# Slow elimination of PCDF/PCDDs in adult fish and crabs transferred from a contaminated fjord to a clean laboratory environment.

**Jon Knutzen**
Norwegian Institute for Water Research (NIVA),
P.O. Box 176 Kjelsås, 0411 Oslo, Norway.

**Martin Schlabach**
Norwegian Institute for Air Research (NILU),
P.O.Box 100, 2007 Kjeller, Norway.

## 1. Background

The Frierfjord in Norway has been recipient for large amounts polychlorinated dibenzo-furans/dibenzo-p-dioxins (PCDF/PCDDs) from a magnesium factory for nearly 30 years, resulting in high concentrations in fish and shellfish [1]). From a yearly amount of perhaps as much as 3 - 5 kg toxicity equivalents (TEQ [2])) before 1976, the load was reduced to 300 - 500 g per year in the period 1977 - 1989, and further to 7 g/year in 1992 and about 2 g/year 1992 - 1994.

After the more than 99% reduction in load from 1989 - 90 to 1991, TEQ in edible organisms have decreased about 60 - 95%. However, the TEQ concentrations still are considerably above levels which would warrant withdrawal of the prohibition on sales and advice against consumption of seafood [3]).

The question is: How many years will it take to reach acceptable levels? To throw more light upon this problem, cod (*Gadus morhua*), flounder (*Platichthys flesus*) and crabs (*Cancer pagurus*) were transferred from the Frierfjord to the NIVA Marine Research Station Solbergstrand for depuration studies in uncontaminated water.

## 2. Experimental

15/38 cod (1. and 2. experiment), 46 flounders and 77/17 (female/male) crabs were transferred from Frierfjorden to Solbergstrand. Difficulties with sampling of cod necessitated the use of variably sized specimen: Mostly 400 - 1000 g (min./max. 160 - 2100 g) at time of sampling for analysis. Flounders, at sampling from the experimental basins, were 150 - 870 g, mostly 300 - 600 g. The crabs had carapace width of 13 - 18 cm. Growth was not recorded.

The fish studies were conducted in a 1.5 x 1.5 x 0.65 m glass fibre basin (water volume about 900 l) with Oslofjord water from 40/60 m. Mean water residence time was about one hour, temperature 6 - 9° C and salinity 30 - 35 ‰ S. The fish were given commercial fother once a day. In the first experiment cod and flounder were kept together.

Crabs were kept separately in a 15 $m^3$ glass fibre basin of radius about 4 m, and with a simulated natural bottom (stones with algae, gravel). The crabs were fed once a week. Mean theoretical renewal time for the water was about 8 hours,

In the first 4 months study 5 of 15 specimen of cod died within one week, presumably from infected net wounds. The accompanying flounders were sampled over 7 months, in which 15 of 46 died, evenly distributed over the period. In the second experiment with only cod, the fish were treated with formalin at start, and only 5 of 38 died in the course of the 8 months study. 3 female crabs died in the course of 16 months, 1 male in 7 months.

Composite samples (liver and fillet of fish, hepatopancreas (digestive gland) of crabs, n = 5) were analyzed at start and at intervals appearing from the figures presenting the results. For the liver and hepatopancreas samples, whole organs were homogenized, for fillet samples 15 - 20 g dorsal muscle from each of 5 fish.

Analysis of PCDF/PCDDs were according to a method certified after intercalibration exercises conducted by WHO and Analytical Quality Assurance/University of Tübingen, Germany [4]).

### 3. Results and discussion

TEQ in cod liver decreased about 90% in the two experiments (figure 1). As liver fat increased considerably during the studies, reduction was even larger on lipid basis - more than 95%.

Analysis of composite cod fillet samples at start and end of the two experiments showed about the same reduction as in liver.

However, as growth was not measured, the relative contribution from this factor is unknown. For cod of the size employed, growth dilution may have caused about half of the total decrase in TEQ concentration, perhaps even more.

In the experimental animals 123478/123678-HxCDF, 2378-TCDD, and 23478-PeCDF represent about 90% of sum TEQ. The start levels of all these congeners were reduced about proportionally with sum TEQ.

Contrary to the cod results, no decrease in TEQ was observed in flounder fillet (figure 2). The lack of dilution from growth indicates that the experimental animals have been stressed. Relatively high lethality (15 of 46) supports this, likewise frequent occurrence of external and internal injures.

Also in crab hepatopancreas decrease was slow or questionable. Due to fluctuating fat content the results presented in figure 3 are given on lipid basis. It is seen that in female crabs TEQ contamination apparently was stabilized at less than 50% of the initial level. Individual variations, which for other persistent organochlorines is known to span at least one order for magnitude in both crabs and fish (unpubl.), probably is the main cause of the observed fluctuations. (With the slow depuration of PCDF/PCDD indicated, a mean of the values recorded from the first month may be more representative of the start concentrations than the single value from day 0. Monitoring data from 1991 and 1992 with TEQ-levels more than 50% above the presented start concentrations support this).

Studies of dioxin elimination from fish of comparable size are scarce, and similar observations in large crustaceans apparently lacking. Studies of small sized fish are of limited relevance, due to the difference

in surface: volume ratio, which make the diffusion part of the total elimination process more important in small fish.

For 2378-TCDD in 1.5 kg carp Kuehl et al. [5]) found a half life of about 300 days (possible growth dilution not considered). No reduction in 23478-PCDF levels was recorded in a 21 days depuration study with 75 - 175 g rainbow trout [6]). On the other hand, for OCDF/OCDD body burden half life of only 1 - about 2 weeks has been reported for 1 kg specimen of the same species [7]).

