effects at a rate of $\rho = 3\%$.

Changes either in CVD death risk, ΔD_G, or in the risk of nonfatal CVD incidents, ΔH_G, are predicted using a relative risk model:

$$\Delta D_G = D_G \cdot [\omega \cdot (1 - \exp(-\varphi \cdot \mu \cdot \Delta M_H) - (1 - \exp(-\zeta \cdot \Delta P)] \text{ and}$$
$$\Delta H_G = H_G \cdot \omega \cdot [(1 - \exp(-\varphi \cdot (1 - \mu) \cdot \Delta M_H) - (1 - \exp(-\xi \cdot \Delta P)], \quad (4)$$

where D_G and H_G are the current numbers of fatal and nonfatal heart attacks in individuals of gender G, aged 40 years and older (Rice et al. [16] emphasize that current heart attack risk reflects an unobservable baseline combined with the impact of current MeHg exposure levels); μ represents the fraction of heart attacks that are fatal; φ, ζ, and ξ represent the hair mercury to heart attack relationship (fractional change in risk per μg Hg/g hair) and the PUFA to fatal (ζ) and nonfatal (ξ) heart attack relationship (fractional change in risk per mg PUFA), respectively; ΔM_H and ΔP denote changes in the hair mercury concentration and the PUFA intake as studied in a specific consumption scenario. We include a causality parameter, ω, reflecting the limited evidence on a causal association between hair mercury level and total heart attack risk [16] and between PUFA intake and nonfatal heart attacks [3].

Specification of relationships. In Table 2, we provide probabilistic characterizations of the parameters that go into the health effects valuation model. Due to the limited space, we do not give a full justification for the characterization of every parameter but focus on some important features of the model. The reader is referred to [16] for a detailed account of the assumptions underlying the MeHg effects modeling and to [31] for a discussion of the PUFA effects modeling.

The probabilistic characterizations of the MeHg intake to blood parameter, β (μg MeHg/L blood per μg MeHg/day) and the blood to hair mercury parameter, λ (μg Hg/g hair per μg MeHg/L blood), are aggregate population parameters reflecting changes in the equilibrium hair and blood mercury concentrations that would result from changes in the daily MeHg intake. These parameters have been extensively analyzed in toxicokinetic studies and are therefore known with relative certainty.

The hair mercury to IQ parameter, γ (IQ points per μg MeHg/g maternal hair), reflects changes in IQ points in children (as measured by a battery of neurodevelopment tests) that would result from a unit change in maternal hair mercury concentrations during pregnancy. One important point with the available dose–response functions [6,7] is that they are likely confounded with the beneficial effects of the fatty acids in fish. Therefore, Rice et al. [15] propose to adjust the values found in the literature by multiplying the central estimates ($\gamma = 0.18$ in [5] and $\gamma = 0.20$ in [6]) by a factor of 1.5 to offset the likely downward bias from inadequate confounder control. Support for the size of this adjustment is provided by Budtz-Jørgensen et al. [32], who used structural equation modeling to quantify confounding effects of fish intake on the size of effects of MeHg.

There is less, albeit some, uncertainty about the size of the PUFA to IQ parameter, ι (IQ points per 100 mg DHA maternal intake/day). A recent meta-analysis [5] of eight randomized controlled trials on cognitive development in children who had received PUFA supplementation suggests that one specific n3-fatty acid, docosahexaenoic acid (DHA), has significant beneficial impact on neurodevelopment, and that daily intake of 100 mg DHA is expected to increase IQ by 0.13 points.

The IQ to earnings parameter, η (Percentage change in $ per IQ point), seeks to express the monetary benefit of a permanent one point IQ increase in a typical child. We draw on values proposed by Heckman et al. [33] to estimate the impact of a marginal change in IQ on lifetime earnings *L*.

