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James M. Haynes
*The College at Brockport*, jhaynes@brockport.edu

Sara Tucker Wellman
*The College at Brockport*, wellmanst@aol.com

Kerrie J. Beckett

James J. Pagano

Scott D. Fitzgerald

***See next page for additional authors***



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# Histological Lesions in Mink Jaws are a Highly Sensitive Biomarker of Effect after Exposure to TCDD-like Chemicals: Field and Literature-based Confirmations

James M. Haynes[1,6], Sara T. Wellman[1], Kerrie J. Beckett[2], James J. Pagano[3], Scott D. Fitzgerald[4], and Steven J. Bursian[5]

[1]Department of Environmental Science and Biology
The College at Brockport
State University of New York
Brockport, NY 14420

[2]Stantec Consulting Services, Inc.
30 Park Drive
Topsham, ME 04086

[3]Environmental Research Center
The College at Oswego
State University of New York
Oswego, NY 13126

[4]Diagnostic Center for Population and Animal Health
Michigan State University
East Lansing, MI 48910

[5]Department of Animal Science
Michigan State University
East Lansing, MI 48824

[6]Copies: www.brockport.edu/envsci

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## Abstract

The mink (*Mustela vison*) is one of the most sensitive mammals to 2,3,7,8 tetrachlorodibenzo-*p*-dioxin (TCDD)-like chemicals. By literature review we established that a histological lesion of the jaw bone of mink, evidenced by squamous epithelial hyperplasia in the gingival tissue that forms nests or cords that infiltrate the periodontal ligament and alveolar bone causing osteolysis of the mandible and maxilla that could lead to squamous cell carcinoma, is the most sensitive known biomarker of effect following exposure of mink to TCDD-like chemicals. Lesions have been observed when total TCDD toxic equivalents (TEQ: dioxins, furans, co-planar polychlorinated biphenyls or PCBs) in liver exceed 40 ng/kg-ww or when total PCB exceeds 1,698 ng/g-ww. This is the second report of histological evidence of this lesion in wild-caught mink, and it is the first report of the lesion being grossly detectable in naturally exposed mink. Some mink living near the south shore of Lake Ontario (exposed to the lake's food web) but not inland mink (not exposed to the lake's food web) accumulate more than 40 ng total TEQ/kg or

1,698 ng total PCB/kg in liver. Because of its sensitivity, the jaw lesion biomarker is very useful for assessing the health of wildlife populations exposed to TCDD-like chemicals.

## Introduction

The Lake Ontario food web is contaminated with polychlorinated biphenyls (PCBs), dioxins and furans (O'Toole et al. 2006, Carlson and Schwackhamer 2006). Assessment of mink jaw tissue for mandibular and maxillary squamous epithelial proliferation offers one of the most sensitive methods to detect piscivorous mammalian exposure to these chemicals, including 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), polychlorinated dibenzofurans (PCDF), and co-planar TCDD-like PCBs (Beckett et al. 2005). Environmental exposure to these chemicals manifests as a histologically detectable lesion identified as mandibular and maxillary squamous epithelial proliferation or hyperplasia in the gingival tissue of the maxilla and mandible of mink (*Mustela vison*) (Beckett et al. 2005, Render et al. 2000a, b, 2001). The lesion is characterized by proliferation of squamous epithelial cells that form nests or cords that infiltrate the periodontal ligament and alveolar bone causing osteolysis of the mandible and maxilla (Beckett et al. 2005). The chemical, dose, and duration of exposure contribute to the level or stage of progression of the lesion (Beckett 2004). Squamous epithelial proliferation appears to progress to squamous cell carcinoma (SCC) in mink gingival tissue (Bursian et al. 2006a, b, Beckett et al. 2005). SCC is extremely invasive and destructive, meeting certain criteria of malignant carcinogenicity, and has been shown to be fatal to mink in laboratory studies (Beckett 2004).

This study originated from a project (Haynes et al. 2009) that identified "degradation of fish and wildlife populations" and "bird or animal deformities and reproductive problems" as potential problems in the Rochester Embayment of Lake Ontario Area of Concern (RAP 1993, 1997). Here we employ two approaches to determine if histological lesions in mink jaws are a highly sensitive biomarker of effect after exposure to TCDD-like chemicals. First, we reviewed the literature to correlate concentrations of TCDD-like chemicals in mink tissues with adverse effects. That review provided values to compare with tissue residues we found in mink (Haynes et al. 2009). Second, the prevalence of the oral lesion was assessed in wild, trapped mink that were naturally exposed to environmental contaminants in the Lake Ontario watershed.

