Submission
No 97

INQUIRY INTO DENTAL SERVICES IN NSW

Organisation: Australian Fluoridation Information Network

Name: Ms Ailsa Boyden

Position: Spokesperson

Telephone:

Date Received: 2/06/2005

Theme:

Summary

Committee Social Issues - SUBMISSION TO NSW Dental Inquiry

From: "Ailsa Boyden" <boydens@mrbean.net.au>
To: "The Director Standing Committee on Social Issues" <socialissues@parliament.nsw.gov.au>
Date: 2/06/2005 1:10 AM
Subject: SUBMISSION TO NSW Dental Inquiry

The Director
Standing Committee on Social Issues
Legislative Council
Parliament House
Sydney 2000
socialissues@parliament.nsw.gov.au.

Dear Director,

I have only in the last ten minutes heard about the NSW Dental Inquiry and respectfully, belatedly, and apologetically tender (below) a copy of my submission sent to the NSW Standing Committee on Public Works in relation to Coastal Infrastructure.

I believe that this submission is very relevant to your NSW Dental Inquiry. Would you please be so kind as to let me know if this submission will be accepted for consideration?

Yours truly,

Ailsa Boyden
Spokesperson
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.....

The Committee Manager
Ms Carolynne James
NSW Standing Committee on Public Works
C/- Natasa.Tosic@parliament.nsw.gov.au

Dear Ms James,

SUBMISSION: NSW Parliamentary Inquiry into Coastal Infrastructure re:

- **Point 4:** Best practice methods to plan, manage and provide infrastructure to coastal growth areas;
  Point 5: Management of social, environmental and economic considerations associated with infrastructure provision in coastal growth areas.

There is strong evidence to suggest that fluoride is not biologically safe and that fluoridation presents a risk to the long-term health of the natural environment. The reasons for fluoridation do not seem soundly based in that the effects of fluoride ingestion have never been systematically studied and there is no clear proof that fluoride consumption helps reduce tooth decay. To add a major source of long-term pollution into water supplies is an unnecessary risk and a grossly unnecessary expenditure from the public purse. This money could be put to far better use through direct dental care to those who need it most.

Fluoride accumulations threaten sustainability; therefore, I wish to draw your attention to the potential for an ever-increasing build-up of highly-toxic fluoride in the environment from various sources.

Fluoride

- is acutely toxic
  is more poisonous than lead
  is only slightly less toxic than arsenic
  is non-biodegradable
  is accumulative and
  is a 'protoplasmic poison' that inhibits enzymes that are essential to all life: e.g. Prozac is a fluorinated medication whose generic name is fluoxetine. In a trial using children aged 8 to 17 years who were given fluoxetine, the results revealed that treatment with fluoxetine was linked to a decrease in the alkaline phosphatase enzyme which is crucial to bone development - the children on fluoxetine gained an average 1cm less in height and 1.1kg less in weight than those treated with a placebo. (See attachment re_*Trial finds that Prozac (fluoxetine) may stunt children's growth.*)

Toothpaste a major source of fluoride pollution

The evidence that very small amounts of fluoride are poisonous can be gauged by the fact that in the US, only a pea-size amount of fluoride toothpaste is recommended for each tooth-cleaning session. This toothpaste has to carry a warning to the effect "if you swallow more than needed to clean teeth contact a poison control centre or a professional person immediately." In other words, more than a pea-size amount is hazardous to health; yet untold and unacknowledged amounts of fluoride from toothpaste (and other dental caries prophylactics) along with fluoridated medications (e.g. Prozac) are being released continuously into the environment. The rate of fluoride discharge is not being monitored; nor are its effects on human and other biological health.

Professional fluoride dental treatments (as administered by dentists) contain lethal doses of fluoride: e.g. a 3-year-old child died as a result of accidentally swallowing his professionally administered dental rinse, instead of spitting it out. Does this high-dosage fluoride also enter sewerage systems and then waterways? What is its effect? We simply do not know and apparently are not attempting to find out.
(See attachment: *3-year-old dies from swallowing fluoride mouth rinse administered in dentist's chair.*)

Fluoridated water

Despite growing evidence that fluoride is harmful to human health and the environment, NSW Health is still pursuing a program of extending fluoridation throughout NSW. This plan includes fluoridating all communities along its eastern seaboard and serves to achieve nothing more that adding highly-toxic non-biodegradable fluoridating agents to the chemical cocktail that makes its way, via community water supplies, into waterways and biological systems. Many hundreds of tonnes of fluoridating agents are used annually in individual capital cities alone.

Fluoridating agents are environmental pollutants

The fluoridating agents generally used in most fluoridation schemes in Australia are captured in pollution control 'scrubbers' during the manufacture of phosphate fertiliser. The chemicals are captured in scrubbers to prevent their escape into the environment where they are classed as a pollutant. It is illegal to dump silicofluorides at sea but somehow it has come to pass that it is considered desirable to put those same chemicals into our drinking water.
(See www.fluoridealert.org/phosphate/overview.htm for an overview of the phosphate industry.)

Silicofluorides are delivered to water supplies as industrial-grade products which are contaminated with arsenic, lead and other heavy metals. They are used despite never having been toxicologically tested for their effects on human health. Silicofluoride was finally accepted for preliminary testing by the US National Toxicology Program in 2002 on the grounds that it is used in 91% of fluoridation systems and has never been toxiciologically tested but that the historical assumptions about its safety have not been substantiated by the experimental evidence. Silicofluoride was accepted onto the Australaian chemicals database in 1990 as an 'existing chemical' but has never been formally studied for its effects on human or other biological health.

To add insult to environmental injury, these contaminated industrial-grade environmental poisons are often sourced from Japan Belgium and China for instance. None of these countries fluoridates the majority of their population. China considers fluoride too harmful to health to add to its own community water supplies.

Material safety data sheets for fluoridating agents

The Material Safety Data Sheet (MSDS) for the fluoridating agent hydrofluorosilicic Acid (obtained under Freedom of Information from Melbourne Water) states under the heading "Safe handling information", Spills and Disposal:

- "*...Do not contaminate streams, rivers or water courses.*" (emphasis added)

In relation to spills of the fluoridating agent sodium silicofluoride, the MSDS obtained from Melbourne Water states:

- " *...Prevent from entering drains, sewers, streams or other bodies of water...*" (emphasis added)

The MSDS lists the **chronic effects** of this agent as

- "Prolonged and repeated exposure may cause embrittlement and densificationof bones, increased calcification of ligaments and vertabrae resulting in spinal stiffness. Enzyme system effect and pulmonary fibrosis have been reported."

It seems reasonable to assume that the widespread accumulations of fluoride in coastal and marine environments are paving the way for future biological and ecological catastrophes.

Harm outweighs benefits so costs of fluoridation are unjustified

The installation of fluoridation costs approximately $1 million per water supply, with an on-going annual cost of over $60,000. When fluoridation studies are closely examined one finds that the reported benefits of fluoridation are exaggerated and the adverse effects are under estimated; in fact, MANY parents are having to pay for expensive restorative dental treatments owing to the high percentage of children whose teeth have been damaged from excessive fluoride ingestion. This visibly reveals itself as undermineralization of teeth and dental staining (dental fluorosis) - the result

of chronic poisoning of tooth-forming cells by fluoride while the teeth are developing. For instance the Spencer *et al* 1996 study found a 56% rate of dental fluorosis in SA and 40% of dental fluorosis in WA amongst the fluoridated children studied. They also found that approximately 30% of the NEVER-fluoridated children studied, in these states, had dental fluorosis and this is presumed due to the increasing incidence of fluoride in modern foods (processed with fluoridated water) and modern medications.

It appears from our investigations that no Australian agency is monitoring the present rate of fluoride ingestion. Nor are they systematically monitoring the incidence of adverse effects on health from fluoride ingestion. The much-awaited NHMRC study into fluoride's health effects was cancelled in 2002 by the Australian Health Administration Corporation on the grounds of "insufficient resources". The NSW Government appears unwilling to recognise this situation and is continuing to promote fluoridation as being both 'safe' and 'effective'. Actually the lack of even the most basic fluoride studies means that neither of these descriptions is correct.

Fluoridation is NOT delivering the claimed benefits

Perth (fluoridated since 1968) is experiencing a dental crisis with "toddlers as young as one-year having general anaesthetics to remove or crown rotten teeth". (See http://fluoridealert.org/news/2179.html.)

Fluoridated Sydney is also experiencing a dental crisis (Sydney Morning Herald, February 15/16).

The Child Dental Health Survey 2000 (Table 12) shows that most Health Regions in 100% fluoridated Sydney have worse rates of dental decay in children's permanent teeth than largely un-fluoridated communities such as the NSW Mid North Coast Health Region (only about one-third fluoridated). Furthermore, the Nambucca Shire of the Mid North Coast has worse dental decay rates in children's permanent teeth than non-fluoridated Kempsey and Port Macquarie-Hastings.

The Australian researchers Armfield and Spencer reported, "... we found no significant effect of fluoridated water on the permanent caries experience among 10- to 15-year-olds..." (when compared with those drinking bottled or rain water). (Armfield JM & Spencer AJ (2004) *Community Dentistry and Oral Epidemiology,* 32 (4):283)

Data compiled by the World Health Organization shows tooth decay declined at a similar rate in all Western Countries after the 1950's, irrespective of each country's water or salt fluoridation status (www.fluoridealert.org/WHO-DMFT.htm).

The US Centers for Disease Control report that "preschoolers cavities increased despite 60 years of water fluoridation, 50 years of fluoridated toothpaste and a virtually 100% fluoridated food supply." (See http://www.cdc.gov/nchs/ppt/hpdata2010/focusareas/fa21.ppt)

Fluorinated medications

MANY prescription medications are fluorinated: e.g. the anti-depressant Prozac (fluoxetine). Sufficient fluoride is now making its way from humans into waterways from Prozac alone that its residue is being found in British waterways.

Although Prozac does not have a licence for use on children, there has been a massive rise in the number of UK doctors and psychiatrists prescribing it for children on an "off-licence" basis and a considerable number of adults are routinely prescribed this fluorinated medication. In 2004 it was reported that Prozac was now found in measurable amounts in the groundwaters of the UK. How many adults and children are excreting this substance in NSW's coastal areas, as well as fluoride from other fluorinated medications?

Fluorinated pesticides

For decades prior to community water fluoridation, fluoride was commonly used as rodenticides, insecticides, wood preservatives, etc. It is still used for many industrial applications as well as in rodenticides, insecticides, and agricultural sprays. (See attachment re_*Environmental_Adverse effects of fluorinated and fluoride pesticides.*)

Prior to being used as a fluoridating agent in Melbourne's community water supply, sodium silicofluoride was registered under Victoria's 1958 Pesticides Act and was a known active ingredient in the slug and snail killer "Snaleen".

The older use of fluorides as pesticides does not justify accepting them as 'existing chemicals' in terms of human consumption.

