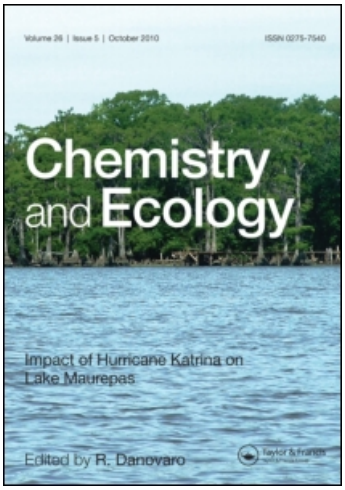

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RISK ASSESSMENT AND MANAGEMENT OF CONTAMINATION OF EELS (*ANGUILLA* SPP.) BY PERSISTENT XENOBIOTIC ORGANOCHLORINE COMPOUNDS

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Contamination of anguillid eels by organochlorine pesticides and PCBs is reviewed using a risk assessment-management approach. Quantitative structure-activity relationships, biomonitoring of body residues and field and laboratory studies demonstrate hazards and risks of persistence; organic carbon binding, sediment transport and sinks; volatility, aerial transport and deposition; hydrophobicity/lipophilicity, bioaccumulation, bioconcentration and dietary biomagnification. Risks of acute narcotic toxicity are generally lower than those of chronic effects. Cause-effect relationships and critical loadings are, however, unclear. Critical environmental and body residue levels to protect eels and animal and humans consumption must be formulated and interpreted with caution. Risks for eels at different life-stages and in different habitats are compared with respect to routes of uptake, elimination, bioaccumulation and biomagnification. It is concluded that organochlorine contamination has not been a major cause of recent declines in eel recruitment. The use of eels in biomonitoring and biomarker studies are recommended. Appropriate methodologies and uses in risk assessment and management are discussed.

Keywords: Organochlorine pesticides; PCBs; *Anguilla* spp.; risk assessment; risk management; ecotoxicology; human food residues

INTRODUCTION

Xenobiotic organochlorine pesticides and compounds such as polychlorinated biphenyls (PCBs) are normally of low solubility and hence concentration in water. They are, however, potentially hazardous

chemicals because of their persistence, widespread distribution and because they can bioaccumulate in biota. They can therefore potentially exert high toxicity relative to low ambient environmental concentrations (Phillips, 1995). Anguillid eels are of considerable interest in relation to such pollutants from both biological and practical points of view. First, they are particularly prone to accumulation of liposoluble organochlorines because of their high body fat content and ubiquitous life style. Organochlorine toxicity has been suggested as one of the possible causes of the drastic falls in recruitment of eels to Europe and North America over the last 15–20 years (Knights *et al.*, 1996). High contaminant burdens could also, through dietary biotransfer and biomagnification, affect secondary consumers such as piscivorous birds and mammals. Furthermore, eels (fresh-cooked or smoked) are consumed by man, raising concerns about human health effects.

The other main interest is in the potential uses of eels as biomonitors of spatial and temporal trends of contaminants in aquatic environments. Body burdens, due to bioaccumulation, can provide valuable information on levels of organochlorines in water and sediments that are generally too low to measure accurately. Furthermore, eels are tolerant of confinement and laboratory stresses and can be useful in toxicity testing and studies of uptake, elimination and pathophysiology. Metabolic responses can also be used as biomarkers of dose-response and pathological relationships and critical concentrations.

This review focuses on these issues, updating the general one of Brusle (1991). Many past studies on eels have, however, been carried out piecemeal, often lacking clear and quantifiable objectives and scientific rigour. This has been reflected in uncertainties about impacts on eels and eel stocks and in deriving environmental quality standards and recommended residue levels in eels consumed by man and other animals. To achieve a more objective overview, this review uses a risk assessment and management approach, analagous to those developed in fields such as occupational health and safety.

Risk assessment is gaining recognition in ecotoxicology because it involves structured and rigorous approaches, based on best available scientific data. It attempts to quantify hazards (i.e. potential for damage) and risks (i.e. actual likelihood of damage). The more robust the estimations of exposures and effects, the more objective and realistic

can be the guidance as to appropriate risk management procedures (Suter, 1995: DoE, 1995). This review discusses applications of risk assessment to organochlorine contamination of eels, by hazard identification (sources, emissions, toxic species and exposure assessment), hazard analysis (receptor toxic effects), risk assessment (environmental exposure-effects and vulnerability relationships) and risk evaluation (acute and chronic toxic impacts). This leads to a critical overview of past, present and possible future risk management approaches, both with regards to protecting eel populations and stocks and to protecting consumers, including man. The use of eels in biomonitoring and in biomarker studies are considered critically (including sampling and analytical methods and data interpretation). In addition to the published literature, information has been gathered on studies by governmental, fisheries and other agencies. Information has also been supplied by members of the European Inland Fisheries Advisory Commission (EIFAC, FAO) Working Party on Eel.

The main objectives of this review are to provide information to aid ecotoxicologists and fisheries scientists, managers and agencies in planning and interpreting studies and to aid decision-making about management options.

HAZARD IDENTIFICATION

Before considering specific hazards for eels (and their consumers), it is important to define the general hazards associated with persistent organochlorines.