## Methods

### *Literature Review*

Basu et al. (2007) reviewed the toxic effects of persistent organic chemicals on mink, covering field and lab studies and the use of mink in hazard assessments. Leonards et al. (1995) and Kannan et al. (2000) reviewed the toxicity of PCBs to mink. Rather than duplicate those efforts, we focused on studies that linked dietary concentrations of TCDD-like chemicals to tissue residues (Haynes et al. 2009), as well as to reproductive and other effects (Haynes et al. 2007, Appendix 4). We concentrated on reports of chronic exposures, as they best represent the exposures of mink to TCDD-like chemicals in the environment. We used Academic Search Premier, BioOne, BasicBIOSIS, InfoTrac, OneFile, JSTOR, and ScienceDirect as search engines.

*Assessing the Prevalence of Oral Lesions*

To assess adverse effects on mink, we examined three specimens captured in each of the four regions described in Haynes et al. 2009). These three mink were the individuals with the highest and lowest measured total PCB concentration in liver as well as the individual mink closest to the average total PCB concentration in liver among the 10 mink captured in each region. Previously frozen mink heads were preserved in 10% buffered formalin and examined clinically. Following the procedures of Beckett et al. (2005), mink heads were placed in Surgipath® Decalcifier II (hydrochloric acid) (Surgipath Medical Industries, Inc., Richmond, IL) and tissues were trimmed during the decalcifying process. The mandible and maxilla (i.e., lower and upper jaw, respectively) were sectioned to 5 µm, processed for paraffin embedding, mounted on microscopic slides, and stained with hematoxylin and eosin for subsequent histological assessment. Suspected lesions were verified by a board-certified veterinary pathologist (co-author S.D.F.).

## Results and Discussion

*Literature Review*

Dioxins and Furans. We identified studies linking dietary concentrations to tissue concentrations in mink for dioxins, furans and PCBs (Table 1). The toxicity of dioxins and furans to mink is well-established (Basu et al. 2007, Hochstein et al. 1998, 2001, Render et al. 2000a, 2001), and Lowest Observable Adverse Effect Levels (LOAELs) are usually reported as 2,3,7,8-dibenzo-*p*-dioxin (TCDD) toxic equivalents (TEQ) in ng/g (dietary) or pg/g (tissue residue) wet weight (ww). Many studies have evaluated toxic end points in terms of dietary concentrations but not as tissue concentrations in the mink. Render et al. (2000a) reported that a dietary concentration of 5 ng TCDD/g fed to adult females for six months caused proliferation of squamous epithelial cells in bone adjacent to teeth, and Render et al. (2001) found the same effect in 6- and 12-week-old kits fed 2.4 ng TCDD/g for as little as 14 days. Hochstein et al. (1998) reported that 1 ng TCDD/g caused 62.5% mortality in adult female mink fed for 125 days. Hochstein et al. (2001) found that when mink dams were fed 0.053 ng TCDD/g, kit survival was reduced to 47% vs. 83% in the control group (0.0006 ng TCDD/g in the diet). These studies did not report tissue concentrations.

Tissue as well as dietary concentrations were reported by several researchers who fed wild-caught fish to mink (Table 1). Since the fish used as the source of TCDD-like chemicals contained PCBs, as well as dioxins and furans, their total TEQ values also included contributions from the coplanar PCBs in the fish. Heaton et al. (1995) and Tillitt et al. (1996) reported reduced 3- and 6-week-old kit survival at a maternal dietary concentration of 22.4 pg TEQ/g (0.72 µg total PCB [TPCB]/g), which resulted in maternal liver concentrations of 208.3 pg TEQ/g (2.19 µg TPCB/g). Bursian et al. (2006b, c) reported increased mortality in 3- to 6-week-old mink kits whose dams had been fed 68.5 pg TEQ/g (3.7 µg TPCB/g), resulting in maternal liver residue concentrations of 218.4 pg TEQ/g (3.133 µg TPCB/g). Bursian et al. (2006a, b, c) also found jaw lesions in 27- and 31-week-old juveniles fed 47 pg TEQ/g (1.1 µg TPCB/g) and 9.2 pg TEQ/g (0.96 µg TPCB/g), respectively, with corresponding juvenile liver concentrations of 75 pg TEQ/g (16 µg TPCB/g) and 40.2 pg TEQ/g (1.7 µg TPCB/g).

