A SERVING OF FARMED SALMON: HOW THE RISKS OUTWEIGH THE BENEFITS FOR NORWEGIANS

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Introduction

In Norway and the United States, cancer is currently the leading cause of death for those under 80 years of age, with mortality in Norwegians from heart and circulatory diseases being the lowest recorded in over 100 years.[1,2] Recent research on the human genetic variability with the enzymes responsible for the disposition of dioxin and dioxin-like compounds indicate that women with a variant of the cytochrome P450 1A1 (CYP 1A1) gene called m2 are at a greater risk for breast cancer when exposed to polychlorinated biphenyls (PCBs).[3] Currently, women living in North America have the highest rate of breast cancer in the world, and this cancer is the most frequent type found in Norwegian and European women.[4-6]

There are significant risks associated with fatty fish consumption. It has been concluded that fatty fish intake is the major source of dioxin, PCB, and polybrominated diphenyl ether (PBDE) exposure in the Norwegian population.[7,8] Studies by Hites and co-authors conclude from global assessments that Norwegian farmed salmon contain some of the highest concentrations of organohalogen contaminants, and that such farmed salmon should not be eaten more than once every five months due to increased cancer risk.[9-11] Such advice stems from both the United States Environmental Protection Agency (U.S. EPA) and the International Agency for Research on Cancer (IARC) as they have classified polychlorinated dibenzo-p-dioxins (PCDDs) as known human carcinogens, and PCBs as probably carcinogenic. The potential cancer risk from food borne brominated flame retardants, mixed chloro-bromo dioxin and furan derivatives, or their metabolites, remains to be evaluated. Organohalogen uptake in farmed Atlantic salmon from fish feed also shows that a selective enrichment exists for the most toxic congeners of PCDDs, PCBs, and PBDEs.[12,13]

Major differences of opinion exist within the scientific community over whether the dose-response curve for dioxin and dioxin-like compounds is best represented as non-linear (incorporating a threshold) or linear. The World Health Organization (WHO) and others strongly support a non-linear dose-response relationship for dioxin-like compounds and cancer, whereas U.S. EPA characterizes the curve as linear. In the recently completed U.S. EPA risk assessment for carcinogens, there were extensive discussions of approaches for extrapolations of risk for these compounds to low dose exposures.[14] As the German Federal Environmental Agency has commented, there is further confusion with regards to whether the tolerable intake and exposure of these contaminants should be described on a daily, weekly, or monthly basis.[15]

Fatty fish consumption is generally thought to be beneficial in the diet as they are a significant source of omega-3 fatty acids. Accordingly, the WHO and a number of countries (Canada, Sweden, United Kingdom, Australia, Japan) have made formal population-based dietary recommendations for omega-3s. Based on the assumption that the health benefits outweigh the risks, the current advice from national and international authorities is to increase fish

consumption.[16-18]A potential way to quantitatively evaluate the risks versus the benefits of a particular food item, that is understandable to consumers, is to examine the contents in a recommended serving size relative to the established tolerable daily intake. Such a comparison for selected contaminants and marine omega-3 concentrations is presented here for farmed Norwegian salmon.

Results and Discussion

The typical recommendation for the marine omega-3 fatty acids, eicosapentaenoic acid (EPA) plus docosahexaenoic acid (DHA), is a consumption of 0.2 - 0.5 g per day.[19] With regards to EPA and DHA tolerable intake, a number of scientific studies show that consumption levels of EPA and DHA over 3 g per day may lead to certain toxicities such as excessive bleeding, and therefore the U.S. Food and Drug Administration (U.S. FDA) recommends that consumption of these fatty acids not exceed 3 g per day.[20] The results shown in Table 1 for EPA and DHA from a serving of farmed salmon indicate that a Norwegian serving size of 200 g exceeds the daily tolerable intake of these omega-3s. In addition, researchers from the University of East Anglia and eight other institutions have recently demonstrated that when the results of over 85 studies with were pooled into a meta analysis, the unbiased results showed no strong evidence that marine omega-3s had an effect on overall deaths, heart disease, stroke or cancer.[21]