Sources and Chemical Species

The use of xenobiotic organochlorine insecticides increased markedly after World War II as agriculture became more intensive. Other important uses have been in timber treatment, moth-proofing and control of nuisance insects and disease vectors. Of particular concern have been pesticides such as DDT (and its metabolites such as DDE), the cyclodienes ('drins' such as aldrin and dieldrin) and cyclohexanes (such as lindane, gamma-HCH). Attention later came to encompass other complex anthropogenic halogenated compounds, arising mainly

from industrial applications (e.g. hexachlorobenzene, HCB and polychlorinated and polybrominated biphenyls, PCBs and PBBs) and from combustion processes (e.g. dioxins and dibenzofurans). Many of these compounds comprise a range of isomers or congeners. PCBs, for example, occur in up to 209 CB congener forms and are sold in commercial formulations, such as Arochlor 1240 (40% chlorine) and Arochlor 1260 (60% chlorine). They are stable di-electric compounds, making them ideal as stabilisers in plastics and in fire-retardant products. Their main use has been in hydraulic fluids and in cooling systems for electrical transformers and capacitors. The wide range of organochlorine isomers/congeners make it difficult to identify accurately specific hazards, each showing differences in persistence, liposolubility and toxicity. Furthermore, transformations to other toxic chemical forms can occur through physicochemical processes (e.g. photoxidation) in the environment and, by metabolism, in biota (Lyman, 1995). For example, DDT can be metabolised by some animals to more toxic DDE. Thus careful sample collection, preparation and clean-up and sensitive analytical methods are essential to identify accurately and quantify organochlorine compounds. This will be discussed later in relation to toxicity assessment and biomonitoring.

Exposure Assessment

Acute toxicity hazards can arise from concentrated point-source emissions (e.g. during manufacture, transport and direct application) and from accidental spillages. Such events are, however, rare. Generally, long-term chronic emissions from non-point sources are of greater concern, e.g. through spray drift, surface run-off, soil leaching, waste disposal and aerial deposition. Illegal disposal and diffuse discharges can elevate levels in sewage effluents and sludges. Particularly severe chronic contamination tends to arise in river systems with high densities of intensive agriculture, industry and urban populations. Downstream transport exacerbates problems in lower river stretches, in estuaries and in enclosed and poorly-flushed seas, such as the Baltic and North Sea. Such problems have had to be tackled by international initiatives, leading to restrictions on manufacture and use and the eventual banning of the most hazardous organochlorines. Residual concentrations should therefore have decreased subsequently. These

issues are discussed in more detail later, with reference to risk management and the use of eels as biomonitors. Hazards to animals and man consuming eels are also reviewed.

Environmental Hazards

The principal exposure hazards of organochlorines arise from their physiochemical properties which make them recalcitrant to degradation and that affect their environmental distribution and bioavailabilty. These hazardous properties can be predicted via quantitative structure-activity relationships (QSARs), which depend on molecular bonding, structure and functional characteristics (e.g. see Lipnick, 1995; Phillips, 1995; Suter, 1995).

Anthropogenic xenobiotic organochlorines are resistant to physicochemical and biodegradation because of the stabilising actions of chlorine substituents and bonding. Half-lives can therefore be long, ranging in water from > 190 days for HCH to > 700 days for DDT (NRA, 1995a). Half-lives in soils and sediments are, however, generally very much longer, e.g. 3–25 years for DDT. This is because organochlorines can sorb to particulate organic carbon which protects them from photolysis, oxidation and microbial aerobic degradation. Tendencies to bind to carbon can be predicted from *n*-octanol-carbon partition coefficients (K_{oc}). For example, HCH with a log $K_{oc} < 4$ binds less strongly than DDT and PCB congeners, with log $K_{oc} > 5$ and > 4–6 respectively. Inorganic particles with only surficial bacterial biofilms show a low organochlorine loading. Conversely, humus and detritus particles with high surface area-volume ratios and TOC (total organic carbon) content can show both surface adsorption and absorption into the matrix. Fine organic sediments entering surface waters can be important in downstream transport and dispersion of organochlorines and hazard levels will increase in lentic depositing ecosystems (Larsson, 1985). Although sediment-binding may limit bioavailability, remobilisation can occur, e.g. due to soil erosion or resuspension of sediments by storm-flows or dredging. Bioturbation processes can also be important, as can digestive action during passage through the guts of detritivores and filter-feeders (Ingersoll, 1995). Hazards are therefore enhanced for organisms living in close association with contaminated sediments and for consumers of such organisms.

Xenobiotic organochlorines are non-polar and highly hydrophobic and lipophilic. Their aqueous solubility is therefore very low, generally < 1 μg l^{-1}. This led to early assumptions that toxic hazards in aquatic environments would be low. The lipophilic properties of organochlorine pesticides are fundamental to their action, i.e. ease of penetration of the body wall of target species. Liposolubility also promotes accumulation in body fats. This property, together with recalcitrance to enzymatic degradation, enhances organochlorine toxicity. What was not recognised initially was that these properties (also possessed incidentally by other non-pesticidal xenobiotic organochlorines) would increase the hazards of bioaccumulation of residues in the body fat of non-target animals.

Liposolubility can be predicted from octanol-water partition coefficients (K_{ow}) (Connell, 1995; Lipnick, 1995). Bioconcentration from water and/or sediments occurs for organochlorines with log $K_{ow} < 5$. Uptake is then primarily determined by the concentration gradient between water and blood (Connell, 1995). Hazards would then be expected to be highest for small organisms with high surface area:volume ratios, coupled with high ventilatory rates. Conversely, organochlorines with log $K_{ow} > 5$ tend to bind more strongly to lipids and are preferentially biotransferred from prey organisms to predators via their diet. Biotransfer is believed to be the primary route for organochlorines with log $K_{ow} > 6$. Thus biomagnification can occur between successive trophic levels, posing potentially high hazards for top carnivores, including man. Comparisons of relative hazards to different eel life stages (and hazards to consumers) are discussed below. Above log $K_{ow} = 6.5$, rates of bioaccumulation/magnification tend to decrease because molecular size increases and entry through cell membranes is inhibited.