Fluoride in fertilizer

One has only to read the analysis of fertilizers to note the amount of high percentage of fluoride in some; along with arsenic, lead, cadmium and mercury which are all accumulative in the soil and harmful to the environment. In fact some of these fertilizers warn against their regular use because of the potential for these toxins to build up in the soil.

Fluoridation unnecessarily adds more fluoride into the environment. The idea that fluoride reduces tooth decay has led many scientists not to consider the effects of fluoride on human and biological health but rather to blindly accept that 1ppm fluoride in water has been shown to be 'safe'. I submit that fluoride is not biologically safe and that fluoridation only serves to maintain the illusion that it is.

Fluoride for various applications (commercial, industrial, rural)

Some commercial, industrial and rural uses for fluoridating compounds include: for disinfecting fermentation apparatus in breweries and distilleries; electrolytic refining of lead; electroplating; hardening cement; crumbling lime or brick work; removal of lime from hides during the tanning process; removal of moulds; timber preservative; insecticides; rodenticides; as a remedy for the destruction or elimination of intestinal worms (anthelmintic); as an agent to destroy lice (pediculicide); as an agent that kills ticks and mites (acaricide); as a constituent of vitreous enamel and glass mixes; as a steel deglassing agent; in electroplating; in fluxes; in heat-treating salt compositions; in preserving wood; in pastes and mucilages; in the manufacture of coated paper; frosting glass and for the removal of hydrogenfluoride from exhaust gases to reduce air pollution. A sinister aspect of sodium fluoride which is still used in some water supplies and toothpaste is its use as a major ingredient in the manufacture of sarin gas.

This is just a sample of the industrial uses of fluoride and therefore the sources of fluoride in the modern environment. It is unacceptable to continue adding fluoride to water supplies on the assumption that modern life does not involve much more fluoride exposure than we have ever been exposed to before. It is negligent for governments to continue to promote fluoridation while also not undertaking any monitoring of the incidence or effect of fluoride consumption.

Dental crisis looming

NSW Health's shortsightedness in considering fluoridation to be the answer to dental problems is irresponsible to the extreme. NSW Health is directing considerable sums of money into fluoridating (and the advertised promotion of fluoridation) on the grounds that fluoridation helps reduce tooth decay. But the published evidence shows that fluoride consumption is more likely to increase the incidence of dental fluorosis and therefore the need for dental services. Instead of adding to the cumulative problem NSW Health should surely be directing these funds into upgrading dental

services (including, recruiting suitably-qualified overseas dentists AND subsidising the dental training of adequate numbers of Australians so that the future dental needs of the public can be met by dental professionals). To not do so, will be to lead the people of NSW into a danger zone of a massive undersupply of dentists and dental services. Australiawide, dentists are increasingly in short supply. It seems apparent that the federal government has no intention of re-introducing dental subsidies to states. State premiers must show leadership now to avert a dental disaster of major proportions as well as lobbying for the introduction of Dental Medicare.

Sustainability

Sustainability is the practice of foreseeing potential problems and then implementing measures to prevent them happening. NSW Health appears to be blinkered when it comes to gauging the effects of projected decades of highly-toxic runoff from fluoridating agents and, also, the long-term negative effects of relying upon non-effective fluoridation to protect people's dental health. Instead, NSW Health should be funding and fast tracking students into universities for dental training, as well as promoting preventive measures such as oral hygiene and healthy diets as a priority.

The environment is not a limitless sink to remain unchanged regardless of mankind's thoughtless acts towards it. It is a sustainer of life for us all, from the infinitesimally small to the largest creatures on earth. All of these are as dependent upon the health of the environment as man is dependent for life on oxygen. Coastal vegetation and waterways will more and more become the recipient of all sorts of undesirable man-made pollutants such as sewage, pesticides, fertilizers, medications, household cleaners, and other harmful compounds; while the ocean is the ultimate receptacle for receiving the run-off from waterways which also drain the coastal vegetation and upstream polluting materials.

Conclusion

I urge you most strongly to advise the NSW Government to redirect funding away from fluoridation schemes and into the training of an adequate number of dentists, as well as the provision of dental care for the financially disadvantaged. Fluoridation should not be continued in the absence of even the most basic of health and safety studies.

I urge you most strongly to give very serious consideration to the long-term effects of fluoride build-up in the coastal and marine environment from all sources; and its potential for adverse effects on all biological life - most especially our children.

I do not require this submission to be treated as confidential. Please contact me for clarification of any points. Please advise me of the outcome of your Inquiry.

Yours truly,

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(See http://www.fluoridealert.org/pesticides/effects.environmental.q-z.htm for this article online)

Environmental - Adverse Effects
Fluorinated and Fluoride Pesticides
beginning with **A-E** • **F-G** • **H-P** • **Q-Z**

Note: This is not an exhaustive list.
When time allows more information will be added.

Quinoxyfen - **Fungicide** - CAS No. 124495-18-7

Examination and possible vote on a draft Commission Decision making it possible for Member States to extend provisional authorisations granted for the new active substances carfentrazone, cinidon-ethyl, cyhalofop-butyl, ethoxysulfuron, famoxadone, flazasulfuron, flufenacet, flumioxazine, flurtamone, fosthiazate, isoxaflutole, metalaxyl-M, Pseudomonas chlororaphis, quinoxyfen, Spodoptera exigua nuclear polyhedrosis virus and sulfosulfuron (SANCO/3963/2001 rev 6).

... Sweden supports a decision of non-inclusion of quinoxyfen in Annex I to Directive 91/414/EEC. The possible environmental impact of quinoxyfen cannot be shown to be acceptable with sufficient security. This is due to the high persistence, high potential for bioaccumulation and indicated potential for long-range transport. Substances like quinoxyfen may accumulate in various environmental compartments, including biota, and the effects of such accumulation are unpredictable. The Swedish opinion is that whatever the final results of the additional, ongoing study on organic matter breakdown may be, those results cannot be sufficient to demonstrate an acceptable impact.
Ref: SHORT REPORT OF THE MEETING OF THE STANDING COMMITTEE ON PLANT HEALTH HELD ON 7 DECEMBER 2001 IN BRUXELLES. SCPH 5/01. European Commission.
http://www.fluorideaction.org/pesticides/quinoxyfen.eu.dec.7.2001.pdf

Silafluofen - **Insecticide** - CAS No. 105024-66-6

Classified as a "severe marine pollutant".
Ref: Transport Canada. APPENDIX 1 MARINE POLLUTANTS. Online March 24, 2003.
http://www.tc.gc.ca/acts-regulations/general/t/tdg/regulations/tdg001/part_2.htm

Sodium Fluoride - **Wood Preservative, US EPA List 4B Inert** - CAS No. 7681-49-4

Effects of Fluoride on Fish Passage. The upstream migration of adult spring chinook salmon in the Columbia River has been subject to unusually long delays

at John Day Dam. During the spring migration period, average passage times for radio-tagged salmonids at John Day Dam were 158 and 156 hours in 1979 and 1980, respectively. In contrast, average passage time at Bonneville Dam was less than 48 hours and at The Dalles Dam it was less than 24 hours. In addition, passage times for salmonids in the fall of 1982 were twice as long at John Day Dam as they were at The Dalles and McNary Dams. The delay of nearly 1 week at John Day Dam appeared to contribute to increased mortality and may have affected the spawning success of migrating adult salmonids.

...In 1982, preliminary studies conducted by CZES Division personnel assessed the distributions of many pollutants near John Day Dam. The results of this investigation suggested that the fish-passage delays might be related to contaminants discharged at an aluminum smelter outfall located on the Washington shore 1.6 km upstream from John Day Dam. In particular, high concentrations of fluoride in the vicinity of John Day Dam (0.3-0.5 mg/L in 1982) prompted investigators to focus sampling and research efforts on this contaminant.

In 1983 and 1984, behavior tests were conducted in which over 600 returning salmonids (chinook, coho, and chum, O. keta, salmon) were captured and tested with different concentrations of fluoride in a two-choice flume located in the spawning channel of Big Beef Creek, Washington. The conclusion from these experiments was that the behavior of upstream-migrating adult salmon would be adversely affected by fluoride concentrations of about 0.5 mg/L and that concentrations of 0.2 mg F/L were at or below the threshold for fluoride sensitivity of chinook and coho salmon.

Beginning in 1983 and continuing through 1986, fluoride discharges from the aluminum plant were greatly reduced. This was initially due to modifications in the plant's pollution-discharge system. However, it was also during this period that the Washington Department of Ecology (WDOE) took an active interest in the results of the CZES Division's water quality and behavior tests. The WDOE lowered significantly the discharge limitations for a number of contaminants, including fluoride, in the aluminum plant's wastewater discharge permit. With the reduction in fluoride discharged from the aluminum plant, there was a corresponding drop in fluoride concentrations in the river near the outfall and John Day Dam. Concurrently, fish passage delays and interdam losses of adult salmon decreased to acceptable levels.
Ref: April 1993. NOAA Technical Memorandum NMFS-NWFSC-7. Coastal Zone and Estuarine Studies Division. RESEARCH ACTIVITIES AND ACCOMPLISHMENTS. 1980-89. Edited by Douglas B. Dey. National Marine Fisheries Service, Northwest Fisheries Science Center, Coastal Zone and Estuarine Studies Division, 2725 Montlake Blvd. E., Seattle WA 98112

[Earthworms]. The impact of four fluorides (NaF, KF, FCH2COONa [Sodium fluoroacetate] and CaF2) in sublethal concentrations on the earthworm Eisenia fetida was

investigated (model experiments) in relation to its growth, maturity (clitellum-development) and fertility (number of cocoons and number of hatchlings). Fluoride-accumulation was determined at the end of the 22 weeks' test period. In higher concentrations NaF, KF and FCH2COONa reduced growth of E. fetida significantly. CaF2 had no effect. The maturity was delayed through higher concentrations of NaF and KF in the substrate. In the case of CaF2, most worms had a fully developed clitellum. Most cocoons were found in the experiments with FCH2COONa. Small concentrations of NaF, KF an FCH2COONa obviously raised cocoon-numbers, whereas higher concentrations of NaF and KF reduced it. Only NaF reduced the number of hatchlings per cocoon significantly. At the end of the test, all worms from the variants with NaF, KF and CaF2 had a significantly higher fluoride [abstract truncated]
Ref: Influence of different fluorides in sublethal concentrations on growth, fertility and fluoride-accumulation of Eisenia foetida (Oligochaeta, Lumbricidae). [Earthworm]; by VOGEL J, OTTOW J CG. PEDOBIOLOGIA; 36 (2). 1992. 121-128. [From Toxline at Toxnet].

