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12/03/98 11:53 AM

To: Georjean L. Adams/US-Corporate/3M/US@3M-Corporate
cc:
Subject: Risk to the environment due to the presence of PFOS

In the attached analysis of possible risk of ecological harm due to environmental levels of PFOS, I concluded there is a significant risk of harm. In addition to this, my review of the available data on the properties of PFOS, indicates that the sink for it in the environment is biota. We know of nothing that it has more affinity for than blood serum and liver components. Other persistent pollutants have a high affinity for sediment, but PFOS does not. So, PFOS can't be trapped and buried like they can. It likely continues to partition into biota from the other compartments in the environment; thus the levels we are seeing in eagles and other biota is likely to climb each year.

I believe all this taken together constitutes a significant risk that should be reported to EPA under TSCA 8e.

Pioneer Food Chain Risk Assessment of PFO

Pioneer Risk Assessment of Adverse Effects in Marine Mammals from PFOS in the Food Chain

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3 December 1998

Introduction:

PFOS (perfluorooctanesulfonate) was found in two of three populations of naïve rats--that is, rats not knowingly exposed to PFOS. The rats that had PFOS in their blood were fed food that contained fishmeal. Incomplete studies indicate that PFOS is in fishmeal. This is consistent with the observation that fledgling eagles that eat predominately fish contain PFOS in their blood.

Most fishmeal is made from menhaden (*Brevoortia tyrranus*)(Draft Report: Sources of Fish Food Constituents By John Giesy and Paul Mehrle, September 6, 1998). Menhaden eat plankton and are considered a second link in the US Atlantic coastal food chain. They are in turn eaten by other species of fish, which are in turn eaten by other fish, mammals and birds. The mass of menhaden is so great that their catch represents 40% of the US commercial finfish fisheries.

Since these fish appear to contain PFOS, it seemed prudent to calculate the amount of PFOS that might be transferred up the food chain and compare this value to a concentration that causes adverse effects. These calculations do not contain precise data so the evaluation is approximate. The purpose is to see whether predicted environmental concentrations are anywhere near concentrations that cause effects.

This assessment was organized and performed in accordance with the guidance given by EPA in Guidelines for Ecological Risk Assessment (EPA/630/R-95/002F April 1998). The process presented in these guidelines is a repetitive one where a risk assessment is updated or redone when new information is available. The new information can be generated by other processes or generation can be driven by the risk assessment.

Problem Formulation:

A study of rats not purposely exposed to PFOS found that they had significant levels of it in their livers. The likely source was the fishmeal in their diet. If PFOS is in fish then other fish, fish eating mammals and birds are consuming PFOS. The concentrations found are not likely to cause toxic responses, but what about after biomagnification through a food chain?

The hypothesis to test: The concentration of PFOS in food of marine animals causes adverse effects.

It was decided to keep the problem narrow in this assessment in order to simplify the analysis. This is not to imply that this is the most important or only problem. In addition this is a pioneer and possibly first iteration of this assessment. Refinements in the analysis and more data will allow other iterations if this pioneer assessment indicates there may be a significant risk. The purpose of this pioneer assessment is to determine the magnitude of the risk and whether other iterations are warranted.

Analysis

Characterization of Exposure:

The task of this characterization is to estimate the concentration of PFOS in the food chain organisms that eat menhaden and the concentration in the animals comprising the next two steps in the food chain. Two examples of the many food chains possible are:

Menhaden -> cod -> seal -> killer whale

Menhaden ->carnivores fish 1 -> carnivores fish 2 -> seal

For the purposes of this analysis the biomagnification by fish and mammals is about the same. This is supported by the metabolic rates, daily consumption and growth rates published (Fugacity-Based Model of PCB Bioaccumulation in Complex Aquatic Food Webs, Jan Campfens and Donald Mackay in Environ. Sci. Technol. 31, 577-583(1997) and Wildlife Exposure Factors Handbook, EPA/600/R-93/187 December 1993). Based on the data in these references and recollection from other readings, an average biomagnification value is 9.

