

3M has compiled several different study results that appear to indicate the potential for significant environmental contamination by the toxic chemical PFOS. The studies are with diverse species in widely different environments (laboratory rats and bobwhite to wild eaglets and fish) and involve very few individuals. However, the results, when considered together, suggest a pattern of exposure and concern for biological magnification to levels that could be at toxic levels.

Biological magnification is a trophic process in which retained substances become more concentrated with each link in the food chain. Bio-magnification may result in toxic levels in the highest trophic levels. 3M now has fragmentary, but scientifically credible evidence, that PFOS may have the potential to present bio-magnification to levels of concern, as outlined below. To ensure the accuracy of these preliminary conclusions, it is necessary to conduct more extensive field-testing of higher trophic level organisms.

Assumptions and preliminary conclusions:

1. PFOS bioaccumulates over time with repeated dosing, with highest levels found in the oldest individuals. As older food-source animals are eaten by longer-lived animals in higher trophic levels, the levels cumulative dose/body burdens in the higher trophic level individuals can increase dramatically. Supporting data: naïve rat liver study; 3M employee blood study.
2. PFOS is widely present in wild fish. We do not know the source of contamination. Supporting data: 13-27 ppb in fish meal used in laboratory rat food, composed of dried menhaden, a lower trophic level salt water seafood source; ~~rehydrated~~ whole, hydrated menhaden are predicted to have levels between 2 and 5 ppb. Other wild freshwater fish have detectable, but to date, unquantified, amounts of PFOS; these levels The concentrations of PFOS in these fish have not been quantified at this point in time; however, they are suspected to be on the order of 1 to 10 ppb.
3. PFOS can biomagnify, increasing in concentration in trophic levels that eat contaminated organisms. PFOS has been found to be retained and accumulate with subsequent dosing, partitioning primarily to blood and liver, with liver concentrations being typically several times higher than blood. Supporting data: see previous 8e submissions/health white paper. [PFOS in human serum, TSCA 8e submission, 5-15-98; Two-generation rat reproduction study, TSCA 8e submission, 9-14-98]

**Exhibit
1575**

State of Minnesota v. 3M Co.,
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4. Fish-eating birds: PFOS is present in the offspring of two species of wild, high trophic level species that feed on fish. These levels are consistent with feeding on fish with 1 ppb PFOS. Supporting measured data: American bald eaglets at 30-494 ppb in blood plasma; one adult at 1047 ppb in blood plasma. Baltic sea eaglet at 93 - 215 ppb in blood plasma. ~~Indicating a geographic difference, Pacific albatross blood plasma had barely detectable PFOS, assumed to be on the order of 1 ppb.~~ This is significant due to the geographical location and the fact that food sources for the pacific albatross would well up from the ocean.
5. Sea mammals: Contamination seen in menhaden would indicate the likelihood of increased PFOS in higher sea mammals in a food chain that includes menhaden. ~~Calculations-~~ Estimations based on a PFOS-contaminated food chain beginning with containing menhaden with 1 ppb, PFOS yields cumulative doses ~~body burdens~~ of 35 ppm in seals and 13000 ppm in killer whales at sexual maturity [provide detail on how numbers were derived]. Since indications in mammals, including retired exposed fluorochemical workers, suggest a very long half-life of elimination from the body, cumulative dose can be reasonably assumed to approximate body burden. Food chain: menhaden → cod → seals → killer whales. Since each level is longer-lived and thus ingests more contaminated food over their lifetime, the levels body burdens increase dramatically. ~~No data exists yet on these higher levels~~ While the fish-meal analysis can be used to establish body burden for menhaden, no direct measurements of body burdens in higher-trophic-level species have been made at this time.

Assumptions:

- Only food sources incorporating PFOS are consumed at all trophic levels.
- No significant uptake from water is assumed for the higher trophic levels. (Menhaden PFOS sources are unknown.)
- The values assume identical, 100% retention rates in all species. Cumulative body burden is thus considered to be equivalent to cumulative dose. ~~Primate testing appears to indicate high trophic levels may retain a higher percentage of PFOS than lower trophic levels.~~ Comparative Toxicity and/or pharmacokinetic data suggests that PFOS is essentially completely absorbed from the digestive tract and is poorly eliminated. ~~Rats are more efficient at eliminating an absorbed dose of PFOS than humans or monkeys, yet~~ All of these species appear to exhibit long half-lives of elimination. In part, this is due to efficient enterohepatic recirculation and sequestration in blood and liver tissue. Since there is no known metabolism of PFOS and covalent interactions are highly unlikely, distribution and accumulation in tissue becomes dependent on solubility, physiology and association with proteins and cellular components. The only major interspecies differences that should affect distribution and accumulation would be physiology and content of association sites. Association with blood and liver proteins should be similar between species. Urinary and biliary excretion may be somewhat variable between species.

6. Toxicity testing in various species indicate suggest that wild animals at the highest trophic levels may develop cumulative body burdens/doses near or exceeding toxic levels, assuming they consume only contaminated food. Supporting laboratory data (values are cumulative doses): Bobwhite LOAEL <12 ppm (single oral dose at 12 ppm produced severe toxic effects). Rat LOAEL 104 – 112 ppm (perinatal death in two-generation reproductive study) [~~Data just obtained may suggest this is lower~~]. Primate LOEL 56 ppm (lowered cholesterol). Primate LOAEL <135 ppm (severe cholesterol reduction, enlarged liver, weight loss, ~~possible 1/12 death not clearly attributed to PFOS until pathology report received~~) (two deaths among the six high dose males occurred late in the study which have not yet been conclusively attributed to PFOS administration) . Primate LD50 < 225 ppm. Comments:

- If doses are not retained at 100%, as assumed in #4, the corresponding toxic levels in terms of body burden are reduced proportionally; i.e., the conclusion [which conclusion??] is unchanged.
- Species differences conventionally call for dividing the effect levels by a factor of 10x.
- Acute testing vs. chronic exposures conventionally call for an additional factor of 10x reduction in effect levels.
- Blood levels are assumed to be the same as total body burden concentrations, insofar as there appears to be selective concentration into blood and liver, less in muscle tissue (likely due to the presence of blood), and essentially no presence in fat (PFOS is oleophobic).

The above assumptions indicate a *toxic level* [is this based on application of the safety factors above? It is not entirely clear what these values represent.] to eagles of <120 ppb and to seals and whales of 1 – 1.3 ppm [body burden and/or serum level???]. These levels have been seen in measurements in the former (albeit in very few animals) and predicted from food chain biomagnification in the latter. [These values as a “toxic level” do not seem to be supported by the data. We have not seen direct mammalian toxicity at serum levels less than 150 ppm, including our most sensitive indicator, total serum cholesterol reduction. Are these meant as “no expected effect levels?” If so, I believe I can support that conclusion based on human epidemiology and other data available to us.]

The attached chart attempts to collate these data and body burden predictions. Taken together, these data and assumptions reasonably support a preliminary conclusion that there is substantial risk to high trophic level environmental organisms if they primarily eat PFOS contaminated food. Certainly it is necessary to conduct environmental sampling of various trophic levels to fully confirm this conclusion. 3M is conducting such testing. [Are we attaching a chart????]