To date, scientific evidence on a causal relationship between MeHg exposure and fatal cardiovascular risks is mixed. Rice et al. [15] reviewed four epidemiological studies that control for confounding effects of PUFA on the risk of heart attacks. They concluded that the strength of the alleged association found in two of the four studies is modest and that the design of these studies does not permit strong evidence on the causality between MeHg exposure and CVD risk. They propose a plausibility parameter, ω, to quantify the belief of a causal relationship between increased MeHg exposure and the risk of heart attacks. Based on their assessment of the epidemiological evidence they assign a subjective probability of 1/3 that the epidemiological associations are causal (i.e., $\omega = 1$) and 2/3 that they are not (i.e., $\omega = 0$). Since a new study [9] supports the notion that increased MeHg exposure promotes CVD risk, we assign a subjective probability of 1/2 to the event that the epidemiological associations are causal.

Hair mercury to heart attack risk parameter, φ (fractional increase in risk per μg Hg/g hair), reflects the relative reduction in the risk of heart attacks. The parameter is based on an epidemiologic study [34] in which confounding effects of fish consumption (i.e. PUFA intake) were limited. Since this study associated MeHg with increases in the risk of both fatal and nonfatal heart attacks, we adjust φ by the heart attack mortality rate, μ (ratio of fatal to all heart attacks). In contrast, studies of the preventive effects of PUFAs on cardiovascular health have focused on CVD death [3,19] and nonfatal incidents [2,18]. Recognizing this, we define two separate parameters, the PUFA to fatal heart attack risk parameter, ζ (fractional decrease in fatal risk per mg PUFA intake/day), and the PUFA to nonfatal

heart attack risk parameter, ξ (fractional decrease in fatal risk per mg PUFA intake/day), for which we assume that a maximal risk reduction of 25% can be obtained by consuming 250 mg PUFA/day [2].

The lag effect in risk reduction or increase, τ (in years), is inherently uncertain because the mode of action for MeHg-associated heart attacks is not well-understood. We follow Rice et al. [15] and take studies in ex-smokers as a surrogate measure, which report the heart attack risk to decline to levels of never smokers between 2 and 10 years after quitting smoking.

Assumptions on benefit transfer variables. Kochi et al. [35] provide a recent meta-analysis using a Bayes pooling method to combine 197 VSL estimates from 40 selected studies. Their estimated composite distribution of Bayes adjusted VSL has a mean of $5.4 million and a standard deviation of $2.4 million. We approximate this distribution by a normal distribution truncated at zero. The value of nonfatal heart attacks is taken from EPA's clean air assessment [28], which quantifies the cost-of-illness to be in a range of $90,000 to $170,000 per of a nonfatal heart attack avoided. The valuation of (permanent) changes in IQ is based on an estimate of $2.45 million for undiscounted lifetime earnings [36]. Assuming a working lifetime of 45 years and a constant discounting rate of $\rho = 3\%$, we pegged the net present value of lifetime earnings L at $0.73 million. Further details of the distributional assumptions on the benefit transfer variables are given in Table 2.

Simulation. The simulation is based on a routine written in R (and is available upon request). Each simulation comprises 10,000 random draws and is repeated 500 times. Sensitivity analyses are based on Pearson's rank correlation measure, which allows identifying model uncertainties and on variations in selected input parameters to derive upper and lower bounds on our main results.

Results

We present the simulated distribution of the per capita health effects (in $/person) due to changes in fish consumption in Table 3, assuming that 25% of the population do not eat fish at all [21]. Under the perfect compliance scenario (column 2), the mean present per capita value of these health effects is only $0.22/person ($68 million across the population). The simulated distribution is weighted by observed consumption behavior from the NHANES survey [21] and suggests that a good part (63%) of female consumers between 16 and 45 years are already consuming fish in good compliance with the mercury advisory. Thus, in the simulation they do not change their consumption behavior. (We note that, since we use NHANES data from 2007-08, the advisory likely had an effect on consumption behavior and cannot be used to estimate the health economic value of the advisory per se).