Polychlorinated Biphenyls. Bursian et al. (2006c) found that the dietary $LC_{10}$ and $LC_{20}$ for total PCB were 0.231 and 0.984 μg TPCB/g, respectively, and estimated a threshold dietary concentration of 33.2 pg TPCB/g based on kit survival. Restum et al. (1998) reported reduced whelping in dams fed 0.25 μg TPCB/g, resulting in a liver concentration of 860 ng/g. Halbrook et al. (1999) found a trend (P = 0.069) for reduced litter size in dams fed 1.36 μg Aroclor 1260 equivalent (EQ) PCB/g in the diet, resulting in a maternal liver concentration of 7.25 μg Aroclor 1260 EQ/g and a maternal adipose concentration of 129 μg Aroclor 1260 EQ/g. However, this effect may have been confounded by the presence of mercury (0.22 μg/g) in the fish used in the diet, resulting in 3.67 μg/g in the maternal livers. Beckett et al. (2008) reported that dietary exposure to 3,3'4,4',5-pentachlorobiphenyl (PCB 126, the congener with the highest TEF, 0.1; USEPA 1989) resulted in impaired reproductive performance in dams (with confirmed matings) exposed to diets containing 2.4 (LOAEL) and 24 ng PCB 126/g feed. No significant differences were reported between the control group and the 0.24 (NOAEL) ng PCB 126/g-fed group for the measured reproductive parameters.

Co-planar PCBs were not analyzed in the original study by Haynes et al. (2009). However, there are distinct relationships between total TEQ, PCB TEQ, and dioxin-furan TEQ in mink (Tansy et al. 2003, Martin et al. 2006). Multiplying dioxin-furan TEQ by a factor of 1.25-2 gives a reasonable estimate of total TEQ in mink (Haynes et al. 2009), and this is what we did to bracket the likely range of total TEQ in our mink (Table 2).

*Assessing the Prevalence of Oral Lesions*

Twelve mink heads were processed and jaws were examined for gross and histological evidence of exposure to persistent organic chemicals (Table 3). Mink 17, captured near the shore of Lake Ontario within the Rochester Embayment AOC, exhibited histological and gross evidence of oral neoplasia and squamous cell carcinoma and, also, had the highest hepatic total PCB (5,871 ng/g-ww) and the third highest dioxin-furan TEQ (21.3 ng/kg-ww) concentrations reported by Haynes et al. (2009). The concentration of total PCB in mink 17 exceeded the LOAEL for the oral lesion (1,698 ng/g-ww) by a factor greater than three while the worst case scenario for total TEQ in mink 17 (dioxin-furan TEQ*2) was 42.6 ng/kg-ww, just above the 40.2 ng/kg-ww LOAEL (Tables 1 and 2).

Mink 17's lesion was grossly apparent during the processing stages, particularly in the left maxilla in which small proliferative areas containing nodular-like growths and swellings were recognized along the outside of the last premolar along the dental arcade. Mink 17 was diagnosed with severe lesions in the four jaw quadrants (right and left maxillae and mandible). Histopathologically, cellular cords and nests extended deep into the jaw bone in which the lesion was apparent. More than 50% of the jaw bone had been replaced with cords and/or nests of squamous epithelial cells. This is the first report of the lesion being grossly detectable in a naturally exposed mink and, therefore, suggests increased credibility for using the lesion as a biomarker for exposure to and effect of TCDD-like chemicals.

Three other mink (21, 63, 46) each had suspicious areas within the gingival tissue suggestive of the lesion but the degree of freeze-thaw artifact and tissue damage from tooth extraction for aging was too extensive to confirm the etiology of the tissue damage. Also, the concentrations of total PCB and total TEQ (the worst case, dioxin-furan TEQ*2, is shown in Table 3) in mink 21, 46 and 63 were far below concentrations observed to cause jaw lesions (Table 1). In addition, mink 49, with dioxin-furan TEQ of 47.6 ng/kg-ww that exceeded the 40

ng/kg-ww LOAEL shown in Table 1, exhibited no evidence of the lesion. Mink 49 also had low total PCB (755 ng/g-ww) compared to the 1,698 ng/g-ww LOAEL for jaw lesions.

