Biomarkers
- specificities & discussions -

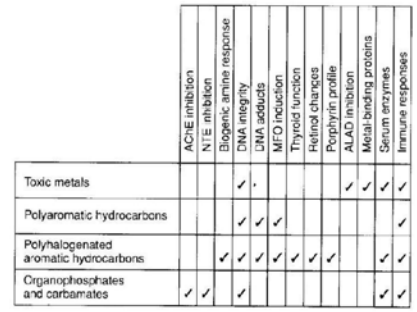

Figure 10.1 Biomarkers available for different classes of pollutants.

Table 10.2 Specificity of biomarkers

Highly specific	Inhibition of ALAD by lead
Moderately specific	Inhibition of AChE by OPs and carbamates
	Induction of porphyria by some PHAHs
Relatively nonspecific	Induction of MFO enzyme systems
	Sister chromatid exchange

Table 10.3 Biomarkers for different classes of pollutants

Environmental pollutant	Biomarker	Reliability index*
Toxic metals	DNA integrity	s
	Metal-binding proteins	s,d
	ALAD inhibition	s,d,p
	Immune response	s
	Levels of serum enzymes	s
PAHs	DNA/haemoglobin adducts	s,d,p
	DNA integrity	s
	MFO induction	s,d
	Immune response	s
PHAHs	Biogenic amines response	s
	DNA/haemoglobin adducts	s,d
	DNA integrity	s
	MFO induction	s
	Porphyrin profile	s
	Retinol changes	s
	Immune response	s
OPs	AChE inhibition	s,d,p
	Neuroesterases inhibition	s,d,p
	DNA integrity	s
	Enzyme profiles	s
	Immune responses	s

* s = signal of potential problem
d = definitive indicator of type or class of pollutant
p = predictive indicator of long-term adverse effect
Expanded from Shugart *et al.* (1989).

Table 10.4 Evaluation of biological responses to assess physiological injury caused by pollutants

	Response is often result of exposure to pollutant	Exposure to pollutant is known to cause response in free-ranging organisms	Exposure to pollutant is known to cause response in controlled experiments	Response is practical to measure
	1. Responses that meet all criteria			
Eggshell thinning	Yes	Yes	Yes	Yes
Reduced avian reproduction	Yes	Yes	Yes	Yes
ChE inhibition	Yes	Yes	Yes	Yes
ALAD inhibition	Yes	Yes	Yes	Yes
	2. Responses that met three criteria			
MFO induction	Yes	No	Yes	Yes
NTE inhibition	Yes	No	Yes	Yes
Thyroid dysfunction	No	Yes	Yes	Yes
Alteration in glutathione	No	Yes	Yes	Yes
	3. Responses that met two criteria			
Increased blood porphyrin	No*	No*	Yes	Yes
Alterations to neurotransmitter enzymes	*No*	No	Yes	Yes
Metallothionein induction	*No*	No	Yes	Yes

Table 10.4 Evaluation of biological responses to assess physiological injury caused by pollutants

	Response is often result of exposure to pollutant	Exposure to pollutant is known to cause response in free-ranging organisms	Exposure to pollutant is known to cause response in controlled experiments	Response is practical to measure
Release of organ-specific enzymes	No	No	Yes	Yes
Reduced mammalian reproduction	No	Yes	Yes	No
ATP inhibition	No	No	Yes	Yes
Adrenal dysfunction	No	No	Yes	Yes
Gonadal dysfunction	No	No	Yes	Yes
Pituitary dysfunction	No	No	Yes	Yes
MFO inhibition	No	No	Yes	Yes
Alterations to carbohydrate, lipid and protein metabolism	No	No	Yes	Yes
Changes in DNA/RNA content or synthesis	No	No	Yes	Yes
Changes in basal metabolic rate	No	No	Yes	Yes
Impaired thermoregulation	No	No	Yes	Yes
Impaired intestinal transport	No	No	Yes	Yes
Impaired renal function	No	No	Yes	Yes
Clinical blood chemistry	No	No	Yes	Yes
Haematological alterations	No	No	Yes	Yes

Biomarkers & effects
case studies, examples, summary

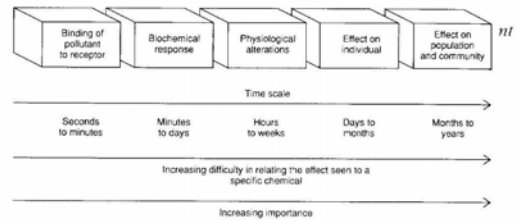

Figure 10.5 Linkages between biochemical, physiological, individual and population responses to pollutants.

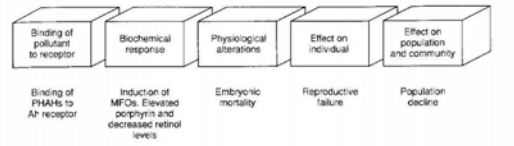

Figure 10.6 Linkages in the reproductive failure of fish-eating birds and the Ah receptor.