In accordance with the NHANES data, 37% of the consumers are simulated to change their intake of fish so that they comply with the advisory. Some 34% of the consumers would benefit from a reduction in the amount of fish they currently eat or from turning away from certain high mercury fish. Since some high mercury fish are also rich in PUFAs, there are also a small number of simulated consumers (3%) who might risk a net harm to their unborn child when seeking to comply with the advisory.

In columns 3-5 of Table 3, we provide simulated distribution for the undesirable side effects scenario assuming that reductions in fish consumption by non-target individuals are in the order of 5%, 10%, and 20%. The results confirm that net losses in public health due to an overreaction to the mercury advisory by non-target individuals may be large. We estimate that across the population the expected loss of such an over-compliance may sum up to $2,986 - $12,048 million, depending on how much fish consumption is reduced. The distribution of health effects across the simulated cohort suggests that 7-9% of the consumers would obtain a net health benefit from reducing their intake of fish. The vast majority of consumers, however, would be harmed by altering their consumption behavior as this would increase the relative risk to suffer a heart attack in the given year by 0.77% on average. In other words, one would expect some 2,900 more CVD deaths across the U.S. population.

Sensitivity analysis. Table 4 displays Pearson's rank correlations (R) between each independent variable and the monetized neurodevelopmental health effects (column 2) and the total health effects (column 3-5). Notably, we find that the model is robust with respect to the uncertainty in the toxicokinetic input parameters and in the benefit transfer values ($R < 0.01$). This is clearly different from the model presented in Rice et al. [16], wherein the authors assume an exogenous, uniform drop in MeHg intake.

Once one allows for endogenous changes in MeHg and PUFA intake, as we do by using NHANES consumption data, individual consumption behavior becomes the most important driver of health effects. In scenario (2), we assumed a drop in fish consumption expressed on a percentage basis. So the more fish a consumer used to eat, the larger was her reduction in MeHg and in PUFAs after taking notice of the advisory. This explains the large negative correlation between the average amount of fish eaten per day, Q, and the total monetized health effects ($R < –0.47$). (Note that in scenario (1), we do not assume a percentage change in fish consumption and thus this correlation is much weaker). As required, total health effects are sensitive to reductions in PUFAs ($R < –0.55$) and MeHg ($R > 0.15$). The signs of these correlation coefficients state that the larger the reduction in MeHg intake, ΔM, the smaller will be the monetized net health loss; vice versa, the larger the reduction in PUFA intake, ΔP, the larger will be the monetized net health loss.

Discussion

The analysis presented above perfectly illustrates the risk-risk tradeoffs that consumers inevitably face in many food decisions. It is difficult, if not impossible, to eliminate the risks of contamination via food without increasing other health risks that may be even more harmful. Our estimates of the health effects resulting from different (over-)reactions to the USFDA mercury advisory encompass a broad range of individual outcomes depending on which and how much fish people eat. We have shown that reductions in fish consumption would cause net health losses to the largest part of the studied population.

The net loss results from the inclusion of cardiovascular effects of MeHg exposure and PUFA intake since, in line with earlier research on fish consumption [18,19], we find that the societal value of the expected effects on cardiovascular health are likely to dwarf those associated with neurodevelopmental health. Excluding these effects, our estimates suggest a small but positive net effect on neurodevelopmental health. The largest uncertainty in our estimates is due to the variability in fish consumption, and the necessary impact this variability has on the individual intake of MeHg and PUFA if one assumes a percentage reduction in consumption by non-target consumers.

While over-compliance does not seem to be an obvious reaction, a recent study by Shimshack and Ward [13] suggests that exactly this happened in response to the USFDA mercury advisory issued in 2001. The recognition that a well-intended consumer information campaign may have bad outcomes on public health is of large policy relevance. Indeed, John Q. Public faces a large variety of fish consumption advice from different sources expressing diverse concerns over toxicological, nutritional, economic, and ecological aspects and it is unclear how he evaluates this information in consumption decisions. We conclude that, given the potentially large unintended health effects, it is all the more important to carefully craft advisories in order to inform consumers about potential health risks without urging them into an avoidance behavior that is ultimately damaging.