Sodium fluoroacetate (Compound 1080) - **Insecticide, Rodenticide** - CAS No. 62-74-8

Measured oral LD 50 values of fluoroacetate in the house sparrow, redwinged blackbird, starling and golden eagle are 3.0, 4.22, 2.37, and 1.25 to 5 mg/kg, respectively. In addition, measured acute toxicity data for mammalian wildlife include an oral LD50 of 0.22 to 0.44 mg/kg for mule deer, an oral LD50 of 1.41 mg/kg for male ferrets, and an oral LD 50 of 0.5 to 1.0 mg/kg for bears. EPA believes that there is sufficient evidence for listing sodium fluoroacetate on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the environmental toxicity data for this chemical.
Ref: USEPA/OPPT. Support Document for the Health and Ecological Toxicity Review of TRI Expansion Chemicals. U. S. Environmental Protection Agency, Washington, DC (1993). As cited by US EPA in: Federal Register: January 12, 1994. Part IV. 40 CFR Part 372. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right-to-Know; Proposed Rule.

Sodium fluoroacetate (1080), (also known as sodium monofluoroacetate), is a fluorinated carboxylic acid ester with high to very high toxicity to birds and mammals.
Ref: Australia. National Registration Authority for Agricultural and Veterinary Chemicals. Reconsideration of products containing Sodium fluoroacete (1080) and their labels. Background to the Review and Scope Document.
http://www.fluorideaction.org/pesticides/1080.australia.july.2002.pdf

Sulfentrazone - **Herbicide** - CAS No. 122836-35-5

Phototoxic Pesticide. Light-dependent peroxidizing herbicides (LDPHs). US EPA identified the organofluorine herbicides Acifluorfen, Azafenidin, Carfentrazone-ethyl, Flumiclorac-penty, Flumioxazin, Fluthiacet-methyl, Fomesafen, Lactofen,

Oxadiargyl, Oxadiazon, Oxyfluorfen, Sulfentrazone, Thidiazimin as phototoxic pesticides that act by inhibiting protoporphyringen oxidase in the heme and chlorophyll biosynthetic pathway. [10 out of the 13 pesticides that EPA identified are organofluorines].
SEE http://www.fluoridealert.org/pesticides/PHOTOTOXICITY.PAGE.htm
Ref: December 11, 2001 - US EPA. Revised Environmental Fate and Effects Division Preliminary Risk Assessment for the Oxyfluorfen Reregistration Eligibility Decision Document (also at: http://www.epa.gov/oppsrrd1/reregistration/oxyfluorfen/oxyefedchap.pdf).

-- Environmental Characteristics. Acceptable information from environmental fate studies with respect to the persistence and mobility of sulfentrazone under laboratory and field conditions has been reviewed. Based on the current environmental fate data base, sulfentrazone has the following characteristics: 1) moderately soluble, 2) not susceptible to hydrolysis, 3) extremely susceptible to direct photolysis in water, 4) very stable to photolysis on soil, 5) aerobic half-life of 1.5 years, 6) anaerobic half-life of 9 years, 7) very high mobility in soil (average Koc = 43, Kd < 1), and 8) low volatility from soils and water. With these properties, it appears that sulfentrazone is highly mobile and persistent, and has a strong potential to leach into groundwater and move offsite to surface water.
-- Potential to Contaminate Groundwater. A groundwater exposure estimate for sulfentrazone was conducted based on findings from a prospective groundwater monitoring study in North Carolina. Although the study was incomplete, enough data were collected to confirm that sulfentrazone leaches substantially to groundwater in areas with sandy soils.
-- Aquatic. Sulfentrazone is practically non-toxic to the rainbow trout (LC50) greater 120 ppm) and slightly toxic to the bluegill sunfish (93.8 ppm). The results indicate that sulfentrazone is slightly toxic to fish on an acute basis. The chronic results indicate that sulfentrazone significantly affects young fish survival and growth at aquatic concentrations as low as 5.93 ppm. Sulfentrazone is slightly toxic to aquatic invertebrates on an acute basis. The results from data from chronic freshwater invertebrates indicate that survival of young daphnids is adversely affected at sulfentrazone concentrations as low as 0.51 ppm. The results from acute estuarine and marine animals study are incomplete but indicate that sulfentrazone is highly toxic to estuarine/marine organisms.
Ref: US EPA. Pesticide Fact Sheet. Sulfentrazone Reason for Issuance: Registration of a New Chemical Date Issued: February 27, l997.
http://www.epa.gov/opprd001/factsheets/sulfentrazone.pdf

Sulfluramid - Acaricide, Insecticide - CAS No. 4151-50-2

As a class, fluorinated organic compounds are resistant to photolysis. If released to soil, sulfluramid is expected to have no mobility based upon an estimated Koc of 3.5X10+6. Volatilization from moist soil surfaces is expected to be an important fate process based upon an estimated Henry's Law constant of 5.4 atm-cu m/mole. However, adsorption to soil is expected to attenuate volatilization. As a class, fluorinated organic compounds are resistant to microbial degradation. [Giesy JP, Kannan K; Environ Sci Technol 36: 147A-152A (2002)] If

released into water, sulfluramid is expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilization from water surfaces is expected to be an important fate process based upon this compound's estimated Henry's Law constant. However, volatilization from water surfaces is expected to be attenuated by adsorption to suspended solids and sediment in the water column. The estimated volatilization half-life from a model pond is 107 years if adsorption is considered. An estimated BCF of 500 suggests the potential for bioconcentration in aquatic organisms is high.
Ref: Hazardous Substances Data Bank for SULFLURAMID CASRN: 4151-50-2.
http://www.fluorideaction.org/pesticides/sulfluramid.hsdb.oct.2003.htm

Ref: Acute Aquatic Ecotoxicity Summaries for Sulfluramid on All Taxa Groups . PAN Pesticides Database - Chemical Toxicity Studies on Aquatic Organisms.
http://www.pesticideinfo.org/List_AquireAcuteSum.jsp?Rec_Id=PC34496

Common Name	Scientific Name	Avg Species LC50 (ug/L)	LC50 Std Dev	Number of Studies	Avg Species Rating
Fish					
Rainbow trout,donaldson trout	Oncorhynchus mykiss	210.0	-	1	Highly Toxic
Fathead minnow	Pimephales promelas	5,054	4,866	2	Moderately Toxic

tau-Fluvalinate - **Acaricide, Insecticide** - CAS No. 102851-06-9

-- Considerable accumulation of tau fluvalinate was observed in wax. Depending on the location of the samples tau fluvalinate concentrations varied from 0.2 mg/kg - 5.5 mg/kg, with a maximum of 26.9 mg/kg in one wax sample collected from a frame positioned next to a strip.
-- Accumulation in wax is the result of the stability of tau fluvalinate in this matrix, its lipophilic character and the fact that wax is normally reused over several seasons. Monitoring of tau fluvalinate residues in honey and wax in Belgium in 1989-1992 revealed that residues in wax increased exponentially when the wax was reused over the years. Transfer of tau fluvalinate residues from wax to honey was shown to be negligible. However, the high tau fluvalinate residues in wax should be taken into consideratin in the evaluation of tau fluvalinate since contamination of honey with wax particles has to be expected (0.5 % content of water insoluble particles in honey is allowed).
Ref: Revised Summary Report. EMEA/MRL/021-REV1/95. Committee for Veterinary Medicinal Products. The European Agency for the Evaluation of Medicinal Products.
http://www.fluorideaction.org/pesticides/tau.fluvalinate.1995.review.pdf

-- Environmental hazards:

Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
-- Ecological Information:
Very toxic to aquatic organisms.
Ecotoxicity:
Fish: LC 50 > 0.024 mg/l, 96 hours (rainbow trout).
Algae: EC 50 = >100 mg/l (72 hours).
Daphnia: LC 50 = 0.011 mg/l (48 hours)
Ref: Material Safety Data Sheet for Klartan. : Code no. R-10834.G. Makhteshim Agan (UK) Limited Unit 16, Thatcham Business Village, Colthrop Way, Thatcham Berkshire RG19 4LW.
http://www.fluorideaction.org/pesticides/tau.fluvalinate.msds.klarta.pdf

Teflon (PTFE: polytetrafluoroethylene) - EPA List 3 Inert - CAS No. 9002-84-0

Due to length, see special section on
- Teflon's Thermal Decompostion Products
- Birds

Teflubenzuron - Insecticide - CAS No. 83121-18-0

TOXIC chemicals used on salmon farms could be killing off key elements of the marine food chain, according to a report leaked to a leading scientific magazine. "New Scientist" magazine has obtained a copy of a 178-page report which forms part of the ongoing £4 million study into the industry, which was launched by the UK government in 1999. In the leaked document it is alleged that chemicals such as cypermethrin, azamethiphos or teflubenzuron are damaging small crustaceans and other marine wildlife, which could be crucial to the survival of other species. These chemicals are often used by farmers to rid fish of sea lice.
- See also April 25, 2002 press release from Friends of the Earth, Scotland.
Ref: Leaked Report Claims Toxins Are Hitting Marine Food Chain Fish Farming Today- Fish Farming Today. April 25, 2002

-- There is very little information available on the environmental fate and ecological effects of teflubenzuron in aquatic environments. The specific mode of action of teflubenzuron means it is highly toxic to aquatic crustacean invertebrates, but low in toxicity to fish, mammals and birds. As with emamectin benzoate, it is likely that the sediments will act as a sink for teflubenzuron and so sediment associated organisms are more likely to be affected by this chemical.
-- It is difficult to predict the ecological risk of teflubenzuron to the marine environment because of the current lack of information. Results from field studies referred to in SEPAÕs environmental risk assessment suggest that the use of teflubenzuron for sea lice control may present a moderate to high environmental risk. It seems unlikely that teflubenzuron will be widely used for sea lice control in Scotland, but if use does increase, investigation into the potential long-term

impacts of this chemical on the marine environment is recommended.
Ref: REVIEW AND SYNTHESIS OF THE ENVIRONMENTAL IMPACTS OF AQUACULTURE. The Scottish Association for Marine Science and Napier University. Scottish Executive Central Research Unit. 2002.
http://www.scotland.gov.uk/cru/kd01/green/reia.pdf