Henry's law constant (H) is another key QSAR, being effectively an air:water partition coefficient. Compounds with log $H > 2$ show an increasing tendency to volatilise. Organochlorine pesticides and PCBs do not generally volatilise easily, but this is often the main route of loss from soils and water and hence for aerial dispersal. This is particularly so for organochlorines such as HCBs with a log H of 4. Subsequent aerial deposition explains the presence of low concentrations of organochlorines at sites distant from obvious sources, e.g. in isolated lakes and even remote continents, such as Antarctica.

A further complicating factor in assessing hazards of organochlorines is that different isomers and congeners have different QSAR

characteristics. For example, DDT typically degrades to DDD which binds preferentially to sediments, whereas in biota DDT can be metabolised to more recalcitrant DDE. Aldrin is metabolically broken down to the more stable dieldrin form. PCB congeners consist of a biphenyl ring with different patterns of 1–10 chlorine substituents. Penta-CBs with a 4,4′ substitution are more recalcitrant than other penta-CBs and hexa-, hepta- and octa-CBs are even more persistent. QSAR-related hazards are discussed further below in relation to spatial biomonitoring.

HAZARD ANALYSIS

Receptor Toxic Effects

Acute toxicity tests show that 96h LC50 values for persistent organochlorines are generally low for fish ($< 50\ \mu g\,l^{-1}$) compared to environmental concentrations. Different organochlorines have slightly different toxicities. PCB products and residues comprise mixtures of congeners with different toxicities. Relative toxicities can be related to a commercial mixture of known composition, such as Arochlor 1240 or 1260. More precise estimates can be derived by comparing the toxicity of a congener to that of highly toxic 2, 3, 7, 8-tetrachlorodibenzo[*p*]dioxin (TCDD) and calculating a toxic equivalent factor (TEF) (Safe, 1990). Multiplying TEFs for each CB congener and summing them yields an overall TCDD toxic equivalent (TEQ) for a mixture. This can then be compared with a reference value. Few TEFs or critical TEQs have been determined for fish, let alone eels, and reference has to be made to values derived to estimate risks to man of consuming contaminated fish. Thus, for example, the most toxic CBs tend to be coplanar, especially CB 126 which has a TEF of 0.1, compared to CB 189 with a TEF of 0.001.

A major problem in determining toxic effects of xenobiotic organochlorines is that they do not directly form or break covalent bonds in biological systems. Therefore, they do not cause specific biochemical lesions and affect discrete enzymes, metabolic pathways or structural chemicals. Instead, they appear to act as non-polar and non-specific narcotics when a critical concentration is attained. For most organochlorines of high K_{ow}, this concentration is similar across a wide range of animals, at 3.2–12.2 $mol\,kg^{-1}$ wet weight (Connell, 1995). They appear to act primarily by entering cell membranes, due

to their liposolubility, and upsetting general membrane structure and functions by physical distortion. Thus they can disturb ion transport mechanisms, hydromineral balance, neurophysiological function and control of hormonal systems. In the natural environment, sub-lethal symptoms (with disturbances in respiration, liver structure and function and carbohydrate metabolism) are difficult to distinguish from generalised stress-response syndromes. True cause-effect relationships are often obscured by responses to other concurrent stresses, such as low dissolved oxygen (DO) or other pollutant chemicals. Acutely toxic concentrations (e.g. due to a major spillage) are rarely attained in the natural environment and bioaccumulation generally poses the greatest hazards. This can cause chronic sublethal effects, e.g. by reducing tolerance to other stressors or by lowering fecundity. Internal concentrations could become lethal if a critical loading level is exceeded, e.g. due to release of bioaccumulated residues on mobilisation of fat reserves.

Indirect chronic hazards can also result from normal protective biotransformations. Organochlorines can induce the production of hepatic mixed-function microsomal monoxygenase (MFO) enzymes (especially P-450 enzyme). These act to introduce polar functional groups to render the organochlorine molecules more water soluble. They are then conjugated with peptides and sugars (such as glucuronide) to aid excretion. MFOs can, however, convert some compounds to metabolites that form covalent bonds and affect essential biochemicals, such as steroid hormones, certain vitamins and fatty and bile acids (Di Giulio *et al.*, 1995). Chronic disturbances of metabolic, endocrine and immunological systems can result. Organochlorines may also have genotoxic and other carcinogenic effects (Shugart, 1995). Such potential effects are discussed later, as are the potential uses of metabolic and physiological responses as 'biomarkers' to assess the critical levels at which organochlorines begin to exert deleterious effects.

RISK ESTIMATION

The Life Cycle and Habits of Eels

To help estimate the risks to eels of exposure to organochlorine hazards, it is necessary to understand their basic life history and habits. First, they have a unique catadromous life cycle which can potentially

lead to exposure during all life stages. For example, European eels (*Anguilla anguilla* L.) (as far as is known) only breed once in the Sargasso Sea in the western Atlantic and migrate as planktonic leptocephalus larvae via oceanic currents towards Europe (Tesch, 1977; Deelder, 1984). On the Continental Shelf, they metamorphose into transparent 'glass eels' and then orientate towards and enter estuaries from Scandinavia to the Mediterranean. After metamorphosis into the pigmented 'elver' stage, some eels migrate upstream whereas others remain in estuarine and coastal waters. Immature 'yellow' eels may resume upstream migration in future years, colonising deep into river catchments and still waters. They commonly feed on benthic macroinvertebrates, although larger eels can become piscivorous. *A. anguilla* males mature ('silver') after an average of about 9 years at $>30-35$ cm. Females mature after 12–14 years at $>45-50$ cm before migrating downstream and back to the breeding grounds. Eels are, however, essentially warm-water species (optimum 25–30°C), and become quiescent at temperatures below about 8–10°C, lying buried in sediments. Growth rates are therefore very temperature-dependent and freshwater life spans can vary greatly. They are not only euryhaline but are also tolerant of a wide range of natural environmental stressors, such as varying temperatures and low DO and pH. Thus eels can be exposed to pollutants over long time scales in a wide range of aquatic environments. Freshwater eels also tend to live most of their lives in localised areas. These features, coupled with their benthic habits, tendencies to bury themselves in sediments and their carnivorous diet, greatly enhance their chances of exposure to organochlorine pollutants.

