Increasing Costs of Access to Resources Cause Rescheduling of Behaviour in American Mink *(Mustela Vison):* Implications for the Assessment of Behavioural Priorities

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Consumer demand studies of animal preference commonly involve the imposition of costs on access to resource compartments rather than direct costs on resource consumption. The aim of this study was to analyse how such increasing access costs alter the scheduling of behaviour, in order to investigate some potential problems with using this technique to measure behavioural priorities. We investigated the effect on mink of placing a cost (a weighted door) on the access to resources. Six mink were individually housed in closed economy test arenas. Each consisted of a home cage (containing food, water and a nest box), an empty compartment and six resource compartments with access to a box of hay, a water bath, a raised platform, a wire cylinder, a novel object, and small toys. Door weights were increased from 0 to 1000 g and the minks' behaviour was recorded on video for 8 h per day. As expected, increasing door weight reduced the number of visits to each compartment but lengthened each visit. Furthermore, as costs on access increased, there were changes in how mink behaved during each visit to resource-compartments. They became increasingly likely to interact with each resource during a visit, and they showed shorter latencies to interact with the resources. The time spent in compartments not interacting with the resources also declined. Overall, these changes in within-visit behaviour meant that the greater the cost on access, the greater the proportion of time spent with a resource devoted to actually interacting with it. Thus, the time spent with a resource over-estimated the actual use of that resource, particularly at lower access costs. These results illustrate the dangers of simply using time spent with a resource as a measure of consumption in studies of behavioural priorities. As a consequence of longer visits and more interactions, the total time spent swimming, manipulating the novel objects and occupying the hay box did not decline with increasing door weight. However, unexpectedly, this compensatory re-scheduling did not occur for all activities, because the time spent occupying the platform, manipulating the toys and the cylinder and the time in the empty compartment declined with higher access costs. This suggests that increasing access costs for unlimited periods of time with resources may, although not strictly valid for the production of demand curves, yield a ranking of behavioural priorities if high enough costs are used. Further research is necessary to see whether the minks' failure to defend time spent in the platform, manipulating toys and the wire cylinder and in the empty compartment does represent the low priority of these activities. Or whether some other factors, such as constraints on the ability to re-schedule the activities prevent mink from compensating for higher costs on access.

Applied Animal Behaviour Science, 2000: 66, 135-151, 9 figs., 1 table, 35 refs.

Feeding Growing Mink *(Mustela vison)* PCB Aroclor® 1254 Does Not Affect Baculum (Os-penis) Development

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Recent studies reported in the literature have shown a negative correlation between the concentrations of environmental contaminants and baculum (os-penis) mass and/or length in wild-trapped mustelids. Henny *et al*. (1996) found that organochlorinated contaminants, including polychlorinated biphenyls (PCBs), were significantly correlated with smaller testes and bacula in juvenile wild-trapped river otter from Oregon. Harding *et al*. (1999) reported a significant negative correlation between PCB concentrations (as Aroclor 1260) and baculum length in juvenile mink, but not in river otter trapped in British Columbia.

The objective of this study was to examine the effects of dietary exposure to a known concentration of the PCB mixture, Aroclor 1254, on baculum development in growing male mink under laboratory conditions.

Bull. Environ. Contam. Toxicol., 2000: 64, 443 – 447, 1 fig., 12 refs.

Proliferation of Periodontal Squamous Epithelium in Mink Fed 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD)

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The maxilla and mandible from 2 adult female mink fed 5.0 ppb 2.3.7,8-tetrachlorodibenzo-p-dioxin (TCDD) for 6 mo were grossly unremarkable, but histologically had nests of squamous epithelium within the periodontal ligament. There was osteolysis of the adjacent alveolar bone.

Veterinary and Human Toxicology, 2000: 42, 85 – 86, 10 refs.

Proliferation of Maxillary and Mandibular Periodontal Squamous Cells in Mink Fed 3,3', 4,4', 5-pentachlorobiphenyl (PCB 126)

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This report characterises squamous cell proliferation in young farm mink *(Mustela vison)* fed a diet supplemented with 0.024 ppm 3,3'.4.4'.5-pentachlorobiphenyl (polychiorinated biphenyl [PCB] congener 126). One to 2 months of dietary exposure to PCB 126 resulted in gross lesions of the upper and lower jaws consisting of mandibular and maxillary nodular proliferation of the gingiva and loose teeth. The maxilla and mandible of the PCB-treated mink were markedly porous because of loss of alveolar bone. Histologically, this osteoporosis was caused by proliferation of squamous cells that formed infiltrating cords. This report clearly documents the fact that the environmental contaminant PCB 126 can cause osteoinvasive squamous proliferation in young mink, although the dose used in the present study was 7 and 36 times higher than what is typically encountered in contaminated bird eggs and fish, respectively.

J. Vet. Diagn. Invest., 2000: 12, 477 – 479, 5 figs., 19 refs.

Studies on Influenza Viruses H10N4 and H10N7 of Avian Origin in Mink

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An influenza A virus, A/mink/Sweden/84 (H10N4), was isolated from farmed mink during an outbreak of respiratory disease, histopathologically characterised by severe interstitial pneumonia. The virus was shown to be of recent avian origin and closely related to concomitantly circulating avian influenza virus. Serological investigations were used to link the isolated virus to the herds involved in the disease outbreak. Experimental infection of adult mink with the virus isolate from the disease outbreak reproduced the disease signs and pathological lesions observed in the field cases. The mink influenza virus also induced an antibody response and spread between mink by contact. The same pathogenesis in mink was observed for two avian influenza viruses of the H10N4 subtype, circulating in the avian population. When mink were infected with the prototype avian H10 influenza virus, A/chicken/Germany/N/49, H1ON7, the animals responded with antibody production and mild pulmonary lesions but neither disease signs nor contact infections were observed. Detailed studies, including demonstration of viral antigen in situ by immunohistochemistry, of the sequential development of pathological lesions in the mink airways after aerosol exposure to H 10N4 or H10N7 revealed that the infections progress very similarly during the first 24 h, but are distinctly different at later stages. The conclusion drawn is that A/mink/Sweden/84, but not A/chicken/Germany/N/49, produces a multiple-cycle replication in mink airways. Since the viral distribution and pathological lesions are very similar during the initial stages of infection we suggest that the two viruses differ in their abilities to replicate and spread within the mink tissues, but that their capacities for viral adherence and entry into mink epithelial cells are comparable.

Veterinary Microbiology, 2000: 74, 101 – 107, 26 refs.