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Brief Communication

TOXICITY OF HALOGENATED OXYQUINOLINES TO DOGS. THE EFFECT OF FEEDING FOULED HERRING*

Acute clinical toxicity of halogenated oxyquinolines in dogs has been reported (*Schantz & Wikström* 1965, *Hangartner* 1965, *Müller* 1967). The clinical picture was comprehensively studied (*Lannek* 1972 b). The dose range in the clinical material was found to vary from about 7 to 250 mg per kg body weight (*Lannek* 1972 a).

In our experiments comprising large series of healthy dogs single doses, gradually increased to 1000 mg per kg, were invaryably tolerated without signs of disease. We have reported recently that a simultaneous feeding of a fat emulsion will increase the absorption of vioform from the intestine manyfold (*Lannek & Lindberg* 1972). By this procedure it was possible to produce a disease, apparently identical to the one observed clinically, and death, with a single dose of 300 mg per kg body weight. It seemed likely, however, that in clinical cases falling ill after ingesting lower doses, an unknown factor had been operating. We tested the hypothesis that this factor is phenolic substances using the same route of detoxification as halogenated oxyquinolines, i.e. coupling to glucuronic and sulfuric acids. It is known that such substances may be formed in excess by putrefactive processes in the intestine. Fresh herring was stored at 35°C for two days to obtain a foul decay. Thirty g fouled herring per kg body weight, mixed with commercial dog food ("Wift") was given each day for 1—3 days to 7 dogs weighing 8—12 kg. This feeding resulted in a strong urinary indican reaction (*Obermayer* 1890). Vioform and a fat emulsion (Intralipid, Vitrum), 5—10 Cal. per kg body weight were administered

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by a gastric tube on the last day. Three dogs fell ill (vioform doses 50 mg, 50 mg and 100 mg/kg, respectively), and two of them were killed in a moribund state. The symptoms were identical to those described (*Schantz & Wikström, Hangartner*), i.e. hyperexcitability, epileptic convulsions, profuse salivation and somnolence. The results support the hypothesis that an intestinal disorder, which is the incitement of therapy, may enhance the susceptibility to vioform significantly and thus be the major cause of the medical intoxication.

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