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[Table 1] MeHg and PUFA concentrations in common fish species.

Fish	Market share (in %)	FDA advisory group	Mean MeHg (in μg/g)	MeHg distributional assumptions [a]	MeHg data source	Mean PUFA (in g/100g)	PUFA distributional assumptions [a]	PUFA data source
Anchovies	0.50	Low	0.043	Approximated (α = 0.229, κ = 0.188)	[23]	1.45	Fitted (α = 6.245, κ = 0.250)	[25]
Bass	0.61	Medium	0.219	Fitted (α = 1.379, κ = 0.215)	[22]	0.60	Approximated (α = 4.654, κ = 0.128)	[24]
Bluefish	0.09	Medium	0.337	Fitted (α = 7.207, κ = 0.047)	[22]	0.77	Approximated (α = 6.023, κ = 0.128)	[24]
Butterfish	0.10	Low	0.058	Approximated (α = 0.309, κ = 0.188)	[23]	0.59	Approximated (α = 4.615, κ = 0.128)	[24]
Catfish	4.80	Low	0.049	Approximated (α = 0.261, κ = 0.188)	[22]	0.22	Approximated (α = 1.713, κ = 0.128)	[24]
Clams	1.70	Low	0.017	Approximated (α = 0. 091, κ = 0.188)	[22]	0.11	Approximated (α = 0. 837, κ = 0.128)	[24]
Cod	4.71	Medium	0.095	Approximated (α = 0. 506, κ = 0.188)	[22]	0.28	Fitted (α = 9.426, κ = 0.029)	[25]
Crab	4.70	Low	0.062	Fitted (α = 0.587, κ = 0.106)	[22]	0.31	Approximated (α = 2.425, κ = 0.128)	[24]
Crawfish	0.60	Low	0.033	Approximated (α = 0.176, κ = 0.188)	[22]	0.17	Approximated (α = 1.330, κ = 0.128)	[24]
Croaker, Atlantic and Pacific	0.30	Medium	0.180	Approximated (α = 0.959, κ = 0.188)	[22]	0.22	Approximated (α = 1.721, κ = 0.128)	[24]
Flatfish	3.60	Low	0.045	Approximated (α = 0.240, κ = 0.188)	[22]	0.25	Fitted (α = 1.962, κ = 0.132)	[25]
Grouper	0.17	Medium	0.465	Approximated (α = 2.477, κ = 0.188)	[22]	0.25	Approximated (α = 1.956, κ = 0.128)	[24]
Haddock and Hake	2.47	Low	0.031	Approximated (α = 0.165, κ = 0.188)	[22]	0.13	Approximated (α = 1.017, κ = 0.128)	[24]
Halibut	0.90	Medium	0.252	Approximated (α = 1.342, κ = 0.188)	[22]	0.19	Approximated (α = 1.518, κ = 0.128)	[24]
Herring	2.50	Low	0.044	Approximated (α = 0.234, κ = 0.188)	[23]	1.61	Fitted (α = 6.245, κ = 0.250)	[25]
Lobster	2.11	Medium	0.273	Approximated (α = 1.451, κ = 0.188)	[22]	0.27	Approximated (α = 2.112, κ = 0.128)	[24]
Mackerel, King	0.05	High	0.730	Approximated (α = 3.888, κ = 0.188)	[23]	0.31	Approximated (α = 2.448, κ = 0.128)	[24]
Mackerel, Atlantic	0.20	Low	0.050	Approximated (α = 0.266, κ = 0.188)	[23]	2.30	Approximated (α = 17.992, κ = 0.128)	[24]
Mackerel, Chub	0.00	Low	0.088	Approximated (α = 0.469, κ = 0.188)	[23]	1.44	Approximated (α = 11.265, κ = 0.128)	[24]
Mackerel, Spanish	0.05	Medium	0.347	Approximated (α = 1.848, κ = 0.188)	[23]	1.34	Approximated (α = 10.482, κ = 0.128)	[24]
Monkfish	1.65	Medium	0.180	Approximated (α = 0.959, κ = 0.188)	[23]	0.61	Approximated (α = 4.772, κ = 0.128)	[24]
Mullet	0.20	Low	0.046	Approximated (α = 0.245, κ = 0.188)	[23]	0.33	Approximated (α = 2.581, κ = 0.128)	[24]
Oysters and Mussels	0.90	Low	0.013	Approximated (α = 0.069, κ = 0.188)	[22]	0.46	Approximated (α = 3.598, κ = 0.128)	[24]