Environmental Exposure Risks; Evidence from Biomonitoring

In the absence of major releases of organochlorines, the highest risks to eels would be expected to arise from long-term chronic exposure from water, sediments and diet in agricultural and/or industrialised catchments, estuaries and enclosed seas. Although there have been few quantitative studies of such risks, indirect evidence is available from spatial biomonitoring of eels. Restrictions on and/or banning of the use of the most hazardous organochlorines should have produced reductions in residue levels over time. Temporal biomonitoring is discussed later, when considering the results of past risk management strategies.

Brusle (1991) reviewed 27 studies, spanning 1969–1989, on organochlorine residue levels in *Anguilla anguilla* from various sites in Europe and in *A. rostrata* in North America. These have proved invaluable in pinpointing 'hot spots' of risk, due to spillages, disposal or unexpected sources and emissions. They have also clarified risks arising from transport, transformation and deposition processes.

Important early studies were carried out in North America as organochlorine contamination from agriculture and industry increased in the 1950–60s, especially in the Great Lakes and in industrialised estuaries and associated coastal waters. Eels were often found to have bioaccumulated organochlorines of high K_{ow} to levels exceeding those in other fish species, because of their fat content. Acutely toxic effects were implicated in large eel kills in the St. Lawrence River and estuary from the 1960s, peaking in the early 1970s. Castonguay *et al.* (1989) analysed organochlorines in eels captured during migration down the river or from different waters in the catchment. Data for 14 pesticides and PCB congeners were subjected to principal component and discriminant analyses. Bioaccumulated residues were related significantly to contaminant profiles in water and sediments typical of parts of Lake Ontario, other lakes and tributaries. Particularly indicative were residues of mirex, a cyclodiene-type pesticide. Mirex caused localised contamination in Lake Ontario and some tributaries due to spills and effluent disposal from a manufacturing plant on the Niagara River (until production was banned in the mid-1970s). This study showed that xenobiotic organochlorines are so persistent in eels that the geographical origins of emigrants could be distinguished and risks related to localised patterns of environmental contamination.

These findings helped stimulate further studies of organochlorine pollution and controls on usage and/or banning of the most hazardous ones. Biomonitoring in the 1990s of emigrant female eels (8–16) years old) showed residue levels had decreased (Hodson *et al.*, 1994). However, analyses over a seven week sampling period showed that later captures tended to be the most contaminated. These eels had possibly come mainly from the most distant and still relatively highly contaminated parts of Lake Ontario. Geographical origins could, however, no longer be reliably distinguished by principal components analysis, mainly because many concentrations were approaching detection limits. Despite controls on organochlorine usages, low concentrations of some

pesticides were still present in most samples. Sediment transport down main rivers was implicated for organochlorines like mirex. There was, however, evidence of more widespread chronic contamination due to aerial transport and deposition, in contrast to the localised high exposure risks typical of the 1970–80s.

Studies of organochlorine contamination of wild eels in Europe gathered pace during the 1970–80s as their value as biomonitors (and the potential risks of biotransfer to consumers) were recognised. Brusle (1991) cited 11 publications on eels from various fresh waters and the North and Baltic Seas. Many again showed that eels were at greater risk of bioaccumulation than other fish. Typical residue concentrations of DDT, DDE and DDD in the 1980s in eels from more contaminated waters were 0.14–2.00, 0.14–0.19 and 0.04–0.10 $mg\,kg^{-1}$ (whole body fresh weight) respectively. Some samples from the lower River Elbe in Germany had concentrations as high as 8 $mg\,kg^{-1}$ (Kruse *et al.*, 1983). For 'clean' waters (rearing ponds and isolated lakes), concentrations were in the order of 0.07–0.13 mg kg^{-1} DDT, 0.20 DDE and 0.04 DDD. Lindane concentrations in eels from 'polluted' waters were 0.11–0.90 (0.11–2.49 total HCH isomers) and from 'clean' waters, 0.008–0.11 $mg\,kg^{-1}$. For dieldrin and total PCBs, higher levels found were in the region of 0.02–0.07 and 0.50–3.58 $mg\,kg^{-1}$ and lower levels were about 0.05 and 0.27 $mg\,kg^{-1}$, respectively. Some of these levels (e.g. in the Rhine and Elbe) were above those recommended for human consumption.

Although these figures give some indications of risk factors in the 1970–80s, realistic comparisons between studies are often complicated by high variability in the data and differences in (or lack of clear information on) sampling and analytical methods and data presentation. Recognition of such problems has led to more careful biomonitoring of spatial and temporal trends and to the promotion of more effective risk management.

An example of the value of localised biomonitoring involved analyses of eels from the Rivers Mole and Taw in SW England (Hamilton, 1985). Dieldrin and DDE residue levels were elevated in 1983 (means > 50 and $> 300\,\mu g\,kg^{-1}$, respectively) at certain sites compared to controls and expected background levels of $< 1\,\mu g\,kg^{-1}$ dieldrin and $< 3\,\mu g\,kg^{-1}$ total DDT. This helped pinpoint a drainage discharge near a disused sheep dip. Activated carbon was used to adsorb contaminants.

Another illustrative example comes from a routine survey by the National Rivers Authority (NRA, now the Environment Agency).

