DIOXIN LEVELS IN EGGS OF GREAT BLUE HERONS (ARDEA HERODIAS) DECLINE RAPIDLY IN RESPONSE TO PROCESS CHANGES IN A NEARBY KRAFT PULP MILL.

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ABSTRACT: Over the interval 1983-1987, elevated PCDD levels were found in the eggs of Great Blue Herons foraging in an area receiving kraft pulp mill effluent. In 1988 the nearby mill began introducing process changes to eliminate PCDDs and PCDFs in its effluent. By 1991 PCDD levels had fallen dramatically in the heron eggs.

INTRODUCTION: A Great Blue heron nesting colony is situated about 0.5 km from the outfall of a kraft pulp mill located at Crofton, British Columbia, on the east coast of Vancouver Island (fig. 1). The herons reside in the area throughout the year and forage for small fish in the intertidal zone adjacent to the mill's outfall. In 1983, and annually since 1986, PCDD and PCDF levels have been monitored in the eggs of the herons. In 1988, studies showed that the elevated levels of PCDD in the eggs were associated with affects on the developing embryo (Bellward et al., 1990[1]; Hart et al., 1991[2]). In early 1988 the mill began introducing process changes to eliminate PCDDs and PCDFs in its effluent. The first steps were to monitor wood chips used as feed stock for chlorophenol contamination, and increase the level of ClO_2 substitution in the bleaching process. In 1989 defoamers free of dioxin precursors were introduced, and additional changes were made to the bleaching process (fig. 2). By early March 1990, two years after it was started, the mill's program to reduce dioxin contamination in its effluent was in place.

METHODS: In 1983, and annually since 1986, one egg/nest was collected from about 5 - 10 nests in the heron colony. The individual eggs were analyzed for PCDD and PCDF residues at the Canadian Wildlife Service's laboratory at the National Wildlife Research Centre in Ottawa. In 1990 only, the eggs were pooled for analysis. The analyses were performed by GC/MS using internal standard quantization with $^{13}C_{12}$ PCDD surrogates (Norstrom et al., 1990[3]).

RESULTS AND DISCUSSION: From 1983 through to 1987 elevated levels of 2378-TCDD, 12378-PnCDD and 123678-HxCDD were monitored in Great Blue heron eggs collected near the kraft mill at Crofton (Elliott et al., 1989[4]). Lower levels of 123789-HxCDD, 2378-TCDF and 23478-PnCDF were also measured but will not be discussed here. In 1987/88, elevated levels of PCDDs and PCDFs were also reported in sediment, bivalves, crabs and

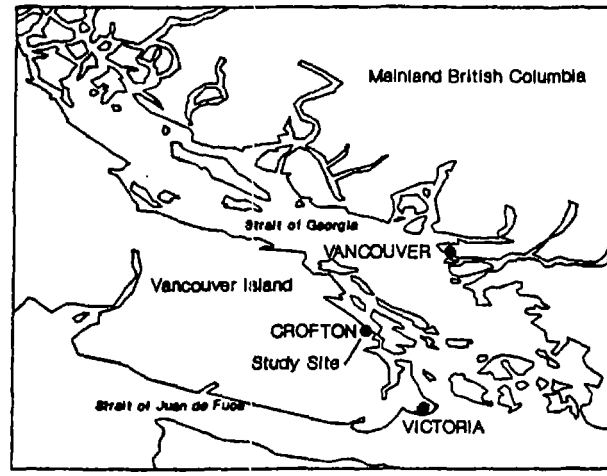

Figure 1

fish collected near the mill's outfall (Norstrom et al., 1988[5]). Around this time, the presence of trace amounts of dioxins in sludge, effluent and bleached pulp confirmed that kraft pulp mills are a source of these compounds (Amendola et al., 1987[6]). In 1990 a program to monitor PCDDs and PCDFs in sediment, oyster and crabs, near the Crofton mill was undertaken (Hatfield Consultants Ltd., 1990[7]).

Beginning in 1988, an intensive industry research program showed that the presence of PCDDs and PCDFs in mill effluent, particularly 2378-TCDD and 2378-TCDF, was a consequence of the high concentration of molecular chlorine used in the bleach plant, and in part to the amount of potential precursor dibenzodioxin (DBD) and dibenzofuran (DBF) in defoamer products, process water and wood furnish itself (Berry et al., 1989[8]). In addition, it was found that large amounts of hexachlorinated dioxin (HxCDD) are formed during digestion when polychlorophenol-treated wood chips are used as feed stock (Luthe et al., 1990[9]). Norstrom et al. (1988[5]) proposed that condensation of chlorophenoxyphenols to HxCDD and PnCDD was occurring.