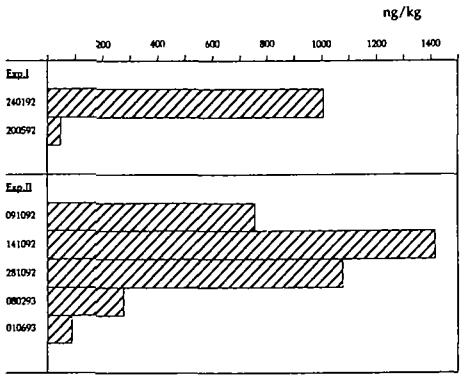


**Figure 1.** Reduction of $TEQ_{PCDF/D}$ in liver of cod (*Gadus morhua*) in the course of two experiments of four (I) and eight months duration, ng $TEQ^2$)/kg w.w.

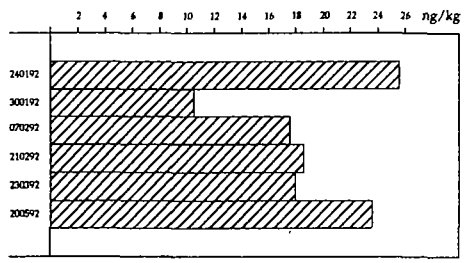


**Figure 2.** $TEQ_{PCDF/D}$ in fillet of contaminated flounder (*Platichthys flesus*) after transference to clean environment, ng/kg w.w.

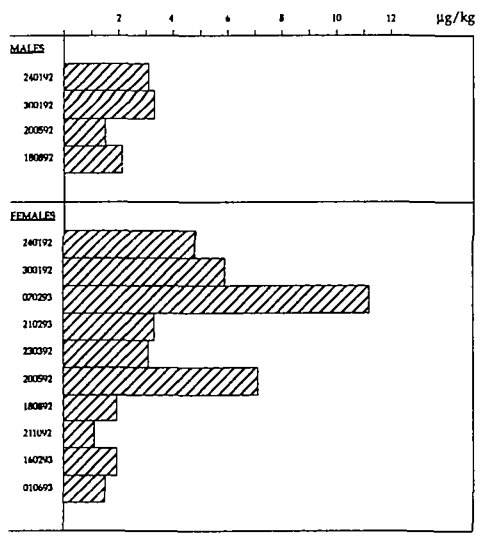


**Figure 3.** $TEQ_{PCDF/D}$ in hepatopancreas of contaminated edible crabs (*Cancer pagurus*) after transference to clean environment, µg/kg fat.

## 4. Conclusions

The problably slow reduction in body burden of PCDF/PCDDs in fish and crabs indicated from the experimental results is in accordance with monitoring data, particularly with regard to trends recorded in the more moderately exposed local populations of fish and crabs at some distance from the Frierfjord [3]. Similar development has been observed in other areas relieved of direct dioxin discharges [8].


[1]) Knutzen, J. and M. Oehme (1989). Polychlorinated dibenzofuran (PCDF) and dibenzo-p-dioxin (PCDD) levels in organisms and sediments from the Frierfjord, southern Norway. Chemosphere, 19, 1897-1909.

[2]) Ahlborg, U.G., A. Brouwer, M.A. Fingerhut, J.L. Jacobson, S.W. Jacobson, S.W. Kennedy, A.A.F. Kettrup, J.H. Koeman, H. Poiger, C. Rappe, S.H. Safe, R.F. Seegal, J.Tuomisto and M. van den Berg (1992). Impact of polychlorinated dibenzo-p-dioxins, dibenzofurans and biphenyls on human and environmental health, with special emphasis on application of the toxic equivalency factor concept. Europ. J. Pharmacol. 228, 179-199.

[3]) Knutzen, J., Aa. Biseth, E. Brevik, N. Green, M. Schlabach and J.U. Skaare (1995). Monitoring of micropollutants in fish and shellfish from the Grenland fjords 1994. Norwegian State Pollution Monitoring Program, report 630/95, NIVA-report 3363. (In Norwegian).

[4]) Oehme, M., M. Schlabach, K. Hummert, B. Luckas and E.S. Nordøy (1995). Determination of levels of polychlorinated dibenzo-p-dioxins, dibenzofurans, biphenyls and pesticides in harp seals from the Greenland Sea. Sci. Total Environ. 162, 75-91.

[5]) Kuehl, D.W., P.M. Cook, A.R. Batterman, D. Lothenbach and B.C. Butterworth (1987). Bioavailability of polychlorinated dibenzo-p-dioxins and dibenzofurans from contaminated Wisconsin river sediment to carp. Chemosphere 16, 667-679.

[6]) Sijm, D.T.H.M., A.L. Yarechewski, D.C.G. Muir, B. Webster, W. Seinen and A. Opperhuizen (1990). Biotransformation and tissue distribution of 1,2,3,7-tetrachlorodibenzo-p-dioxin, 1,2,3,4,7-pentachlorodibenzo-p-dioxin and 2,3,4,7,8-pentachlorodibenzofuran in rainbow trout. Chemosphere 21, 845-866.

[7]) Niimi, A.J. and B.G. Oliver (1986). Biological half-lives of chlorinated dibenzo-p-dioxins and dibenzofurans in rainbow trout (*Salmo gairdneri*). Environ. Toxicol. Chem. 5, 49-53.

[8]) Hagen, M.E., A.G. Colodey, W.D. Knapp and S.C. Davis (1995). Environmental response to decreased dioxin and furan loadings from British Columbia pulp mills. Pp. 251-256 in P. Adriaens et al. (eds): DIOXIN '95, 15th Int. symp. on chlorinated dioxins and related compounds, Edmonton, Canada. Short Papers Vol. 24.