Unexpectedly high residue levels were found in eels from four rivers in SW England, especially the Newlyn River. Mean aldrin, endrin and dieldrin concentrations were 0.10, 0.13 and 7.50 mg kg^{-1}, respectively, in Newlyn eel samples (NRA, 1989). The uses of such cyclodienes had, under EC Directives, supposedly been restricted progressively to reduce risks. Contamination was traced to the use of aldrin for a special limited purpose, protection of daffodil bulbs against large narcissus fly. Aldrin has a high K_{oc} and was sorbing strongly to soil particles but when these were eroded, it was being chemically oxidised to dieldrin in the river. Some contamination was also traced to pesticide treatments for protecting seed potatoes from pests in former permanent grass fields. Further controls on pesticide applications were imposed and residues in eels have since decreased.

An example of risks from a point source of known contamination arose in the Vanajavesi catchment in Finland (Tulonen & Vuorinen, 1996). Some 1200 l of PCB solutions were discharged from 1956 into Lake Kernaalanjarvi from a paper mill laboratory, until disposal was banned in 1984. PCB concentrations in fish from other Finnish waters have generally been low (< 0.05 mg kg^{-1} fresh body weight). Those for seven organochlorines in the Vanajavesi were low (< 18 μg kg^{-1}). However, residues of PCBs in pike, pike-perch and bream from the lake and downstream were found to be so high in the 1980s that consumption by man was forbidden. The catchment does not support natural immigration of eels but stocking occurred in the 1960–70s. Subsequent analyses have revealed the persistence of PCBs in the environment and in eel body lipids. Body residues determined in 1989–90 were still as high as 33.8 mg kg^{-1}, up to 7–15 times those in other fish. Although residue levels had decreased to < 2 mg kg^{-1} by 1993, they were still several times higher than in other fish on a total fresh weight basis. The eels were all large (> 65 cm) and hence probably older females. Most of the males and some females had probably emigrated in earlier years. Thus even if PCB levels in water and, especially, in sediments had been decreasing, residues in eels were probably still high if allowances are made for growth dilution (as discussed further below). Unfortunately, sample sizes from earlier years were too small to make valid comparisons. Residue levels in body fats were < 10 mg kg^{-1}. Dilution may have occurred as fat had been accumulated by up to 40% of body weight in some eels. Organochlorine concentrations were correlated

with fat content but not length or weight, emphasising the bioaccumulation hazards posed by organochlorines with high K_{ow} values. More informative risk assessments were, as in nearly all eel biomonitoring studies, confounded by insufficient data. Furthermore, variability between samples was high because the geographical origins of the large eels were unknown. Some had probably migrated upstream after stocking and had lived in uncontaminated waters until being captured on downstream emigration. Means of optimising sampling to avoid such confusion are discussed later.

Biomonitoring to compare supposedly 'clean' stillwaters and possibly detect local hot spots was carried out by Mason (1993) of British wetlands. The primary aim was to see if bioaccumulation in eels could pose a risk to otters eating them. Mason analysed organochlorine residues in bulked samples of eels collected from 11 reedbed wildlife-conservation sites in 1991. Dieldrin, DDE and PCB levels were <0.03, <0.13 and $<0.13\,mg\,kg^{-1}$ (whole wet weight) respectively, although single maxima (in different wetlands) were 0.21, 0.27 and $0.91\,mg\,kg^{-1}$, respectively. These levels were lower than those found in most UK rivers in extensive British surveys conducted as part of on-going monitoring of residues of organochlorines in human food (as discussed below). There had been no known land- or water-based discharges to the study sites, but aerial deposition must have been occurring. Aerial deposition is also implicated in some other waters remote from agricultural or industrial influence, e.g. in eels from the unpolluted Brazo de la Torre, the main river of a national park in SW Spain (R. McDonald, personal communication).

A study of eels from a relatively small and isolated lake in S. Scandinavia in 1988 showed residue levels of 28 organochlorines were low but that concentrations of individual compounds matched those determined in atmospheric fallout (Larsson *et al.*, 1991). Thus biomonitoring emphasises that although organochlorines generally have low Henry's law constants, volatilisation, aerial transport and deposition can result in widespread dispersal and contamination.

In contrast to the 'local' biomonitoring studies discussed so far, extensive and longer-term surveys have been instituted for some major European catchments and seas, similar to those in North America. The importance of transboundary pollution was emphasised by the massive Sandoz accident in the River Rhine in November 1986. In putting out a

fire at a chemical warehouse near Basle in Switzerland, large quantities of a mixture of organic chemicals and mercury were washed into the river, causing the deaths of about 500,000 fish, including 150,000 eels, for up to 500 km downstream (Guttinger & Stumm, 1992). Although involving organophosphate compounds rather than more persistent organochlorines, the accident was a major stimulus to international initiatives, such as the Ecological Rehabilitation of the River Rhine Programme.

Eels have performed an important role in biomonitoring of the Rhine and Meuse since 1977 by the Netherlands Institute for Fisheries (RIVO-DIO). These have revealed general spatio-temporal trends, but also have pin-pointed particular risks. For example, certain PCB congeners have been found to be unusually high in eels from the River Roer, a tributary of the Meuse, and the Zuid-Willemsvart canal, near the Belgian border (Figure 1). De Boer and Hagel (1994) and de Boer *et al.* (1996) concluded these are due to leakages from hydraulic systems in upstream German and Belgian mining areas. This is supported by the relatively high content of lower chlorinated CBs (e.g. tetra-CBs 52 and 77), typical of commercial hydraulic fluids, compared to more chlorinated (e.g. penta-CB 126) congeners. Another interesting risk feature identified in the Roer in the 1980s was the appearance of residues in eels of tetrachloro-benzyltoluenes (Ugilecs). These had been developed as replacements for PCBs until their hazardous nature was recognised and they too were banned.