Pollutants
& reproductive
processes

Table 3.2 Major pollutant-related effects on reproductive processes

Species	Major findings	Biomarker studies
Mink	Complete reproductive failure of ranch mink fed Great Lakes fish (1). Population declines around the Great Lakes (2). Laboratory studies show sensitivity to PCBs and dioxins (3–5).	None carried out. Examination of reproductive impairment by dioxin equivalents should be made.
Seals	Decreased reproductive success and population declines in the Baltic and Wadden seas (6, 7) Experimental studies on Wadden Sea population suggest correlation with PCBs (8).	Marked changes in retinol and some changes in thyroid (9). Pathological changes in the Baltic population (10).
Raptoral birds	Decreased eggshell thickness leading to reproductive failure and widespread population declines (11, 12) Marked inter-species variation (13).	Caused by DDE (and closely related pesticide dicofol). Inhibition of Ca-ATPase (14) and effects on calmodulin (15).
Fish-eating birds	Decreased reproductive success, embryotoxic, behavioural effects and congenital abnormalities (2, 16, 17).	Good inverse relationship between reproductive success and dioxin equivalents based on AHH induction (18, 19).
Fish	Mortality of fry in hatcheries (20) and in field situations (21). Population effects seen in trout in New Brunswick and salmon in the Great Lakes (21, 22).	None carried out.

Regulation of reproduction

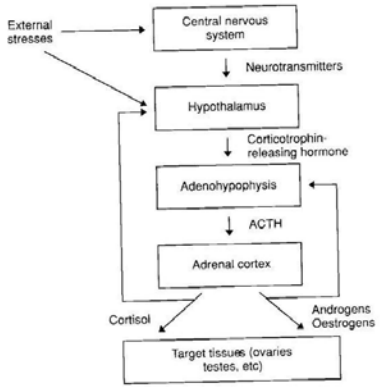

Figure 3.3 Outline of hormonal control of reproduction.

Regulation of reproduction

Table 3.4 Effects of PCBs and DDE on reproductive behaviour, success and hormone levels in doves

Chemical	Dose (ppm)	Courtship behaviour	Time to lay	Nest attendance	Reproductive success	LH	Steroid hormones	Reference
DDE	10	Decreased	–	–	–	–	–	(1)
		–	?	–	–	No effect	–	(2)
	40	–	–	–	Decreased	–	–	(3)
		–	Increased	–	–	Fail to show surge	–	(2)
	50	Decreased	–	–	–	–	–	(1)
	100	Decreased	Increased	Decreased	Decreased	–	–	(4)
PCBs	10	–	Increased	Decreased	Decreased	–	–	(5)
			Increased	–	Decreased	–	Some changes	(6)
	25	Decreased	Increased	Decreased	Decreased	–	–	(7)
Mixture	Low	No effect	Increased	No effect	Decreased	–	Some changes	(8)
	High	Decreased	Increased	Decreased (brooding only)	Decreased	–	Some changes	(8)

References: (1) Haegele and Hudson (1977); (2) Richie and Peterle (1979); (3) Haegele and Hudson (1973); (4) Keith (1978); (5) Peakall and Peakall (1973); (6) Koval *et al.* (1987); (7) Farve (1978); (8) McArthur *et al.* (1983).

Retinoid metabolism & PCDDs

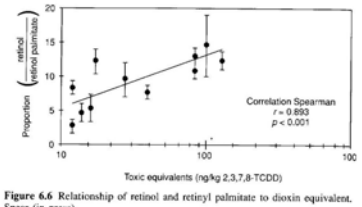

Figure 6.6 Relationship of retinol and retinyl palmitate to dioxin equivalent. Spear (in press).

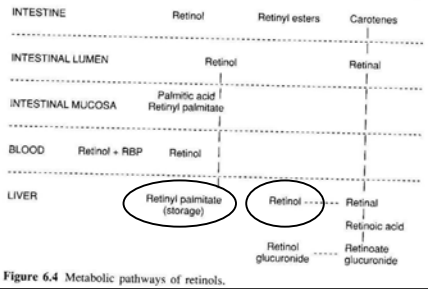

Figure 6.4 Metabolic pathways of retinols.

Variation of retinoid levels at different localities

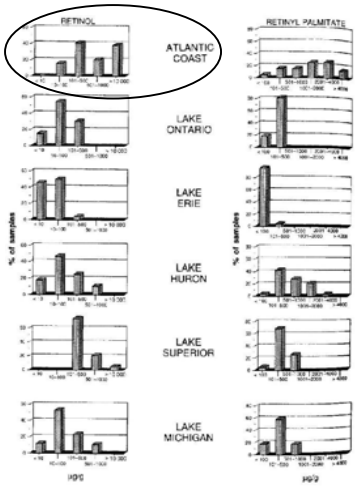

Figure 6.5 Retinol and retinyl palmitate in livers of herring gulls. After Environment Canada (1991).