The observed decrease in EPA and DHA found in Norwegian farmed salmon from 2001 to 2003 shown in Table 1 is likely due to the increased use of vegetable oil in fish feeds in the face of diminishing marine raw materials. This is a welcome trend for food safety, as vegetable oil and Atlantic salmon farmed on vegetable oil based feeds contain higher levels of the only essential omega-3 fatty acid, alpha-linolenic acid (which is the metabolic precursor of EPA and DHA in humans) and significantly lower levels of organohalogens.[22-25]

The intake of a serving size of Norwegian farmed salmon from 2005 (Table 1), with average concentrations for dioxins and DLPCBs of 1.6 pg WHO-TEQ/g fillet, would provide a 60 kg consumer an average intake of approximately 3 and 5 pg/kg bw for 130 and 200 g, respectively. These results are 10-fold greater than the average of 0.3 pg WHO-TEQ/kg bw estimated for a 130 g portion of ruminant meat or poultry.[23] While the selected contaminants in Norwegian farmed salmon shown in Table 1 have well known developmental toxicities, in addition to being carcinogens, no official food advisories exist in Norway for children or women of childbearing age for farmed salmon. An explanation can be found from statements of the National Institute of Nutrition and Seafood Research (NIFES), advisors to the Norwegian Food Safety Authority (Mattilsynet) and Ministry of Fisheries, where they do not publicly support the refining or replacement of fish oils for use in fish feeds because the resulting dioxin and DLPCB levels currently found in Norwegian farmed salmon are below the current legal limit of 8 pg WHO-TEQ/g fillet in the European Union (EU) and Norway.

However, from the occurrence data for farmed salmon shown in Table 1, consumption of a Norwegian serving size of 200 g places a consumer of 60 kg over the upper limit of the WHO tolerable daily intake for dioxins and dioxin-like PCBs. With the recommended 2 meals a week for fatty fish for Norwegians, for just farmed salmon, this equals over 70% of the EU total tolerable weekly intake (TWI) of 14 pg WHO-TEQ/kg body weight (bw) for dioxins and DLPCBs. What is not relayed to consumers is that if a single 200 g portion of ‘food’ actually

contained a level of 8 pg WHO-TEQ/g, this would result in an individual consuming nearly double the EU TWI with an astounding 26 pg WHO-TEQ/kg bw.

For indicator PCBs, the 2004 concentrations shown in Table 1 averaged 12 ng/g skinless fillet in farmed Norwegian salmon, and this has provided 60 kg consumers with 40 ng/kg bw with a 200 g portion. As with dioxins and DLPCBs, the European Food Safety Authority (EFSA) estimates that the average intake of nondioxin-like PCBs for 130 g farmed salmon is about 10-fold higher when compared to ruminant meat, where an intake of 130 g provides 2.7 ng/kg bw.[23] With PBDEs, farmed Norwegian salmon from 2004 contained an average of 2.5 ng/g fillet, resulting in a 130 g portion delivering 5.4 ng/kg bw compared to a 50-fold lower intake of 0.1 ng/kg bw for meat or poultry.[23] Clearly, Norwegian risk-benefit analysis for farmed salmon is based to a great degree on preventing economic loss to the industry and does not reflect appropriate health consumption-based advice to consumers.

It is interesting to note that as the intake levels of dioxins and dioxin-like PCBs in farmed Norwegian salmon exceed established TDIs, the TDI assessments are denounced and exposure levels on a weekly or monthly basis are deemed more acceptable. The longer exposure period, in relation tolerable intake, is believed to correlate better to the long half-life of these contaminants in human tissues. However, as reviewed in Refs.15 and 25, this is just wishful thinking and is not based on an understanding of metabolism or the scientific evidence for the mode of action for toxicological effects (pharmacodynamics) such as CYP1 induction associated with the concentration-time course (pharmacokinetics) of these contaminants. Procarcinogen-activating CYP1 enzymes are considered to play an important role in chemical carcinogenesis, and their activation in animal and human tissues can occur directly after exposure.[26]

With regards to the threshold versus linear curve for dioxin and dioxin-like compound carcinogenesis, we only need to review the current scientific literature to see that both approaches appear correct.[27-29] Liver enzyme induction has been shown previously to be regional with clear borders between induced and uninduced regions in vivo.[29] Initial interactions of dioxin-like ligands with the dioxin-binding site on the aryl hydrocarbon receptor (AhR) may be linearly related to concentrations of these ligands. However, there are many events which follow receptor binding that likely need the components of a threshold or switch model to develop a predictive model for low-dose dioxin exposures.