Perch, freshwater	0.04	Medium	0.140	Approximated (α = 0.746, κ = 0.188)	[22]	0.25	Approximated (α = 1.979, κ = 0.128)	[24]
Pike	0.10	Low	0.31	Approximated (α = 1.651, κ = 0.188)	[22]	0.10	Approximated (α = 0.814, κ = 0.128)	[24]
Pollock	11.05	Low	0.041	Fitted (α = 0.506, κ = 0.087)	[22]	0.42	Approximated (α = 3.293, κ = 0.128)	[24]
Redfish	0.49	Low	0.010	Approximated (α = 0.053, κ = 0.188)	[22]	0.21	Approximated (α = 1.643, κ = 0.128)	[24]
Roughy	0.20	Medium	0.554	Approximated (α = 2.951, κ = 0.188)	[22]	0.02	Approximated (α = 0.149, κ = 0.128)	[24]
Sablefish	0.25	Medium	0.220	Approximated (α = 1.172, κ = 0.188)	[23]	1.40	Approximated (α = 10.913, κ = 0.128)	[24]
Salmon	8.24	Low	0.019	Fitted (α = 0.676, κ = 0.028)	[22]	1.35	Fitted (α = 4.734, κ = 0.285)	[25]
Sardine	1.20	Low	0.016	Approximated (α = 0.085, κ = 0.188)	[22]	0.98	Approximated (α = 7.666, κ = 0.128)	[24]
Scallop	0.80	Low	0.050	Approximated (α = 0.266, κ = 0.188)	[23]	0.10	Approximated (α = 0.806, κ = 0.128)	[24]
Shad	0.06	Low	0.065	Approximated (α = 0.346, κ = 0.188)	[23]	2.47	Fitted (α = 6.245, κ = 0.250)	[25]
Shark	0.13	High	0.988	Fitted (α = 2.451, κ = 0.403)	[22]	0.84	Approximated (α = 6.594, κ = 0.128)	[24]
Shrimp	15.14	Low	0.012	Approximated (α = 0.064, κ = 0.188)	[22]	0.06	Approximated (α = 0.477, κ = 0.128)	[24]
Snapper	0.50	Medium	0.189	Approximated (α = 1.007, κ = 0.188)	[22]	0.31	Approximated (α = 2.433, κ = 0.128)	[24]
Squid	1.03	Low	0.070	Approximated (α = 0.373, κ = 0.188)	[23]	0.49	Approximated (α = 3.817, κ = 0.128)	[24]
Swordfish	0.42	High	0.976	Fitted (α = 3.610, κ = 0.271)	[22]	0.75	Approximated (α = 5.898, κ = 0.128)	[24]
Tilapia	1.87	Low	0.010	Approximated (α = 0.053, κ = 0.188)	[22]	0.09	Approximated (α = 0.712, κ = 0.128)	[24]
Tilefish, Gulf of Mexico	0.01	High	1.450	Approximated (α = 7.723, κ = 0.188)	[23]	0.43	Approximated (α = 3.364, κ = 0.128)	[24]
Tilefish, Atlantic	0.03	Low	0.144	Approximated (α = 0.767, κ = 0.188)	[22]	0.43	Approximated (α = 3.364, κ = 0.128)	[24]
Trout, freshwater	0.69	Low	0.072	Approximated (α = 0. 383, κ = 0.188)	[22]	0.73	Approximated (α = 5.710, κ = 0.128)	[24]
Tuna, light canned	13.35	Low	0.118	Fitted (α = 1.611, κ = 0.073)	[22]	0.20	Approximated (α = 1.565, κ = 0.128)	[24]
Tuna, albacore canned	5.29	Medium	0.353	Fitted (α = 5.782, κ = 0.061)	[22]	0.55	Approximated (α = 4.302, κ = 0.128)	[24]
Tuna (fresh/frozen)	1.79	Medium	0.383	Fitted (α = 1.170, κ = 0.300)	[22]	0.51	Approximated (α = 3.990, κ = 0.128)	[24]
Weakfish (seatrout)	0.06	Medium	0.256	Approximated (α = 1.363, κ = 0.188)	[22]	0.37	Approximated (α = 2.910, κ = 0.128)	[24]
Whitefish	0.22	Low	0.069	Approximated (α = 0.367, κ = 0.188)	[22]	1.26	Approximated (α = 9.841, κ = 0.128)	[24]
Whiting	0.41	Low	0.010	Approximated (α = 0.053, κ = 0.188)	[22]	0.22	Approximated (α = 1.752, κ = 0.128)	[24]