Although teflubenzuron is relatively non-toxic to most marine species (e.g. fish, algae, shellfish), it is potentially highly toxic to any species which undergo moulting within their life cycle. This will therefore include some commercially important marine animals such as lobster, crab, shrimp and some zooplankton species.
-- Subsequent chemical analysis confirmed that measurable concentrations were generally not present in water after treatment and that levels in sediments were variable but followed the predicted dispersion model with measured levels of teflubenzuron extending initially to about 50m from cages in line with the main direction of current. In the study, teflubenzuron was found to persist longer than 6 months, which was longer than expected and hence additional studies were commissioned by Nutreco at the request of SEPA and VMD. The predicted half-life of teflubenzuron in sediment was from 8 to 92 days depending on sediment type (Myrvold, 1997) and it had been expected that 90% of teflubenzuron should have been degraded within 6 months. The indication of a potential for longer persistence is attributed to the site being a ieworst casels site already enriched and impacted by organic wastes with teflubenzuron being retained by binding with organic material (Trouw, 1999). The results from long-term site monitoring finally reported a half life of 104 to 123 days (Trouw, 1999). From this data, SEPA now intend to apply a half life of 115 days as a decay factor when undertaking site loading calculations for consent applications or reviews.
-- Chemical analysis of samples collected on-site of indigenous crustacea was also undertaken. It was concluded that there was a risk that sediment dwelling crustacea, such as edible crab (Cancer) and possibly Norwegian lobster (Nephrops), may accumulate teflubenzuron from contaminated sediment. However, it is known that depuration and loss of teflubenzuron does proceed following initial exposure and uptake (McHenery, 1997) and hence levels may be lost from such species before toxic effects occur (moulting).
-- The half-life of teflubenzuron in sediment suggests that there is a moderate risk of build up in sediment through repeat applications, although the risk of this is reduced where fewer applications are required by correct use of the product strategy.
Ref: Calicide (Teflubenzuron) - Authorisation for use as an in- feed sea lice treatment in marine cage salmon farms. Risk Assessment, EQS and Recommendations (As agreed at the Board Meeting held on 7 th July, 1998 and subsequently updated in July, 1999). Policy No. 29. SCOTTISH ENVIRONMENT PROTECTION AGENCY. Fish Farming Advisory Group.
http://www.fluorideaction.org/pesticides/teflubenzuron.scotlandepa99.pdf

Tefluthrin - **Insecticide** - CAS No. 79538-32-2

Aquatic acute toxicity values for tefluthrin include a rainbow trout 96-hour LC 50 of 0.06 ppb, a bluegill 96-hour LC50 of 0.13 ppb, a sheepshead minnow 96-hour LC50 of 0.13 ppb, a daphnid 48-hour EC50 of 0.07 ppb, and a mysid 96-hour EC 50 of 0.053 ppb. EPA believes that there is sufficient evidence for listing teflurin on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data for this chemical.
Ref: USEPA/OPP. Support Document for the Addition of Chemicals from Federal Insecticide, Fungicide, Rodenticide Act (FIFRA) Active Ingredients to EPCRA Section 313. U. S. Environmental Protection Agency, Washington, DC (1993). As cited by US EPA in: Federal Register: January 12, 1994. Part IV. 40 CFR Part 372. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right-to-Know; Proposed Rule.

Ref: Acute Aquatic Ecotoxicity Summaries for Tefluthrin on All Taxa Groups. PAN Pesticides Database - Chemical Toxicity Studies on Aquatic Organisms
http://www.pesticideinfo.org/List_AquireAcuteSum.jsp?Rec_Id=PC34532

Common Name	Scientific Name	Avg Species LC_{50} (ug/L)	LC_{50} Std Dev	Number of Studies	Avg Species Rating	Outlier Result for Organism Group?
Fish						
Bluegill	Lepomis macrochirus	4.47	4.34	2	Very Highly Toxic	
Rainbow trout,donaldson trout	Oncorhynchus mykiss	3.78	3.72	2	Very Highly Toxic	

1,1,1,2-Tetrafluoroethane (HFC-134a) - **Propellant, US EPA List 4B Inert** - CAS No. 811-97-2

The atmospheric lifetime of 1,1,1,2-tetrafluoroethane has been estimated to range from 12.5 to 24 years. 1,1,1,2-Tetrafluoroethane may also undergo atmospheric removal by wet deposition processes; however, any removed is expected to rapidly re-volatilize to the atmosphere.
Ref: Hazardous Substances Data Bank for 1,1,1,2-TETRAFLUOROETHANE CASRN: 811-97-2.
http://www.fluorideaction.org/pesticides/1,1,1,2-tetrafluoroe.toxnet.htm

TFM (3-Trifluoro-Methyl-4-Nitro-Phenol) - Lampricide, Piscicide - CAS No. 88-30-2

Abstract: Toxicity testing with fish began early in this century, but standardized methods have been developed only within the last three decades. Standardized test procedures promote reproducibility of results; healthy fish properly handled and acclimated to test conditions are a given prerequisite. The principles of acute toxicity testing are important in the design of chronic tests for suspected carcinogens because certain factors influence the activity of chemicals or contaminants. The pH of test water is a critical factor in governing the uptake of

chemicals by fish. Buffering is required so that uniform pH in waters of different hardnesses and different pHs in water of a given hardness are maintained. The importance of water quality control is graphically demonstrated by the lampricide 3-trifluoromethyl-4-nitrophenol; the toxicant is over 50 times more toxic in water at pH 6.5 than at pH 9.5. Results of laboratory tests on toxicity or carcinogenicity of single compounds in a clean environment represent an oversimplification of the real world because organisms are actually exposed to multiple chemicals or stresses. Because the environment is a complex interaction of physical, chemical, and biological factors that are extremely variable and dynamic, simulation of these systems in the laboratory is, at best, artificial; therefore, results developed must be considered to be predictive.
Ref: Marking LL (1984). Procedures for use of freshwater fishes in the development of reproducible toxicological information. Natl Cancer Inst Monogr 1984 May;65:195-9
http://www.ncbi.nlm.nih.gov:80/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=6749253&dopt=Abstract

-- TFM is chemically and biologically very stable. The compound possesses many of the chemical features known to impart persistence to organic compounds... TFM was converted to reduced-TFM with a half-life of less than one week under both aerobic and anaerobic aquatic metabolism conditions. It must be stressed that when reduced-TFM is reported as a reaction product, degradation has not occurred. TFM has just undergone a chemical reduction and under appropriate conditions, reduced-TFM may be re-oxidized to TFM... TFM is expected to remain in solution in the lake system and persist for long periods of time... TFM (C7 H4 F3 NO3 ; M.W. 207.11) is chemically and biologically very stable. An examination of its structure, i.e., aromatic, fluoro-containing, m-substituted phenol, shows that the compound possesses many of the chemical features known to impart persistence to organic compounds. Its pKa is 6.07 and the effect of pH on the toxicity appears to follow closely to the concentration of the lipid-soluble, free phenol form of TFM. This pH sensitivity is used to maximize effectiveness. As pH increases, toxicity, bioaccumulation, and adsorption to sediment decrease. Aqueous solubility of the sodium salt is 5 g/L.[P 24-25].
Ref: Reregistration Eligibility Decision (RED) 3-Trifluoro-Methyl-4-Nitro-Phenol and Niclosamide. US EPA, Office of Prevention, Pesticides And Toxic Substances (7508C). Report No. EPA 738-R-99-007. November 1999.
http://www.fluoridealert.org/PESTICIDES/TFM.Red.1999.pdf

• Note from FAN: The use of TFM in the Great Lakes began at the end of the 1950's. Since that time there has been a dramatic shift from male to female sea lampreys. A confounding factor may be that TFM was found to be contaminated with dioxin in the early 1990s. It is unclear to me whether dioxin influenced this alteration, or TFM alone, or the combination of TFM and dioxin. [EC, Sept. 2003]

-- TFM treatments have been associated with induction of hepatic mixed function oxyganase activity and altered levels of circulating steroids in fish and induced hepatic vitellogenesis in primary cultures of rainbow trout hepatocytes (Hewitt et al. 1997). As such, TFM acts as an estradiol agonist and has a demonstrated

endocrine disrupting effect...
-- Abundance of sea lamprey peaked in several Great Lakes before chemical control began. The sex ratio in these peak populations were predominately males (68-71%). Following a decade of lampricide treatments, populations of sea lampreys showed marked declines and the sex ratios in these populations shifted toward a predominance of females accounting for 72% of the population (Henrich, et al, 1979). This publication by Henrich concludes that lampricides reduced the populations of sea lampreys in the Great Lakes and contributed to the sequential shifting of the sex composition from a predominance of males to a predominance of females. There are no data to support that the endocrine mediated effect associated with TFM is related to the observed sex-ratio shifts among TFM-treated populations of sea lamprey [page 23].
Ref: November 1999 US EPA's Reregistration Eligibility Decision (RED) for 3-Trifluoro-Methyl-4-Nitro-Phenol and Niclosamide).
http://www.fluoridealert.org/PESTICIDES/TFM.Red.1999.pdf

... The sensitivity of mudpuppies, frog tadpoles, and adult frogs to use of 3-trifluoromethyl-4- nitrophenol (TFM) in the Great Lakes has been noted on many occasions. TFM has been used annually since 1958 for the control of sea lampreys throughout the Great Lakes. Amphibians regularly have been found dead in creeks immediately after TFM treatment (Gilderhus and Johnson 1980, Matson 1990). Laboratory tests have confirmed that species native to the Great Lakes Basin, such as the grey tree frog, northern leopard frog, and bullfrog, are sensitive to levels of TFM used for sea lamprey control (Chandler and Marking 1975). Mudpuppy population size decreased by a minimum of 29 per cent after a spray event in the Grand River of Ohio (Matson 1990)...
Ref: Conservation Priorities for the Amphibians and Reptiles of Canada. Sept 2000 report published by World Wildlife Fund Canada and Canadian Amphibian and Reptile Conservation Network. Prepared by David Seburn and Carolyn Seburn.

Abstract. Since 1958, 3-(trifluoromethyl)-4-nitrophenol (TFM) has been used to control the sea lamprey (Petromyzon marinus) in the USA-Canadian Great Lakes Superior, Michigan, Huron and Ontario. A study was conducted to determine the degradability of TFM under laboratory conditions in aqueous (sediment-free) systems. No evidence of microbial degradation of TFM was found up to 80 days. TFM may persist for extended periods of time in the Great Lakes. On the basis of past and present levels of usage of TFM, it was estimated that the concentration of TFM in Lake Superior water could approach, if TFM were a completely conservative chemical, 0.015 mug/l. This concentration was considerably less than the concentrations found to be acutely and chronically toxic to aquatic life. The use of TFM as a sea lamprey larvicide did not represent a hazard to fish and other nontarget aquatic life in the Great Lakes.
Ref: THINGVOLD DA et al. (1981). Persistence of 3-(trifluoromethyl)-4-nitrophenol in aquatic environments. ENVIRON SCI TECHNOL; 15 (11). 1335-

1340.

Excerpts from Technical Report 7. April 2000. Animal Deformities or Reproduction Problems. Prepared for the Lake Erie LaMP Preliminary Beneficial Use Impairment Assessment. Keith A. Grasman, Lead Author. Co-Authors: Christine A. Bishop, William W. Bowerman, James P. Ludwig, Pamela A. Martin.
http://www.epa.gov/glnpo/lakeerie/buia/lamp7.pdf

(page 13): The sensitivity of mudpuppies, frog tadpoles and adult frogs to TFM use in the Great Lakes has been noted (Gilderhus and Johnson, 1980). TFM is intended to control larval sea lamprey and has been used historically in 19 (8 in U.S./11 in Canada) of the 842 tributaries to Lake Erie for sea lamprey (Petromyzon marinus) control. Since 1995, TFM has been applied in Conneaut Creek and the Grand River in Ohio and Big Creek and Big Otter Creek in Ontario. Only four Lake Erie tributaries (Big Creek Ontario, and 3 U.S. tributaries) are currently scheduled for future regular treatments every 4 to 6 years.