In addition to pin-pointing localised risks, the Rhine studies throughout the 1970–80s generally revealed highest organochlorine contamination in eels from the downstream parts of the Rhine basin, especially in the sedimentation areas of Haringvliet-oost (van der Valk *et al.*, 1989; de Boer & Hagel, 1994; de Boer *et al.*, 1996). PCB levels often exceeded those in eels from other European waters (de Boer & Hagel, 1994) and they were comparable to those in the Great Lakes and Delaware Rivers. Levels were only exceeded by those in eels collected in the 1970s from the Hudson River, New York (Sloan *et al.*, 1983). Contaminated sediments are strongly implicated, as was the case for *Anguilla rostrata* (and 4 other species) in surveys in New Jersey, USA, coastal waters polluted by sewage sludge and dredge-dumping (Kennish *et al.*, 1992). PCB residue levels in eels in the late 1970s/early 1980s averaged 1.52–7.2 mg kg^{-1} (as the commercial mixture Arochlor 1254). Many approached the US

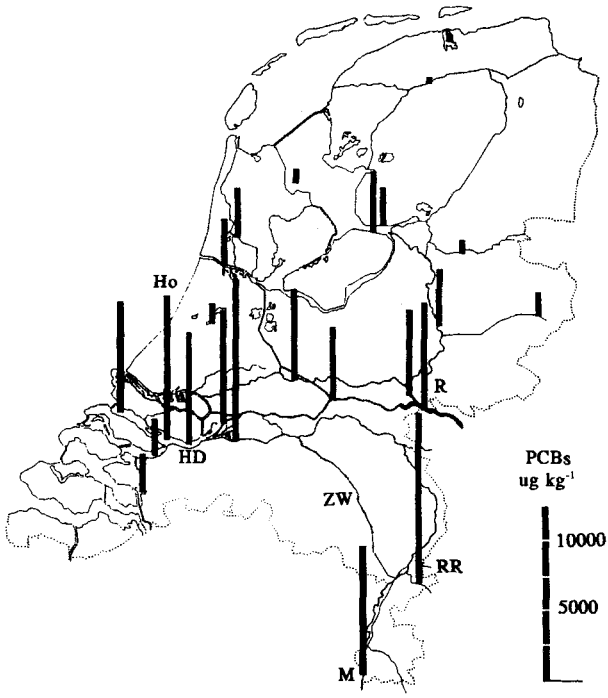

FIGURE 1 Map showing the sum of mean concentrations (μg kg^{-1} whole wet weight) of seven PCB residues in yellow eels sampled from Dutch fresh water sites in 1995. Congeners summed were CBs 28, 52, 101, 118, 138, 153 and 180. R = Lobith, River Rhine, M = Meuse, RR = River Roer, Ho = Haringvliet-oost, HD = Hollands Diep, ZW = Zuid Willemsvaart Canal. See text for further explanation. Adapted from de Boer *et al.* (1996).

Environmental Protection Agency/Food and Drug Administration limit of 2 mg kg^{-1} (Table I).

Transport of sediment-bound organochlorines was clearly demonstrated by Castonguay *et al.* (1989) for the persistent pesticide mirex. As discussed above, contamination from a production plant was found in 1963–8 to be locally restricted in Lake Ontario. By 1982, however, contamination was found to have passed 400 km downstream, in the St. Lawrence River. Its continued presence since production was banned in the 1970s emphasises its persistence in organic sediments.

Total organochlorine residue levels also tended to fall in the Rhine and Meuse during the 1980s. Some increases in PCBs have occurred, especially in the more recalcitrant higher chlorinated congeners. Residue levels in

TABLE I Representative environmental quality standards (EQSs) and food residue guidelines/standards for OC pesticides and PCBs

OC compounds	*EC–EQS (ng l^{-1})*	*Food residue limits (mg kg^{-1})*
Dieldrin	10 (all waters)	0.2 MRL[a] meat fat
		<0.3 MRL, fish liver
		0.1 Canadian Guideline
Total DDT	25 (all waters)	5 MRL, meat fat
		1.5 MRL, fish liver
		5 USA EPA[b], human food
		0.2 Dutch food standard
		0.007 ADI[c]
gamma-HCH	100 (fresh water)	2 MRL, meat fat
	20 (sea water)	0.05 MRL, fish liver
HCB	30 (all waters)	0.10 MRL, fish liver
Total PCBs	None set	0.05 PARCOM upper limit for fish muscle
		1 Dutch, fish flesh[d]
		5 Dutch, eel flesh[d]
		2 USA EPA, human food
		0.02 Canadian food TEQ[e]
		0.065 fish reproduction TEQ[f]
		(<0.05 for otters[g])

Key; (a) MRL, maximum residue levels based on FAO/WHO Codex Alimentarius Commission recommendations; (b) US EPA, US Environmental Protection Agency; (c) ADI, acceptable daily intake (WHO) for a 70 kg adult; (d) Dutch 1984 standards (see de Boer *et al.*, 1994); (e) TEQ, toxic equivalent compared to dioxins; (f) TEQ at which chronic effects on reproduction appear to occur in some Great Lakes fish (see Hodson *et al.*, 1994); (g) maximum daily intake recommended by Mason and co-workers. See text for further explanations.

the 1995 survey (Figures 1 and 2) show that concentrations are still elevated in areas of sediment deposition, such as the Haringvliet, compared to upper river sites, as at Lobith. Disturbance of contaminated sediments (due to dredging) is possibly important but local sources cannot be excluded. De Boer and Hagel (1994) noted that recent peaks in residue levels in Haringvliet-oost, like those in the late 1970s, were characterised by relatively high concentrations of CBs 138 and 158, plus associated elevations in DDT and mercury. These CBs are particularly recalcitrant and the evidence points to emissions from old chemical dumps.