In our literature review, jaw lesions had the lowest LOAELs for any biomarker of effect for TCDD-like chemicals. It appears that only mink with exceptionally high contaminant loads in the study area are at risk for jaw lesions. However, duration of exposure, in addition to concentration, may also play a role in the expression of the lesion (Beckett 2004). Possible reasons for high concentrations of total PCB and dioxin-furan TEQ in mink 17, which exhibited unmistakable evidence of the oral lesion, are presented in Haynes et al. (2009).

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Table 1. Selected endpoints and lowest observed adverse effects levels (LOAEL) reported for polychlorinated biphenyls (PCB), polychlorinated dibenzo-*p*-dioxins (PCDD), polychlorinated dibenzofurans (PCDF) and dioxin toxic equivalents (TEQ) in mink diets and liver tissue (units changed from original sources to ng/g-ww for consistency).

| | | | Concentration (ng/g) | | |
|---|---|---|---|---|---|
| Impairment | Endpoint | Toxin | Diet | Liver | Reference(s) |
| Reproduction | Kit survival 3 & 6 wks | PCB | 0.720 | 2.190 | Heaton et al. 1995 |
| | | PCDD | 0.060 | 2.626 | and Tillitt et al. 1996 |
| | | PCDF | 0.013 | 0.335 | |
| | | TEQ | 0.0224 | 0.208 | |
| Deformities | Jaw lesion in 31-wk kits | PCB | 960 | 1698 | Bursian et al. 2006b, c |
| | | TEQ | 0.0092 | 0.042 | |
| Deformities | Jaw lesion in 27-wk kits | PCB | 1100 | 16000 | Bursian et al. 2006a |
| | | TEQ | 0.047 | 0.075 | |
| Reproduction | Litter size | PCB | 1360 | 7250 | Halbrook et al. 1999 |
| Reproduction | P-1 whelping reduced | PCB | 250 | 860 | Restum et al. 1998 |
| | F-2 kit mortality | PCB | 500 | 464 | |

Table 2. High, low and average (excluding high and low) concentrations of dioxin-furan toxic equivalents (TEQs) in 1ivers from lakeshore (N=10) and inland (N=8) mink with detectable TEQ (ng/kg-wet weight). Dioxin-furan TEQ is multiplied by 1.25 and 2.0 to reflect the literature-based range of potential contributions from coplanar PCBs to total TEQ in mink.

| Location | Value | TEQ | TEQ*1.25 | TEQ*2 |
|---|---|---|---|---|
| Lakeshore | Low | 0.22 | 0.28 | 0.44 |
| | Average (8) | 7.75 | 9.69 | 15.50 |
| | High | 47.62 | 59.53 | 95.24 |
| Inland | Low | 0.01 | 0.01 | 0.02 |
| | Average (6) | 0.28 | 0.35 | 0.56 |
| | High | 4.16 | 5.20 | 8.32 |

Table 3. Tissue concentrations of selected TCDD-like chemicals in liver and adipose tissue of mink collected near Lake Ontario and inland, in and out of the Rochester Embayment Area of Concern (AOC), and corresponding presence and severity of jaw lesions. Dioxin toxic equivalent (TEQ) concentrations for liver are dioxin-furan TEQ*2 (see text and Table 2).

| Collection Location | Total PCB Liver (ng/g-ww) | Adipose TEQ (ng/kg-ww) | Liver TEQ (ng/kg-ww) | Jaw Lesion |
|---|---|---|---|---|
| AOC-Lake | | | | |
| Mink #17 | 5871 | 339 | 43 | Severe |
| Mink #21 | 682 | NA[a] | 7 | FTA[b] |
| Mink #56 | 15 | 8 | BDL[c] | NO[d] |
| AOC-Inland | | | | |
| Mink #63 | 554 | 9 | BDL | FTA |
| Mink #61 | 64 | 9 | BDL | NO |
| Mink #1 | 9 | 0 | 0 | NO |
| Out of AOC-Lake | | | | |
| Mink #49 | 755 | 38 | 77 | NO |
| Mink #46 | 230 | 20 | 4 | FTA |
| Mink #53 | 14 | 5 | 2 | NO |
| Out of AOC-Inland | | | | |
| Mink #30 | 45 | IT[e] | BDL | NO |
| Mink #33 | 19 | 2 | BDL | NO |
| Mink #14 | 7 | AE[f] | 0 | NO |

[a]NA = no adipose tissue
[b]FTA = freeze-thaw artifact
[c]BDL = below detection limit
[d]NO = not observed
[e]IT = insufficient adipose tissue
[f]AE = analytical error