When TFM is used, amphibians have regularly been found dead in creeks immediately after treatment in Lake Erie watersheds and elsewhere in the Great Lakes (Gilderhus and Johnson, 1980; Matson, 1990). Laboratory tests have confirmed that species native to the Great Lakes basin such as gray tree frog, leopard frog, and bullfrog are sensitive to field applied rates of TFM (Chandler and Marking, 1975). In the Grand River, Ohio, Matson (1990) found that in the year following TFM application (1997), mudpuppy population size decreased by a minimum of 29% in the segment treated. In 1999, the Grand River was treated with TFM and dead mudpuppies were found downstream of the application zone within twenty-fours hours.

Because TFM is not bioaccumulative and is only applied periodically in closely controlled and monitored conditions, the associated mudpuppy mortality is often perceived to be insignificant. However, mudpuppies do not become sexually mature until 4 to 6 years of age. Given the past and projected future schedule for TFM applications, there is the potential for the TFM applications to match periods when large numbers of mudpuppy are reaching an age when they can reproduce. In addition, TFM is generally applied in the spring when stream flows are higher. Therefore, TFM has the potential to kill a portion of the existing females before they lay their eggs in May and June. For these reasons, future study is needed to determine the significance of the mortality and the life stages most affected (see section 7.5).

(page 16) • There are conflicting opinions about the significance of non-target species sensitivity, particularly mudpuppy, to TFM (when used for sea lamprey eradication), and its implications for potential impairment. Therefore, the impact of TFM on amphibian populations needs to be assessed by monitoring populations of mudpuppies and other amphibians pre- and postapplication. From a reproductive standpoint, it is particularly important to determine if TFM has greater impacts on certain age classes and/or egg-bearing females.

Thiazopyr - Herbicide - CAS No. 117718-60-2

Freshwater Fish: moderately toxic
-- Rainbow trout: LC50 = 3.4 mg/L
-- Bluegill Sunfish: LC50 = 3.5 mg/L

-- Aquatic Invertebrate: moderately toxic Daphnia magna: LC50 = 6.1 mg/L
-- Mollusc Shell Deposition: highly toxic Eastern Oyster: EC50 = 0.82 mg/L
-- Estuarine Invertebrate Acute Toxicity: Moderately toxic Mysid Shrimp:LC50 = 2.0 mg/L Fish Early Life Stage Toxicity Rainbow trout: NOEL = 0.55 mg/L MATC = 0.74 mg/L
-- Aquatic Invertebrate Life Cycle Toxicity Daphnia magna: NOEL = 0.11 mg/L MATC = 0.16 mg/L Aquatic Plant Growth and Reproduction Selenastrum capricornutun:EC50 = 0,043 mg/L NOEL = 0.018 mg/L
-- Thiazopyr had low toxicity to birds, mammals, honeybees, and earthworms. It was moderately toxic to freshwater and marine fish and Daphnia magna, with moderate to high toxicity to marine invertebrates. Thiazopyr was highly to very highly toxic to nontarget terrestrial and aquatic plants, algae and diatoms.
-- Photodegradation on soil: Thiazopyr degrades very slowly in soil, with an extrapolated half life of 1373 days.
Ref: US EPA. Pesticide Fact Sheet. Thiazopyr Reason for Issuance: Registration of a New Chemical Date Issued: February 20, l997.
http://www.epa.gov/opprd001/factsheets/thiazopyr.pdf

Thidiazimin - **Herbicide** - CAS No. 123249-43-4

Phototoxic Pesticide. Light-dependent peroxidizing herbicides (LDPHs). US EPA identified the organofluorine herbicides Acifluorfen, Azafenidin, Carfentrazone-ethyl, Flumiclorac-penty, Flumioxazin, Fluthiacet-methyl, Fomesafen, Lactofen, Oxadiargyl, Oxadiazon, Oxyfluorfen, Sulfentrazone, Thidiazimin as phototoxic pesticides that act by inhibiting protoporphyringen oxidase in the heme and chlorophyll biosynthetic pathway. [10 out of the 13 pesticides that EPA identified are organofluorines].
SEE http://www.fluoridealert.org/pesticides/PHOTOTOXICITY.PAGE.htm
Ref: December 11, 2001 - US EPA. Revised Environmental Fate and Effects Division Preliminary Risk Assessment for the Oxyfluorfen Reregistration Eligibility Decision Document (also at: http://www.epa.gov/oppsrrd1/reregistration/oxyfluorfen/oxyefedchap.pdf).

Tolylfluanid - **Fungicide** - CAS No. 731-27-1

Water-sediment systems. In a study of hydrolysis, tolylfluanid was readily hydrolyzed into DMST [dimethylaminosulfotoluidine] under all conditions used (pH 4, 7 and 9; 20, 30 and 40°C). The half life of tolylfluanid was calculated to be 11.7 days at pH 4 and 29.1 hours at pH 7 at 22°C in respective sterile buffer solutions. Tolylfluanid was so unstable at pH 9 that no parent compound was left to be detected even in immediate analysis of the sample making the estimation of half life impossible. Another hydrolysis study demonstrated that tolylfluanid was hydrolyzed into DMST, fluoride ion, chloride ion, sulfur and carbon dioxide. DMST, on the other hand, was stable at pH 4, 7 and 9 up to 55°C in respective sterile buffer solutions. The half life of DMST was calculated to be > 1 year at 22°C at pH 4, 7 and 9. (page 262)
Ref: Pesticide residues in food - 2002. Report of the Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and the WHO Core

Assessment Group on Pesticide Residues. Rome, Italy. 16- 25 September 2002. ISBN 92-5-104858-4.
http://www.fluorideaction.org/pesticides/tolylfluanid.fao.2002.pdf

Transfluthrin - Insecticide - CAS No. 118712-89-3

Environmental toxicity tests demonstrated that all products containing transfluthrin should be classified as 'Extremely dangerous to fish and other aquatic life', with a 48 h EC50 for Daphnia magna of 1.7 ug1 and a 96 h LC50 for rainbow trout (Oncorhynchus mykiss) of 0.7 ug 1.
Ref: Evaluation on: Transfluthrin Use as a Public Hygiene Insecticide. September 1997. Prepared by: the UK Health and Safety Executive, Biocides & Pesticides Assessment Unit, Magdalen House, Stanley Precinct, Bootle, Merseyside L20 3QZ. Available from: Department for Environment, Food and Rural Affairs, Pesticides Safety Directorate, Mallard House, Kings Pool, 3 Peasholme Green, York YO1 7PX. UK. Also at
http://www.pesticides.gov.uk/citizen/evaluations/165_confirm-box.htm
• Note: This was transcribed from the copy available on the web. While one can easily read this report on the web, the report is inaccessible, or locked, to any attempt to copy it. Any errors are mine. EC.

Tributyltin fluoride - Antifoulant, Fungicide, Microbiocide - CAS No. 1983-10-4

Tributyltin fluoride: Acute Aquatic Ecotoxicity Summaries for Tributyltin fluoride on All Taxa Groups.
Ref: PAN Pesticides Database - Chemical Toxicity Studies on Aquatic Organisms.
http://www.pesticideinfo.org/List_AquireAcuteSum.jsp?CAS_No=1983-10-4&Rec_Id=PC34614

Common Name	Scientific Name	Avg Species LC50 (ug/L)	LC50 Std Dev	Avg Species Rating
Amphibians				
Frog	Rana temporaria	30.0	-	Very Highly Toxic
Crustaceans				
Fiddler crab	Uca pugilator	800.0	-	Highly Toxic
Fish				
Channel catfish	Ictalurus punctatus	8.15	3.85	Very Highly Toxic
Bluegill	Lepomis macrochirus	7.35	3.65	Very Highly Toxic
Rainbow trout,donaldson trout	Oncorhynchus mykiss	11.2	7.80	Very Highly Toxic

Shell thickening. Alzieu et al. (1982) reported that adult oysters (Crassostrea gigas) developed gel centres in the shell when they were exposed to TBT fluoride at a concentration of 0.2 µg/litre.
Ref: C Alziieu et al. (1982). Influence des peintures antisalissures ˆ base d'organostanniques sur la calcification de la coquille de l'huitre Crassostrea gigas. Rev. Trav. Inst. P□ches Marit., 45: 101-116.
Ref: Tributyltin Compounds. Environmental Health Criteria 116. International Programme on Chemical Safety.
http://www.inchem.org/documents/ehc/ehc/ehc116.htm

Aquatic acute toxicity values for tributyltin fluoride include a bleak fish 96-hour LC 50 of 2.3 ppb, an algae 72-hour EC50 of 9.3 ppb, and a Harpacticoid copepod 96-hour LC 50 of 0.8 ppb. EPA believes that there is sufficient evidence for listing tributyltin fluoride on EPCRA section 313 pursuant to EPCRA section 313(d)(2)(C) based on the available environmental toxicity data.
Ref: USEPA/OPP. Support Document for the Addition of Chemicals from Federal Insecticide, Fungicide, Rodenticide Act (FIFRA) Active Ingredients to EPCRA Section 313. U. S. Environmental Protection Agency, Washington, DC (1993). As cited by US EPA in: Federal Register: January 12, 1994. Part IV. 40 CFR Part 372. Addition of Certain Chemicals; Toxic Chemical Release Reporting; Community Right-to-Know; Proposed Rule.