[a] α = shape parameter, κ = scale parameter of the Gamma distribution.

[Table 2] Parameters used in the health effects valuation model.

Symbol	Definition	Units	Distributional specification	Central tendency	Variability [a]	Source
η	IQ to earnings parameter	Percentage change in $ per IQ point	Triangular distributed	Mode = 0.08	Min = 0.6, Max = 1.2	[33]
λ	Blood to hair mercury parameter	µg Hg/g hair per µg MeHg/L blood	Lognormal distributed	Median = 0.21	GSD = 1.85	[16]
γ	Hair mercury to IQ parameter	IQ points per µg MeHg/g maternal hair	Lognormal distributed	Median = 0.3	GSD = 3	[16]
β	MeHg intake to blood parameter	µg MeHg/L blood per µg MeHg/day	Normal distributed	Mean = 0.6	SD = 0.09	[16]
ι	PUFA to IQ parameter	IQ points per 100 mg DHA maternal intake/day	Normal distributed	Mean = 0.0013	SD = 0.00025	[5]
θ	Average fraction of DHA on PUFA in fish	Percent	Discrete value	0.617	-	[24]
ξ	PUFA to nonfatal heart attack risk parameter	Fractional decrease in fatal risk per mg PUFA intake/day	Triangular distributed	Mode = 0.00115	Min = 0.00099, Max = 0.00137	[2]
ζ	PUFA to fatal heart attack risk parameter	Fractional decrease in fatal risk per mg PUFA intake/day	Triangular distributed	Mode = 0.00102	Min = 0.0006, Max = 0.0012	[19]
φ	Hair mercury to heart attack risk parameter	Fractional increase in risk per µg Hg/g hair	Triangular distributed	Mode = 0.066	Min = 0, Max = 0.17	[16]
μ	Heart attack mortality rate	Ratio of fatal to all heart attacks	Discrete value	0.339		[37]
τ	Lag effect in risk reduction or increase	Years	Uniformly distributed	Mean = 6	Min = 2, Max = 10	[16]
ω	Plausibility of CVD causality parameter	Dimensionless	Bernoulli distributed	Mean = 0.5	SD = 0.5	[16]
ρ	Discount rate	Percent	Discrete value	0.03		[16]
B	Births in the U.S. in year 2009	Number per year	Discrete value	4,131,019	-	[38]
D_G	Number of fatal heart attacks within 100,000 individuals of gender G	Number per year	Discrete value	225 in females and 320 in males	-	[37]
H_G	Number of nonfatal heart attacks within 100,000 individuals of gender G	Number per year	Discrete value	665 in females and 945 in males	-	[37]
L	Discounted lifetime earnings	$ per average lifetime	Discrete value	879,266 (in 2009$)	-	[36]
M	MeHg intake per day	µg/day	Simulated value	Mean = 1.58	SD = 2.61	[Table 1]
N_G	Population older than 40 years	Number	Discrete value	73,858,958 females and 66,082,669 males		[39]
Q	Fish intake per day	g/day	Triangular distributed, weighted by NHANES consumption choices	Mean under Scenario (1) = 14.50 Mean under Scenario (2) = 16.24	SD = 18.11 SD = 21.59	[21,26]
P	PUFA intake per day	mg/day	Simulated value	Mean = 59.76	SD = 95.25	[Table 1]
VHA	Value of avoided heart attack	Million $ per nonfatal heart attack avoided	Triangular distributed	Mode = 0.12	Min = 0.09, Max =0.17	[28]
VSL	Value of statistical life	Million $ per fatal heart attack avoided	Triangular distributed	Mode = 5.5	Min = 1, Max = 10	[35]