Table 1. Average concentrations (SD) and dietary intake estimates for marine omega-3 fatty acids (EPA and DHA) and selected organohalogen contaminants associated with a serving of skinned fillet from Norwegian farmed salmon to a 60 kg (body weight, bw) consumer.

Reference	Concentration in fillet	EFSA 130 g Portion	Norwegian 200 g Portion	Intake Estimate 200 g/60 kg	Daily Recommended or Tolerable Intake
Sum EPA + DHA	(g/100g wet wt)	(g/portion)	(g/portion)		(g/day)
EFSA, 2005	1.9	2.5	3.8	na	
NIFES, 2003	1.5	2.0	3.0	na	**0.2 - 0.5 or < 3****
NIFES, 2001	3.0	4.3	6.0	na	
Sum of Dioxins + DLPCBs	(pg WHO-TEQ/g wet wt)	(pg WHO-TEQ)	(pg WHO-TEQ)	(pg WHO-TEQ/kg bw)	(pg WHO-TEQ/kg bw/day)
NIFES 2005 (n = 44)	1.6 (nd)	204	314	5	
NIFES 2004 (n = 12)	1.6 (nd)	204	314	5	**1-4*****
NIFES 2003 (n = 25)	1.9 (nd)	241	370	6	
100 % fish Oil feed (n = 8)*	1.4 (0.2)	186	286	5	
50% fish Oil feed (n=8)*	0.9 (0.1)	118	182	3	
100 %Vegetable oil feed (n=8)*	0.5 (0.1)	66	102	2	
Sum of Indicator PCBs	(ng/g wet wt)	(ng)	(ng)	(ng/kg bw)	
NIFES 2004 (n = 12)	12 (nd)	1560	2400	40	Not Established
NIFES 2003 (n = 25)	10 (nd)	1300	2000	33	
NIFES 2002 (n =45)	11 (nd)	1430	2200	37	
100 % fish Oil feed (n = 8)*	7.25 (1.34)	943	1450	24	
50% fish Oil feed (n=8)*	4.85 (0.64)	631	970	16	
100 %Vegetable oil feed (n=8)*	3.00 (0.81)	390	600	10	
Sum of Primary PBDEs	(ng/g wet wt)	(ng)	(ng)	(ng/kg bw)	
NIFES 2004 (n = 12)	2.48 (0.50)	322	496	8	Not Established
NIFES 2003 (n = 25)	2.33 (1.07)	303	466	8	
100 % fish Oil feed (n = 8)*	2.14 (0.33)	278	428	7	
50% fish Oil feed (n=8)*	1.70 (0.24)	221	340	6	
100 %Vegetable oil feed (n=8)*	1.05 (0.19)	137	210	4	

na = not applicable; nd = not determined; EFSA, 2005 is Ref. 23; NIFES data is from Ref. 30 and 31;*values from Ref. 25; **Ref. 20; ***World Health Organization (WHO). (1998). Assessment of the health risks of dioxins: re-evaluation of the tolerable daily intake (TDI). *Executive Summary of the WHO Consultation*, May 25-29 1998, Geneva.
The results presented here are in accordance with WHO methods (7 PCDDs, 10 polychlorinated dibenzofurans (PCDFs) and 12 DLPCBs). The indicator PCBs cover 7 congeners primarily found in the food chain (28, 52, 101, 118, 138, 153, and 180) and PBDEs represent congeners 28, 47, 99, 100, 153, 154.

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