Once the sources of formation of the PCDDs and PCDFs were identified the mill instituted several changes to its process to eliminate them from its effluent. In March 1988 the mill began testing chip supplies for polychlorophenol levels and phasing out contaminated chips as feed stock. In late 1988 and early 1989, to reduce the concentration of molecular chlorine below the threshold for enhanced production of TCDD and TCDF, both bleach plants at the mill were converted to 50% ClO_2 substitution. In October 1989, defoamers reformulated with purified oils free of DBD and DBF were introduced. Finally, in late 1989 and early 1990, a bleaching sequence using chlorine first was begun. Studies had shown that when chlorine is applied early in chlorination, the formulation of TCDD and TCDF is significantly reduced (Berry et al., 1989[8]).

Figure 2 shows the affect on 2378-TCDD, 12378-PnCDD and 123678-HxCDD levels in heron eggs as the process changes were put in place. Mean TCDD and HxCDD levels in the eggs of the herons declined throughout the period. Mean levels in 1991 were only 1/10 of concentrations in 1987 and were significantly ($p < .05$) lower for all three congeners in 1991.

Mean 123678-HxCDD levels fell each year from a high of 436 ppt in 1987 to 229 ppt in 1990, and then rapidly to 34 ppt in 1991. 12378-PnCDD levels did not change until 1991 then fell rapidly from 223 ppt to 32 ppt. 2378-TCDD fell from 209 ppt in 1987 to 78 ppt and 102 ppt in 1989 and 1990 respectively, then like the other congeners fell rapidly in 1991 to 16 ppt. The rapid decline in PCDDs in the heron eggs is clearly due to a decrease intake from

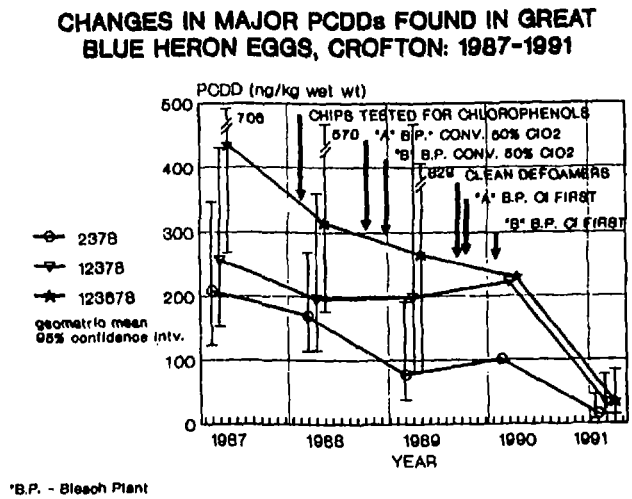

Figure 2

their diet. Therefore, there must have been a decline in PCDD concentrations in heron prey which is probably related to reduced emissions from the mill. The major fish prey of Great Blue herons in the Strait of Georgia are starry founder (Platichthys stellatus), Pacific staghorn sculpin (Leptocottus armatus), threespine stickleback (Gasterosteus aculeatus) and shiner perch (Cymatogaster aggregata) Butler, 1990[10]; Harfenist, in prep.[11]). Based on size preference, we believe that most of the fish eaten by herons are one or two years old. Because PCDD accumulation times are short in young fish, any change in PCDD levels in effluent is more likely to be reflected, assuming that bioavailable PCDDs are mainly coming from the effluent rather than in situ PCDDs in sediment. Because the mill introduced several process changes over a relatively short period of time it is virtually impossible to establish cause and effect relationships between specific process changes and changes in congener levels in heron eggs. The task is made even more difficult because there are two potential sources for HxCDD and possibly PnCDD (direct chlorophenol contamination and mill effluent) and the relative contribution of each source to the total is not known. The steady fall in 123678-HxCDD levels from 1987 through 1990 possibly reflects the declining use of chlorophenol-treated chips by the mill. The loss in 2378-TCDD is consistent with the introduction of ClO_2 substitution, changes in the bleaching process and use of DBD/DBF-free defoamers. It is more difficult to account for 12378-PnCDD levels which remained high through to 1990, apparently unaffected by any of the changes made to that point. Elliot et al. (1989[4]) noted a strong linear correlation between concentrations of 12378-PnCDD and 123678-HxCDD in heron eggs and suggested both were derived from a chlorophenol source.

That relationship is not evident after 1988, possibly due to a more rapid loss to sediments (lower bioavailability) of the HxCDD. The abrupt fall of all congeners in 1991 may be due to a combination of several factors: introduction of chlorine-first bleaching, DBD/DBF-free defoamers, better effluent treatment and foodchain related phenomena, such as changes in prey species composition.

ACKNOWLEDGEMENTS: The authors gratefully acknowledge the contributions of Mary Simon, Andre Breault, Ian Moul and John Morgan to this study.

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