[a] SD = standard deviation; GSD geometric standard deviation.

[Table 3] Simulated distribution of per capita health value (in $/person) due to changes in fish consumption, assuming that in accordance with the NHANES survey data [21] 25% of the population do not eat fish at all.

Percentiles	Scenario (1), perfect compliance with advisory	Scenario (2), reduction in fish consumption by 5%	Scenario (2), reduction in fish consumption by 10%	Scenario (2), reduction in fish consumption by 20%
5%	0.00	-40.06	-79.72	-158.23
25%	0.00	-11.39	-22.65	-45.07
50%	0.00	-4.11	-8.21	-16.42
75%	0.01	-0.11	-2.25	-4.57
90%	0.05	-0.03	-0.10	-0.28
95%	0.17	1.18	1.76	2.68
99%	10.14	13.86	20.25	33.51
Mean	**0.22**	**-9.73**	**-19.62**	**-39.24**
Share of beneficiaries	**34%**	**9%**	**8%**	**7%**

[Table 4] Rank correlation analysis.

Parameters		Scenario (1), perfect compliance	Scenario (2), reduction in consumption by 5%	Scenario (2), reduction in consumption by 10%	Scenario (2), reduction in consumption by 20%
η	IQ to earnings parameter	<0.01	<0.01	<0.01	<0.01
λ	Blood to hair mercury parameter	<0.01	<0.01	<0.01	<0.01
γ	Hair mercury to IQ parameter	<0.01	<0.01	<0.01	<0.01
β	MeHg intake to blood parameter	<0.01	<0.01	<0.01	<0.01
ι	PUFA to IQ parameter	<0.01	<0.01	<0.01	<0.01
ζ	PUFA to fatal heart attack risk parameter	-	<0.01	<0.01	<0.01
ξ	PUFA to nonfatal heart attack risk parameter	-	<0.01	<0.01	<0.01
φ	Hair mercury to heart attack risk parameter	-	<0.01	<0.01	<0.01
τ	Lag effect in risk reduction	-	<0.01	<0.01	<0.01
ω	Plausibility of CVD causality parameter	-	<0.01	<0.01	<0.01
VHA	Value of avoided heart attack	-	<0.01	<0.01	<0.01
VSL	Value of statistical life	-	<0.01	<0.01	<0.01
ΔM	Changes in MeHg intake/day	0.150	0.282	0.288	0.290
ΔP	Changes in PUFA intake/day	-0.046	-0.553	-0.565	-0.571
Q	Fish intake/day	-0.018	-0.475	-0.486	-0.490