In an evaluation of PFOS in the livers of naïve rats an average level found in male and female 10-14 week rats was 0.09 mg/kg. The food label listed fishmeal as the fifth ingredient. According to labeling standards, fishmeal was less than 20% of the food. For this analysis it is assumed that fishmeal made up 16% of the food and that it was the only source of PFOS. This value could be too high which would mean the ultimate calculated tissue levels would be low, but the value cannot be too low by much because the most it could be is 19%. If an animal were to eat 100% fish, as many fish and sea mammals do, it should have about 6 times as much in its livers as these rats do. That level would be 0.56mg/kg (0.09mg/kg/0.16).

This is the estimated level in the liver, which is the organ that contains most of the PFOS. The blood contains the next highest level. Work with rats has shown that blood contains about 1/6 the amount of PFOS to be found in the liver. Assuming that the liver mass is 3% and blood is 8% the mass of an animal and that the concentration in other organs is insignificant in comparison to the levels in liver and blood, then the total body burden of PFOS in the first step of the food chain above menhaden is 0.062mg/kg (0.56mg/kg X 0.03 + (0.56mg/kg /6) X 0.08)

The level in the second food chain link then would be nine times this level or 0.56 mg/kg.

Using data on seals, the accumulated dose at time of whelping is calculated. It is assumed that 100% of PFOS consumed is retained. Data for killer whale food consumption sexual maturation was not immediately available, but they are assumed to be similar.

The mean time for sexual maturity for female harbor seals is 5.5 years and gestation is 11 months (Wildlife Exposure Factors Handbook, EPA/600/R-93/187 December 1993). So for up to 77months before whelping a seal was assumed to be eating fish with the above calculated concentration of 0.56 mg/kg PFOS. Seals eat 6-8% of their weight per day in fish. They eat 13% their first year and 10% when gestating. For the ease of calculation it was assumed that an 80kg seal eats 8% or 6.4kg/day. This works out to 15 X 10^3 kg of fish (77months X 30days/month X 6.4kg/day). Assuming the fish a seal consumed contained 0.56mg/kg, then a seal would consume about 8.4 X 10^3 mg PFOS (0.56mg/kg X 15 X 10^3 kg). This works out to a cumulative dose before whelping of about 105mg/kg (8.4 X10^3 mg/80kg)

This cumulative dose can be used for seals that are two food chain lengths above menhaden. This is probably not the norm. The average seal is probably one food chain length above menhaden. But this calculated cumulative dose probably represents that seen by populations of killer whales that eat seals.

Characterization of Ecological Effects:

In a two generation rat study it was found that 34%or the pups born to animals dosed by gavage 1.6 mg/kg per day PFOS were born dead or died within four days of birth. For the purposes of this assessment rats are assumed to be an adequate test surrogate species for marine mammals. There is uncertainty both to whether marine mammals such as seals, sea lions, and killer whales are less sensitive or more sensitive than rats.

The lowest dose of 1.6mg/kg that caused an adverse effect was given for 6 weeks before mating, the week of mating and the 22 days of gestation for a total of 71 days. The cumulative dose up to the time of whelping is 113mg/kg.

It has been reported that natural populations of sea lions are experiencing significant reproductive dysfunction (Peter Ross, Environmental Contaminants and the Risk of Adverse Effects in Marine Mammals: An Overview, 1998 SETAC Annual Meeting). Also 70% of a ringed seal population was not reproducing. This is higher than the norm. Apparently some field biologists believe killer whale reproductions has declined, but there has been no scientific study to verify this.

Risk Characterization:

The predicted cumulative dose of PFOS through two food chain links to sea mammals such as seals, sea lions, killer whales and porpoises was calculated to be about 105 mg/kg. This value is about the same as the calculated cumulative dose of 113 mg/kg PFOS that causes reproductive impairment to mammals. Thus there is a significant risk. There is not enough input data to calculate uncertainty in this risk. Such data is needed to reevaluate the degree of risk.