Immunotoxicity of xenobiotics

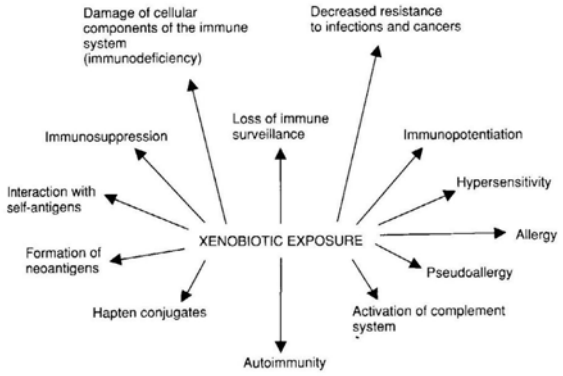

Figure 8.1 Potential immunotoxic effects of xenobiotics.

Immunotoxicity of xenobiotics

Table 8.2 Environmental chemicals that are considered potentially immunotoxic

Chemical class	Immunotoxic effects*
Metals, organometals, metalloids	Suppression of HMI, CMI, NSR; induction of contact hypersensitivity; impairment of host resistance to infections and tumours
Halogenated hydrocarbons (aliphatic and aromatic), aromatic hydrocarbons	Suppression of HMI, CMI; impairment of host resistance to infections and tumours
Heterocyclic oxygen-containing compounds including epoxides, furans, dioxanes	Suppression of HMI, CMI; impairment of host resistance to infections and tumours
Carbamates	Modulation of CMI, HMI; modulation of host resistance to infections and tumours
Organophosphates	Suppression of HMI, CMI; impairment of host resistance to infections and tumours

* Abbreviations: HMI: humoral-mediated immunity, CMI: cell-mediated immunity, NSR: nonspecific response.

Xenobiotics and thyroid regulation

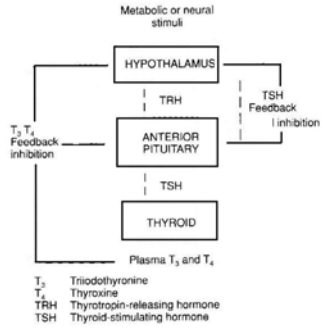

Figure 6.1 Overall regulation of thyroid hormones.

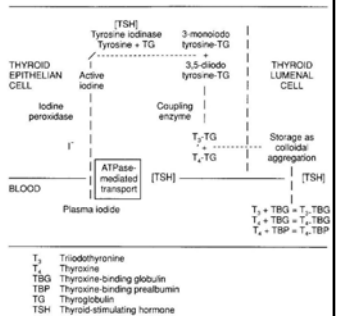

Figure 6.2 Metabolic pathways of the thyroid hormones.

Xenobiotics and thyroid parameters

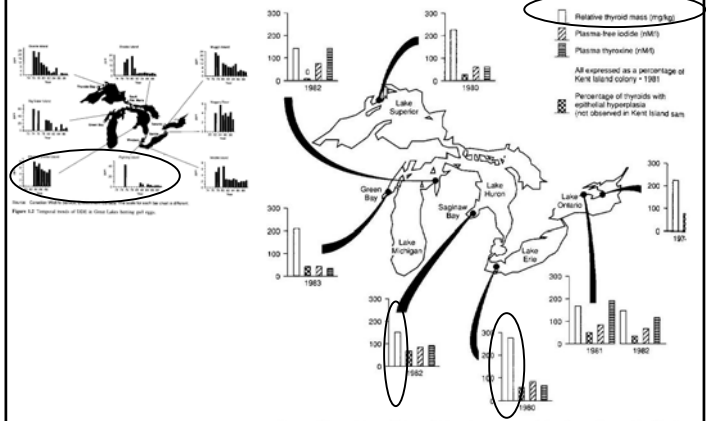

Figure 6.3 Variation of thyroid-related parameters in herring gulls on the Great Lakes. G. Fox, Canadian Wildlife Service, Environment Canada (1991).

Xenobiotics *(non-AcChE)* and neurotoxicity

Biomarkers = neurotransmitter levels

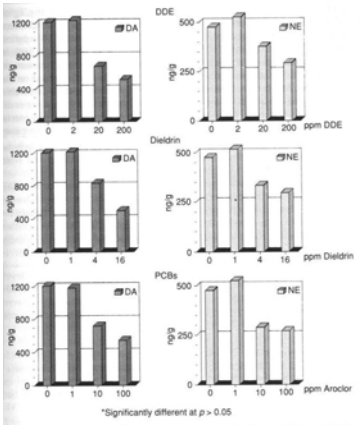

Figure 2.11 Effect of DDE, dieldrin and PCBs on the levels of DA and NE in the brain of the ring-dove. After Heinz *et al*. (1980).