Trichlorofluoromethane (CFC 11) - **Insecticide, Fungicide, Propellant; EPA List 2 Inert** - CAS No. 75-69-4

US EPA: Class 1 Ozone Depleting Substance. Lifetime of Global Warming Potential: 45 years
Ref: http://www.epa.gov/ozone/ods.html

Many gases emitted as a result of industrial and agricultural activities can accumulate in the earth's atmosphere and ultimately contribute to alterations in the vertical distribution and concentrations of stratospheric ozone. Among the most important are those trace gases that have long residence times in the atmosphere. This allows accumulation in the troposphere and a gradual upward migration of the gases into the stratosphere where they contribute to depletion of stratospheric ozone layer. The atmospheric and chemical processes involved are extremely complex. Trace gases of particular concern include certain long lived

chlorofluorocarbons, such as CFC-11, CFC-12, and CFC-113. Since the transport of these gases to the stratosphere is slow, their residence times there are long, and the removal processes are slow, any effect on stratospheric ozone already seen is probably the result of anthropogenic emissions of these gases several decades ago. Those gases already in the atmosphere will continue to exert stratospheric ozone depletion effects well into the next century. /Chlorofluorocarbons/ [WHO; Environmental Health Criteria 113: Fully Halogenated Chlorofluorocarbons p.47 (1990)]
Ref: Hazardous Substances Data Base for TRICHLOROFLUOROMETHANE.
http://www.fluoridealert.org/pesticides/Trichlorofluorometha.TOXNET.htm

Environmental Contamination Concerns
A. Surface Water Volatilization from water surfaces is expected to be an important fate process with estimated volatilization half-lives for a model river and a model lake being four hours and five days, respectively. Hydrolysis is not expected to occur. Bioconcentration in organisms is low to moderate; BCF (Bioconcentration factor: the ratio of the chemical concentration in the organism to that in surrounding water) is from 11-86. Biodegradation, adsorption to sediment, and abiotic degradation are insignificant. Large volumes of Freon may sink to the bottom and gradually bubble up to the surface if the water is not too cold (Hazardtext, 2003B; HSDB, 2001A; HSDB, 2001B).
B. Groundwater In general, Freons that are spilled onto soil have the potential to leach into groundwater, because they do not bind well to soil (Hazardtext, 2003B; HSDB, 2001A; HSDB, 2001B). Fully halogenated hydrocarbons such as Freons 11, 12, and 113 are very resistant to chemical and biological degradation and are likely to be persistent contaminants if they reach groundwater.
D. Soil
If Freon is spilled onto soil, a portion may evaporate from the surface and the remainder will leach downward into the soil. Mobility through the soil is expected to be moderate based on estimated Koc values. Freon does not bind well to soil, and leaching to groundwater is possible (Hazardtext, Preliminary Remediation Goals for Residential Soil (U.S. EPA, 2002, Region IX):
Freon 11 - 390 mg/kg
Freon 12 - 94 mg/kg
Freon 113 - 5600 mg/kg
E. Air
Once released to air, Freon exists solely as a gas. In the atmosphere, fully halogenated Freons diffuse to the troposphere, where they are very stable and can be transported great distances. Wet deposition may result in some loss, but re-volatilization into the atmosphere is likely. The only degradation process is diffusion to the stratosphere, where photolytic destruction of Freons results in depletion of stratospheric ozone, thereby increasing the amount of ultraviolet-B (UV-B) radiation reaching the earthÕs surface (Hazardtext, 2003B; HSDB, 2001A; HSDB, 2001B). Preliminary Remediation Goals for Ambient Air (U.S. EPA, 2002, Region IX):
Freon 11 - 0.73 mg/m 3

Freon 12 - 0.21 mg/m 3
Freon 113 - 31 mg/m 3
Ref: September 24, 2003 (Revised) - FREON [11, 12, 113]. Technical Support Document: Toxicology. Clandestine Drug Labs/ Methamphetamine. Volume 1, Number 11. California EPA, Office of Environmental Health Hazard Assessment (OEHHA), Department of Toxic Substances Control.

Trichlorotrifluoromethane (CFC 113) - **Solvent, US EPA List 2 Inert** - CAS No. 76-13-1

US EPA: Class 1 Ozone Depleting Substance. Lifetime of Global Warming Potential: 85 years
Ref: http://www.epa.gov/ozone/ods.html

The stratospheric lifetime of this compound ranges between 63 and 122 years(5). As a result of this persistence in the atmosphere(5), this vapor-phase compound can be transported long distances and therefore, its concn should be fairly uniform throughout the globe away from known sources(SRC). [(1) Bidleman TF; Environ Sci Technol 22: 361-367 (1988) (2) Boublik T et al; The Vapour Pressures of Pure Substances. 2nd Rev Ed, Amsterdam: Elsevier p. 74 (1984) (3) Horvath AL et al; J Phys Chem Ref Data 28: 395-507 (1999) (4) Dilling WL; pp. 154-97 in Environmental Risk Analysis for Chemicals. Conway RA, ed. NY, NY: Van Nostrand Reinhold Co (1982) (5) Chou CC et al; J Phys Chem 82: 1-7 (1978)]
Ref: Hazardous Substances Data Bank for 1,1,2-TRICHLORO-1,2,2-TRIFLUOROETHANE CASRN: 76-13-1.
http://www.fluorideaction.org/pesticides/trichlorotrifluorome.toxnet.htm

Environmental Contamination Concerns
A. Surface Water. Bioconcentration in organisms is low to moderate; BCF (Bioconcentration factor: the ratio of the chemical concentration in the organism to that in surrounding water) is from 11-86.
B. Groundwater. In general, Freons that are spilled onto soil have the potential to leach into groundwater, because they do not bind well to soil (Hazardtext, 2003B; HSDB, 2001A; HSDB, 2001B). Fully halogenated hydrocarbons such as Freons 11, 12, and 113 are very resistant to chemical and biological degradation and are likely to be persistent contaminants if they reach groundwater.
D. Soil. If Freon is spilled onto soil, a portion may evaporate from the surface and the remainder will leach downward into the soil. Mobility through the soil is expected to be moderate based on estimated Koc values. Freon does not bind well to soil, and leaching to groundwater is possible (Hazardtext, 2003B; HSDB, 2001B).
E. Air. Once released to air, Freon exists solely as a gas. In the atmosphere, fully halogenated Freons diffuse to the troposphere, where they are very stable and can be transported great distances. Wet deposition may result in some loss, but re-volatilization into the atmosphere is likely. The only degradation process is

diffusion to the stratosphere, where photolytic destruction of Freons results in depletion of stratospheric ozone, thereby increasing the amount of ultraviolet-B (UV-B) radiation reaching the earthÕs surface (Hazardtext, 2003B; HSDB, 2001A; HSDB, 2001B).
Ref: September 24, 2003 (Revised). Released November 7, 2003) - FREON [11, 12, 113]. Technical Support Document: Toxicology. Clandestine Drug Labs/ Methamphetamine. Volume 1, Number 11. California EPA, Office of Environmental Health Hazard Assessment (OEHHA), Department of Toxic Substances Control.

Trifloxystrobin - **Fungicide** - CAS No. 141517-21-7

Freshwater Fish and Invertebrate Acute Toxicity
-- Rainbow trout 0.014 ppm (LC50) very highly toxic;
-- Bluegill sunfish 0.054 ppm (LC50) very highly toxic;
-- Water flea 0.025 ppm (LC50) very highly toxic;
Estuarine/Marine Fish and Invertebrate Acute Toxicity Under Flow-through Condition LC50 or EC50 (ppb):
---- Sheepshead minnow 78 (ppb) very highly toxic; ---- Mysid shrimp 8.62 (ppb) very highly toxic;
---- Eastern oyster (shell deposition) 29.3 (ppb) very highly toxic
Ref: US EPA Pesticide Fact Sheet. Trifloxystrobin. Reason for Issuance: New Chemical Registration. Date Issued: September 20, 1999.
http://www.epa.gov/opprd001/factsheets/trifloxystrobin.pdf

... Trifloxystrobin's major isomer, CGA-321113, forms at the average rate of 80% of the applied parent, is persistent, (half life is about 301 days), and soluble, 30.9 ppm and is also mobile. The major degradate minimum Koc is 49, the median Koc is 127 and is also stable to hydrolysis. The major degradate, CGA-321113 is persistent and mobile and has a potential to leach into groundwater. CGA-321113 has been found in the soil profile at the 36 inch depth.
Ref: Federal Register: May 22, 2002 (Volume 67, Number 99)] [Rules and Regulations] [Page 35915-35924]. Trifloxystrobin; Pesticide Tolerance. Final Rule.
http://www.fluorideaction.org/pesticides/trifloxystrobin.fr.may22.02.htm

-- Accumulation in water and/or sediment: Trifloxystrobin will not accumulate. CGA 321113 may accumulate in sediment (see DT50 above).
-- Remarks: Residue relevant for environmental monitoring in water: Surface water Ð trifloxystrobin Groundwater Ð trifloxystrobin. Member states may wish to monitor for NOA 413161 in vulnerable groundwater situations as it could approach the 10□g/l drinking water limit for chlorinated aliphatic compounds compounds even though it is considered not relevant.
Ref: Review report for the active substance trifloxystrobin. Trifloxystrobin. SANCO/4339/2000-Final. 7 April 2003. Finalised in the [European Commission] Standing Committee on the Food Chain and Animal Health at its meeting on 15

April 2003 in view of the inclusion of trifloxystrobin in Annex I of Directive 91/414/EEC.
http://www.fluorideaction.org/pesticides/trifloxystrobin.eu.april.03.pdf

Triflumizole - Fungicide - CAS No. 68694-11-1

Abstract: Laboratory bioassays were conducted to determine the contact **honey bee** toxicity of commercial and candidate neonicotinoid insecticides. The nitro-substituted compounds were the most toxic to the honey bee in our laboratory studies with LD50 values of 18 ng/bee for imidacloprid, 22 ng for clothianidin, 30 ng for thiamethoxam, 75 ng for dinotefuran and 138 ng for nitenpyram. The cyano-substituted neonicotinoids exhibited a much lower toxicity with LD50 values for acetamiprid and thiacloprid of 7.1 and 14.6 g/bee, respectively. Piperonyl butoxide, **triflumizole** and propiconazole increased honey bee toxicity of acetamiprid 6.0-, **244**- and 105-fold and thiacloprid 154-, **1,141**- and 559-fold, respectively, but had a minimal effect on imidacloprid (1.70, 1.85 and 1.52-fold, respectively). The acetamiprid metabolites, N-demethyl acetamiprid, 6-chloro-3-pyridylmethanol and 6-chloro-nicotinic acid when applied topically, produced no mortality at 50 g/bee. These results suggest that P450s are an important mechanism for acetamiprid and thiacloprid detoxification and their low toxicity to honey bees. When honey bees were placed in cages in forced contact with alfalfa treated with acetamiprid and the synergist, triflumizole, in combination at their maximum recommended application rates, no mortality was detected above that of the control.
Ref: Mechanism for the differential toxicity of neonicotinoid insecticides in the honey bee, Apis mellifera; by Takao Iwasa, Naoki Motoyama, John T. Ambrose and R.M.R. Michael Roe. Crop Protection; Volume 23, Issue 5 , May 2004, Pages 371-378.

Ref: PAN Pesticides Database - Chemical Toxicity Studies on Aquatic Organisms.
Acute Aquatic Ecotoxicity Summaries for Triflumizole on All Taxa Groups.
http://www.pesticideinfo.org/List_AquireAcuteSum.jsp?Rec_Id=PC34923

Common Name	Scientific Name	Avg Species LC_{50} (ug/L)	Avg Species Rating
Fish			
Bleak	Alburnus alburnus	400.0	Highly Toxic
Bluegill	Lepomis macrochirus	36.0	Very Highly Toxic
Rainbow trout,donaldson trout	Oncorhynchus mykiss	445.0	Highly Toxic
Molluscs			
Snail	Biomphalaria glabrata	10.0	Very Highly Toxic
Zooplankton			
Harpacticoid copepod	Nitocra spinipes	8.00	Very Highly Toxic

Triflumuron - Insecticide - CAS No. 64628-44-0

Triflumuron is very toxic to aquatic organisms. Triflumuron is toxic to bees.
Triflumuron: Fish toxicity:
LC50 (96 hr) bluegill sunfish (Lepomis macrochirus) > 20.8 µg/L
LC50 (96 h) rainbow trout (onchorhynchus mykiss) > 24.2 µg/L
Triflumuron: Daphnia toxicity:
EC50 (48 h) water flea (Daphnia magna) 1.6 µg/L
Triflumuron: Algal toxicity:
Growth rate: IC50 (72 h) green algae (Desmodesmus subspicatus) > 0.025 mg/L
Ref: August 9, 2004. Material Safety Data Sheet for Alsystin 250 Larvicide. Bayer CropScience.