Concentrations of other organochlorine residues (e.g. HCB, DDT, DDE and DDD) also tended to be elevated at downstream sites in the Rhine and Meuse. Levels have tended to fall over the years of study

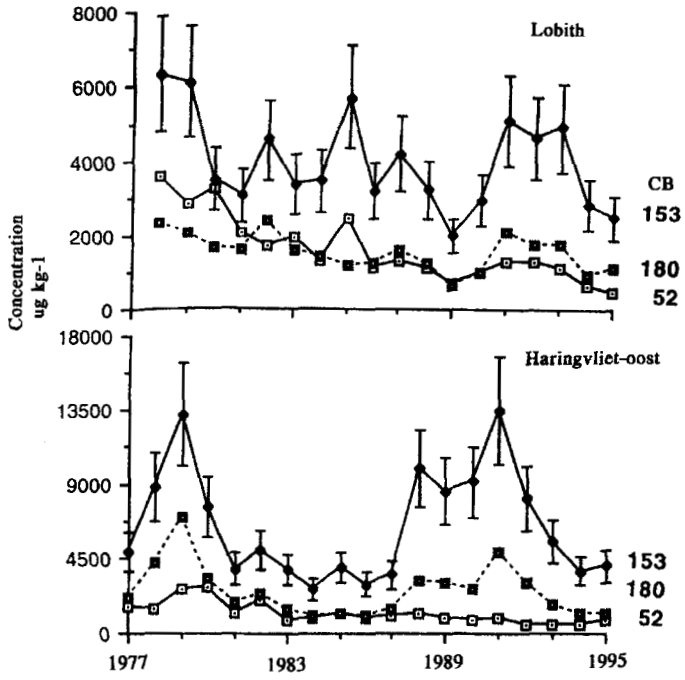

FIGURE 2 Changes in mean concentrations (μg kg^{-1} whole wet weight) of residues of PCB congeners 52, 153 and 180 between 1977–1995 in yellow eels from the River Rhine at Lobith and Haringvliet-oost. Adapted from de Boer *et al.* (1996).

and contamination is now sometimes more closely associated with localised sources. For example, the 1995 data in Figures 3a and 3b indicate that the distribution of dieldrin was related to sediment sinks in estuarine areas and inland agricultural areas. Conversely, HCB residues tended to be higher in industrialised and urban areas of the catchment. This correlates with its continued industrial uses and possible low-level emissions from sewage works (de Boer *et al.*, 1996).

The biomonitoring studies reviewed above emphasise key risks of organochlorine contamination. They have, however, yielded little in the way of robust quantitative data.

Uptake, Accumulation and Elimination

No quantitative studies have been conducted on eels to simultaneously compare concentrations of organochlorines in water, sediments, diet

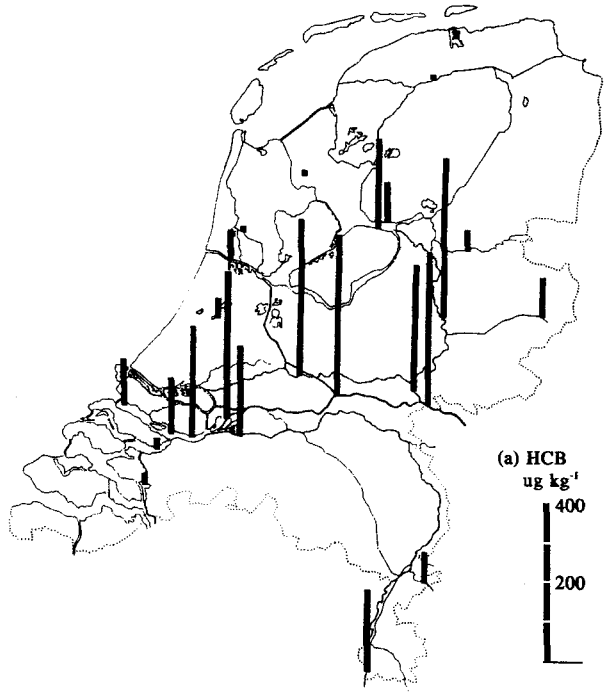

FIGURE 3 Maps showing the mean concentrations (g kg^{-1} whole wet weight) of (a) HCB and (b) dieldrin residues in yellow eels sampled from Dutch fresh waters in 1995. Sites as for Figure 1, see text for further explanation. Adapted from de Boer *et al.* (1996).

and the body. These are needed to clarify critical concentrations for risk management decision-making, as in setting environmental quality standards (EQSs). Direct uptake from water and sediments via the gills (and, possibly, the skin) might be expected to important in small eels (Larsson, 1984). De Boer and Brinkman (1994) reviewed studies on other fish species that show bioconcentration from water can occur quickly, especially for organochlorines with log $K_{ow} < 5$. Glass eels, elvers and small juveniles have relatively high surface area:volume ratios and metabolic and ventilatory rates. These characteristics could enhance uptake but should also aid depuration down concentration gradients when in clean water. Higher metabolic rates might also be associated with more efficient excretion. Work reviewed below suggests, however, that organochlorines are very persistent in eels, even in small ones.