-- Triflumuron is a toxic hazard to juvenile aquatic and terrestrial arthropods
-- Fish toxicity:
LC50: > 10 mg/L (96 h) rainbow trout
LC50: >100 mg/L (96 hours) golden orfe
Daphnia toxicity:
EC50: 0.23 mg/L (48 hours) daphnia magna.
-- Environmental fate, persistence and degradation:
Degradation of active constituent Ð half life:
t l/2: 960 days at pH4 (22 0 C)
t l/2: 580 days at pH7 (22 0 C)
tl/2: 11 days at pH9 (22 0 C)
Ref: Material Safety Data Sheet for Intrigue Termite Dust. Supplier: Bayer Environmental Science Ð A Business Group of Bayer CropScience Pty Ltd ABN 87 000 226 022 Address: 391 - 393 Tooronga Road, East Hawthorn Victoria 3123, Australia
http://www.fluorideaction.org/pesticides/triflumuron.msds.bayer.02.pdf

Acute Aquatic Ecotoxicity Summaries for Triflumuron on Fish.
Ref: PAN Pesticides Database - Chemical Toxicity Studies on Aquatic Organisms.
http://www.pesticideinfo.org/List_AquireAcuteSum.jsp?Rec_Id=PC34658&Taxa_Group=Fish

Common Name	Scientific Name	Avg Species LC50 (ug/L)	LC50 Std Dev	Number of Studies	Avg Species Rating
Fish					
Western mosquitofish	Gambusia affinis	10.0	-	1	Very Highly Toxic

Trifluralin - **Herbicide** - CAS No. 1582-09-8

• trifluralin has been added to the OSPAR (Convention for the Protection of the Marine Environment of the North-East Atlantic) List of Chemicals for Priority action in 2002 because it is considered to be a PBT substance fulfilling the criteria for Persistence, Bioaccumulation and Toxicity (page 3).
• 5.2 RISK TO AQUATIC ORGANISMS. *Selenastrum capricornutum* is the most sensitive aquatic organism on an acute time-scale and fathead minnow is the most sensitive species on a chronic time-scale when tested with trifluralin and the lead formulation. Due to the difference in Annex VI trigger value, the risk assessment is driven by the endpoints for fish both on an acute as long term time-scale. The resulting acute TER-value at 1 m from a field (7.9) is below and hence breaches the Annex VI trigger value of 100 so the risk should be considered as high. The rapporteur Member State calculated the risk taking into account buffer zones. This resulted in a TER-value of 110 indicating a low acute risk to fish if a bufferzone of 15 meters is taken into account. The choice of a relevant endpoint for the long-term risk to fish was extensively discussed during the EPCO expert meeting (section ecotoxicology, June 2004). Trifluralin induces vertebral lesions in several fish species, and in some instances this effects is induced after short term exposure (24 hours for brown trout). The meeting agreed that the risk assessment should be based in initial PEC and on the NOEC of 0.3 ?g/L (based on the observed vertebral lesions in the study with fathead minnow) together with an uncertainty factor of 10 to

conduct the risk assessment. This would lead to a TER value of 0.38 when a buffer zone of 15 m is taken into account (without detailed calculations, a bufferzone of 50 m should lead to a TER-value of approximately 1). Consequently the risk for aquatic organisms should be regarded as high. Therefore the risk should be further refined either by higher tier studies or by a refinement of the exposure assessment. Therefore, the expert meeting set the following data requirement: notifier to submit exposure studies with different exposure times using the fathead minnow as the most sensitive fish species. As an alternative microcosm tests with a more realistic exposure regime may be run (page 21-22).

Trifluralin and the metabolites TR-4, TR-7 and TR-14 can be found in concentrations above 10% of the AR in the sediment. Therefore the risk to sediment dwelling organisms needs to be addressed. This risk assessment is available in the addendum 3 of June 2004... (page 22).

Studies on bio-accumulation in fish are available as the logPow exceeds 3 and the DT50 in water exceeds 10. The steady state bioconcentration factor is found to be 5674 which exceeds the Annex VI trigger value of 100 for not readily biodegradable product ... This BCF-value and the fact that the depuration is less than 95% after 14 days triggers a fish full life cycle study which is available with the sheephead minnow. The resulting NOEC from this study is 1.3 µg/L (based on fecundity, no vertebral lesions observed) which is higher than the NOEC which is chosen for the long term risk assessment. As mentioned above a high long term risk to aquatic organisms was identified for which a data requirement is still open. Therefore, EFSA proposes that Member States may reconsider the risk for bioaccumulation when this long term assessment is revised, on receipt of the above mentioned data requirement. Residues in fish were found during the available field monitoring study (page 22-23).

• High risks were identified for aquatic organisms, in particular the chronic risk to fish, which require consideration of appropriate risk mitigation measures. Using the initial predicted environmental concentrations (PEC's) together with the no observed effect level (NOEC) of 0.3 ?g/L leads to a toxicity exposure ratio (TER)-value of 0.38 when a bufferzone of 15 metres is taken into account which is below the Annex VI trigger value of 10 (without detailed calculations, a bufferzone of 50 m should lead to a TER-value of approximately 1). Further data to address this risk is needed and the risk assessment can only be concluded when the outstanding data is evaluated (page 3)

Ref: March 14, 2005. European Food Safety Authority: Conclusion regarding the peer review of the pesticide risk assessment of the active substance trifluralin. EFSA Scientific Report (2005) 28, 1-77.
http://www.fluoridealert.org/pesticides/trifluralin.eu.long.2005.pdf

Note: Still in use in the EU as of October 2003; however the EU is currently reconsidering its use.

"Trifluralin 1582-09-8 Banned. Low degradability, bioaccumulative and toxic to water-living organisms. 1990."

Definition: "Banned. A substance which for health or environmental reasons by an authority decision is either no longer approved for any area of application, or for which an approval or registration has been denied from the first instance."

Ref: Euopean Commission. Appendix 5. Substances which may not be included as active ingredients in approved pesticide products, Chapter 15, Section 2, subsection one.
http://www.kemi.se/lagar_eng/pdf/app5_8.pdf

Ref: PAN Pesticides Database - Chemical Toxicity Studies on Aquatic Organisms. Acute Aquatic Ecotoxicity Summaries for Trifluralin.
http://www.pesticideinfo.org/List_AquireAcuteSum.jsp?Rec_Id=PC35146

Common Name	Scientific Name	Avg Species LC50 (ug/L)	LC50 Std	Number of Studies	Studies Avg Species Rating

			Dev		
Amphibians					
Toad	Bufo bufo japonicus	14,000	-	1	Slightly Toxic
Fowler's toad	Bufo woodhousei fowleri	150.7	37.8	7	Highly Toxic
Fish					
Porgy	Acanthopagrus schlegeli	56.0	-	1	Very Highly Toxic
Bunni fish	Barbus sharpeyi	250.0	-	1	Highly Toxic
Goldfish	Carassius auratus	397.0	312.3	5	Highly Toxic
Agohaze, goby	Chasmichthys dolichognathus	120.0	-	1	Highly Toxic
Pacific herring	Clupea pallasi	5.00	-	1	Very Highly Toxic
Sheepshead minnow	Cyprinodon variegatus	190.0	-	1	Highly Toxic
Common, mirror, colored, carp	Cyprinus carpio	391.2	377.1	5	Highly Toxic
Western mosquitofish	Gambusia affinis	9,630	9,893	5	Moderately Toxic
Green fish	Girella punctata	110.0	-	1	Highly Toxic
Channel catfish	Ictalurus punctatus	800.7	711.2	10	Highly Toxic
Bluegill	Lepomis macrochirus	197.4	233.1	40	Highly Toxic
Largemouth bass	Micropterus salmoides	86.2	19.5	4	Very Highly Toxic
Oriental weatherfish	Misgurnus anguillicaudatus	350.0	-	1	Highly Toxic
Striped mullet	Mugil cephalus	32.0	-	1	Very Highly Toxic
Rainbow trout,donaldson trout	Oncorhynchus mykiss	188.1	326.7	58	Highly Toxic
Medaka, high-eyes	Oryzias latipes	430.0	-	1	Highly Toxic
Red Sea Bream	Pagrus major	23.0	2.16	3	Very Highly Toxic
Hirame, flounder	Paralichthys olivaceus	56.0	-	1	Very Highly Toxic
Grunt	Parapristipoma	33.0		1	Very Highly

	trilineatum				Toxic
Fathead minnow	Pimephales promelas	133.4	33.3	8	Highly Toxic
Harlequinfish, red rasbora	Rasbora heteromorpha	733.3	188.6	3	Highly Toxic
Jacopever	Sebastes schlegeli	74.0	-	1	Very Highly Toxic
Yellowtail	Seriola quinqueradiata	5.00	-	1	Very Highly Toxic
Walleye	Stizostedion vitreum vitreum	180.0	-	1	Highly Toxic

Triflusulfuron-methyl - Herbicide - CAS No. 126535-15-7

Potential Ground Water Contaminant.
Ref: PAN Pesticides Database - Chemicals for Triflusulfuron-methyl.
http://www.pesticideinfo.org/Detail_Chemical.jsp?Rec_Id=PC34661

Triphenyltin fluoride- Antifoulant, Algaecide, Herbicide - CAS No. 379-52-2

Severe marine pollutant.
Ref: Material Safety Data Sheet ACC# 99193. Triphenyltin fluoride.
https://fscimage.fishersci.com/msds/99193.htm

Ref: PAN Pesticides Database - Chemical Toxicity Studies on Aquatic Organisms. Acute Aquatic Ecotoxicity Summaries for Triphenyltin fluoride on All Taxa Groups.
http://www.pesticideinfo.org/List_AquireAcuteSum.jsp?Rec_Id=PC34678

Common Name	Scientific Name	Avg Species LC_{50} (ug/L)	Avg Species Rating
Fish			
Bleak	Alburnus alburnus	400.0	Highly Toxic
Bluegill	Lepomis macrochirus	36.0	Very Highly Toxic
Rainbow trout,donaldson trout	Oncorhynchus mykiss	445.0	Highly Toxic
Molluscs			
Snail	Biomphalaria glabrata	10.0	Very Highly Toxic
Zooplankton			
Harpacticoid copepod	Nitocra spinipes	8.00	Very Highly Toxic

Committee Social Issues - Trial finds that Prozac (fluoxetine) may stunt children's growth

From: "Ailsa Boyden" <boydens@mrbean.net.au>
Date: 31/05/2005 12:14 AM
Subject: Trial finds that Prozac (fluoxetine) may stunt children's growth

PROZAC in news again. Link to enzyme decrease crucial to bone development..
FDA says Lilly had declined 1-yr study.
Sent: Tuesday, November 23, 2004 2:23 PM
Subject: Fluoro-drug PROZAC in news again. Link to enzyme decrease crucial to bone development.. FDA says Lilly had declined 1-yr study.

Prozac (aka FLUOxetine)
Sunday Herald
21 November 2004
Trial finds that Prozac may stunt children's growth
25,000 young Britons prescribed drug
By Liam McDougall, Health Correspondent

A CLINICAL trial by the makers of Prozac has revealed evidence the antidepressant could harm the growth and development of children.

The Sunday Herald has uncovered details of a 19-week trial carried out by Eli Lilly where children aged between eight and 17 years old who were given the drug gained an average of 1cm less in height and 1.1kg less in weight than others treated with a placebo.

The results revealed that treatment with fluoxetine, the generic name for Prozac, was also linked to a decrease in levels of alkaline phosphatase, an enzyme crucial to bone development.

Despite concerns from America's Food and Drug Administration (FDA) in 2001, which requested that Lilly carry out a further one-year trial to ensure the drug was safe to be used by children, no such test has ever been done.

According to FDA documents seen by the Sunday Herald, Lilly refused to carry out a more thorough trial, but was granted a licence to prescribe Prozac to children a year later. In the FDA paper Andrew Mosholder, medical officer for the FDA, writes: "Nineteen weeks of fluoxetine treatment was associated with reduced growth velocity relative to placebo.

"On balance, I believe that this trial provides evidence of reduced
growth velocity with fluoxetine treatment, and I believe labelling should reflect the finding. In my opinion, the sponsor has not provided an adequate rationale for declining to do a one-year study as we requested."

British experts on psychiatric drugs last night expressed astonishment about the trial, the results of which have never been highlighted to doctors or printed by Eli Lilly on UK Prozac patient information leaflets.

News about the possible harm to children comes at a time when the Medicines and Healthcare Products Regulatory Authority (MHRA), the UK drug licensing agency, is considering an application by Lilly to have Prozac licensed for use on under-18s.
In the last year, thousands of British children have moved on to Prozac after the MHRA ruled that all antidepressants in the SSRI class, with the exception of Prozac, should be banned from use on

children. The change followed a major inquiry by the authority that found the drugs could cause suicide.

Although Prozac does not have a licence for use on children, the decision to ban all other SSRIs has led to a massive rise in the number of doctors and psychiatrists prescribing the drug to children on an "off-licence" basis. Before the MHRA ban, experts say 25,000 children under the age of 18 in the UK were being prescribed antidepressants. Between 2000 and 2002 the number of antidepressant prescriptions for children soared by 68%.

Dr David Healy, director of the north Wales department of psychological medicine, whose warnings that antidepressants could cause suicide led to the MHRA inquiry, expressed concern at the Prozac trial results. He said: "Very few people will have heard of this. Doctors who are giving Prozac to children and who read the published scientific articles won't see anywhere that clinical trials have indicated that children on Prozac don't grow as well.
"This was flagged up by the FDA two years ago and the FDA asked the company to do further work. It should give people who may be thinking about using the drug, some cause for concern."

He said that in the past, trials on antidepressants had been carried out and been reported by pharmaceutical firms "in very misleading terms".

"In the Prozac children's trials there is evidence that the children didn't grow and put on weight in the same way," he said. "That's the kind of thing you just don't get to hear about. For those who are in the process of development this is potentially a very important issue.

"You'd have thought that in the first instance the company or the MHRA, who are responsible for making sure these drugs are on the market, would ensure that an appropriate label was placed on them so that we would know what the risks are and know what to look out for.

"But when you look at the label for Prozac there is no mention of concerns about the development of children. All it says is that this product is not recommended for children. But the number of children taking Prozac in the UK has increased hugely in the last two years."
Dr Andrew Herxheimer, a clinical pharmacologist and founding editor of the Drug And Therapeutics Bulletin, said: "I think the news about the possible effect on children's growth is serious. The effects of antidepressants in children is a hugely under-researched area.
"When there are doubts the benefit of the doubt has to be given to the patients and not to the drug. But both the MHRA and Eli Lilly are not in the business of admitting doubt."

Dr Iain McClure a child and adolescent psychiatrist and spokesman for the Royal College of Psychiatrists in Scotland, said that despite prescribing Prozac to children for the last five years he had not encountered problems with side-effects. He added: "Over the last few years I've been using fluoxetine with young people and I have not experienced any difficulties with side-effects and I've seen genuine therapeutic results.
"All I can do is speak to my own clinical experience of using fluoxetine with young people. I haven't had such evidence brought to my attention."

Dr Harvey Marcovitch, a consultant paediatrician and associate editor of the BMJ, said: "There is lots of information that is not generally available and pharmaceutical companies have been accused of publishing good news and burying bad news for years. As a journal editor, I believe that every trial that's ever conducted ought to be published somewhere regardless of whether the results are embarrassing to somebody or not."

Andrew Day, a spokesman for Eli Lilly, said the company was designing a "long-term" study into the effects of Prozac on children's development. He added: "We have a clear and transparent policy. Any and all clinical data that we have is shared with all regulatory authorities."

A spokeswoman for the MHRA said: "Eli Lilly was encouraged to put in a licence application for Prozac and that is being considered at the moment."

http://www.sundayherald.com/46216
> >
> > ******
> > Avoid fluoridecontaining drugs : If you are taking the following,
contact
> > your doctor
> > for a fluoride-free alternative:
> >
> > Prozac
> > (fluoxetine), Rohypnol (flunitrazepam), Diflucan
> > (fluconazole, Flixonase or Flixotide
> > (fluticasone), Stelazine (trifluoperazine, Fluanxol or
> > Depixol (flupenthixol) or Floxapen
> > (flucloxacillin) and asthma drugs that use propellants
> > containg fluoride: Ventolin and Becotide
> >
> > ALSO
> > CIPRO: another nasty fluoroquinolone
> >
> > Two antimalarial drugs quinacrine and chloroquine

From: "Ailsa Boyden" <boydens@mrbean.net.au>
Date: Fri, Apr 8, 2005 10:54 am
Subject: afin_poisoning usa kennerly _ http://pmeiers.bei.t-online.de/kennerly.htm

William Kennerly (3) died after "routine" topical fluoride treatment

From the "New York Times", Saturday, Jan. 20, 1979
$750,000 Given In Child's Death In Fluoride Case.
Boy, 3, Was in City Clinic for Routine Cleaning
By Robert D. McFadden
A State Supreme Court jury awarded $750,000 yesterday to the parents of a 3-year-old Brooklyn boy who, on his first trip to the dentist in 1974, was given a lethal dose of fluoride at a city dental clinic and then ignored for nearly five hours in the waiting rooms of a pediatric clinic and Brookdale Hospital while his mother pleaded for help, and he lapsed into a coma and died.
The award - $600,000 for the wrongful death of the boy, William Kennerly, and $150,000 for the pain and suffering he endured in the hours before his death - was by far the largest ever made in New York State for the death of a 3-year-old, according to the lawyers for the parents, Clay Kennerly, 48, an $8,000-a-year city exterminator, and his wife, Inez, 42, of 300 Dumont Avenue in the Brownsville section.
The child, according to testimony at a four-day trial in State Supreme Court in Brooklyn, suffered spasms of vomiting and nausea, headaches and dizziness, and had to be revived from a coma by an injection of adrenaline into his heart several hours after his ordeal began.
The boy was then made to wait - in shock, another coma and finally in a state of cardiac arrest - for more than an hour before getting further treatment, witnesses said.
Stomach-Pumping Suggested
Other testimony indicated that the boy might have been saved at almost any time during the four hours and 40 minutes before he died by having had his stomach pumped or by having him drink some milk or lime water, which would have changed the fatal fluoride compound he had been given into a nontoxic calcium fluoride.
The defendants in the case were New York City, its Health and Hospitals Corporation and one of its clinics, the Brownsville Dental Health Center; Brookdale Hospital and its Brookdale Ambulatory Pediatric Care Unit; Dr. Bradford George, a dentist; Roslyn Cohen, a dental technician, and Dr. Pretti Bathia, a Brookdale pediatric clinic physician.
After more than a day of testimony by Mrs. Kennerly and medical and toxicological experts, and what were described as thorough investigations by the city and Brookdale Hospital, lawyers for the defendants on Thursday conceded liability, and the jury of five women and one man was instructed by Justice John A. Monteleone to return a verdict for the plaintiffs and to decide the damages to be assessed.
After the jury's verdict at 2:30 P.M., the defendants' lawyers, George W. Weiler for the city and James Hayes for Brookdale Hospital, moved to set the award aside as excessive. Justice Monteleone denied the motion, but said he would consider written motions for a reduction.
Some Reduction Proposed
Morton L. Panken, the lawyer for the Kennerly's, who have eight other children, said the family would not object to a reduction of the $600,000 award for wrongful death to $500,000 because that was the amount originally asked in the suit. But he said the family had sought $500,000 more for the child's suffering and would resist efforts to cut the $150,000 awarded for that
The story of the boy's death was related by his mother during the trial. She recalled that she took William, born on Feb. 7, 1971, for his first dental checkup on May 24, 1974, to the Brownsville Dental Health Center, a city clinic at 259 Bristol St.

There, he was examined by Dr. George, who found no dental caries and turned the boy over to Miss Cohen, a dental hygienist, for a routine teeth cleaning procedure. After cleaning, witnesses explained, Miss Cohen, using a swab, spread a stannous fluoride solution in the form of a jell over the boy's teeth as a decay preventive measure.

Fluoride in small amounts is mixed into various brands of toothpaste and the drinking water of some communities to prevent tooth decay. When used by a dentist or dental hygienist after a teeth cleaning, the fluoride jell is in a relatively strong solution, and a patient is told not to swallow it. Instead, after the solution is allowed to remain on the teeth briefly, the patient is given water and told to wash his mouth out and expectorate.

Fatal Solution Swallowed

According to Mrs. Kennerly, Miss Cohen was engrossed in conversation with a co-worker while working on William and, after handing him a cup of water, failed to instruct him to wash his mouth out with it and spit out the solution. Mrs. Kennerly said that Miss Cohen was not paying attention when William drank the water about 9:30 A.M.

In drinking the water, according to a Nassau County toxicologist, Dr. Jesse Bidanset, William ingested 45 cubic centimeters of 2 percent stannous fluoride solution, triple an amount sufficient to have been fatal.

Upon leaving the dentists chair, William began vomiting, sweating and complaining of headache and dizziness. His mother, appealing to the dentist, was told the child had been given only routine treatment. But she was not satisfied and was sent to the Brookdale Ambulatory Pediatric care unit in the same building.

Click here to read about another "routine" treatment settled out of court ...

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