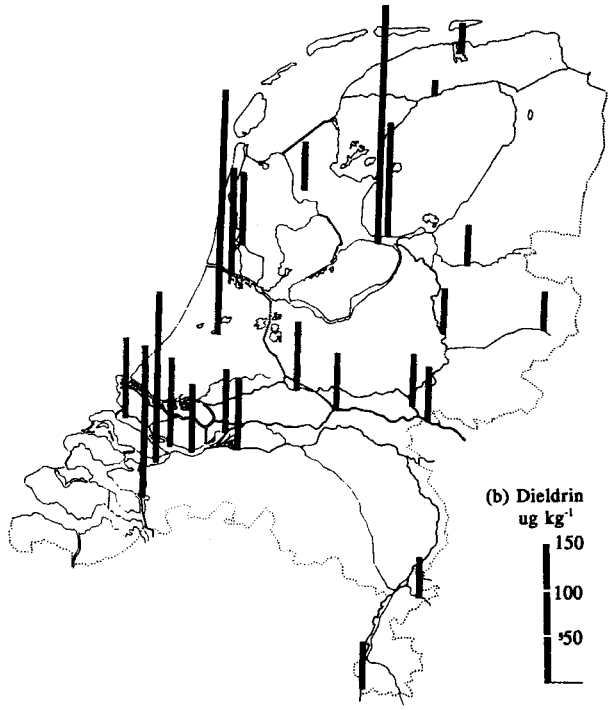

FIGURE 3 (Continued)

In contrast, risks from food-chain dietary transfer and biomagnification of organochlorines would be expected to be greater for highly hydrophobic compounds with log $K_{ow} > 6$. Eels of all sizes could be at risk because they commonly consume benthic macroinvertebrates living in contaminated sediments (Tesch, 1977; Deelder, 1984). Larger eels are commonly piscivorous and feeding at a higher trophic level could further enhance biomagnification of organochlorines later in life.

Bioconcentration factors (BCF, concentration in biota: concentration in water at equilibrium) have not been determined for eels. However, for a range of fish species occupying higher trophic levels, lipophilicity/hydrophobicity is a major determining factor, yielding the general relationship $\log \text{BCF} = 0.79 \log K_{ow} - 0.40$ ($n = 122$, $r^2 = 0.86$) (see Lipnick, 1995, and Spacie *et al.*, 1995). BCF values determined ranged from 3 to 14, but were as high as 30. Eels might be expected to show higher values because of their higher fat content.

More information is available for eels on biota:sediment accumulation factors (BSAFs), calculated on a normalised body lipid to total organic carbon (TOC) basis. Van der Oost *et al.*, (1996), for example, compared BSAFs for a range of organochlorines in six freshwater sites in Netherlands. Values were generally low for HCHs, drins and heptachlor pesticides and extremely low for dioxins and furans. Concentrations in fats of PCBs, DDTs, HCB, dioxins and furans tended to correlate with those in sediments, implying this was an important source of such organochlorines. BSAFs for PCBs, DDTs and HCB were, however, relatively high, suggesting that dietary magnification of these high K_{ow} organochlorines was contributing. Differences between individual PCB congeners tended to agree with those determined by de Boer and Brinkman (1994). They found log BSAFs varied between 1 and 5, organochlorines of higher K_{ow} showing the highest values (especially for the recalcitrant CBs 138 and 153 with log $K_{ow} > 7$). However, as discussed further below, it is possible that some lower CBs (and organochlorines such as HCH) can be metabolised to a limited extent.

Biological concentration factors for dietary transfer of organochlorines of high K_{ow} are commonly in the order of tens of thousands for fish at higher trophic levels (NRA, 1995b). No specific studies on dietary transfer via natural prey have been carried out on eels, but de Boer and Pieters (1991) used commercial formulated diets under intensive aquaculture conditions. Fish meal and lipids are important constituents of such feeds, but levels of organochlorine pesticides (DDT, HCH and dieldrin) and selected CBs (and mercury) were found to be low. Over a period of 1–1.5 years (from glass eel to eels of 60–80g), assimilation of both organochlorines and mercury was estimated (taking into account growth dilution) to be 50–70%. Assimilation efficiencies at high feeding rates under aquaculture conditions might be expected to be reduced because of rapid gastro-intestinal transit times. However, this would be offset by the high digestibility of commercial diets (especially of unsaturated lipids) compared to natural prey items (Knights, 1985). Other fish species show comparable organochlorine assimilation efficiencies (Spacie *et al.*, 1995). The authors suggest that any lack of accumulation was due to poor absorption during passage through the gut.

No significant excretion was claimed to have occurred over the duration of these experiments. The persistence of organochlorine contaminants in eels is well illustrated by long-term experiments conducted

by de Boer *et al.* (1994). Approximately 2700 tagged eels (30–40 cm) were taken from polluted areas of the Rhine (Hollands Diep, Figure 1) in 1981 and transferred to a relatively isolated Dutch lake. The concentrations of most organochlorines, relative to whole body wet weight, decreased over eight years of study. However, transferred eels were found to grow by an average of 23 ± 10 g (3 cm) per year, causing an annual growth dilution of 25–50%. Taking this into account, whole body burdens of residues decreased much more slowly (or not at all). There was, however, little difference in initial mean burdens of dieldrin in eels from the Rhine and those already present in the lake in 1981. Furthermore, levels (as $\mu g\ eel^{-1}$) changed little over eight years, suggesting some local sources and/or aerial deposition. Similar sources of contamination of the lake by gamma-HCH were also implicated, indigenous eels containing higher average concentrations than Rhine eels. Average body burdens of HCH trebled over the course of the study. Elimination half-lives (corrected for growth dilution) were estimated to range from about 480 days for HCB to 340–1450 days for tetra- and penta-CBs. No measurable elimination occurred to most hexa- and all of the hepta- and octa-CBs analysed. Such persistencies correlate with known recalcitrance and K_{ow} characteristics. Although not discussed in the paper, tabulated data show mean lengths of eels increased to over 40–50 cm after 7–8 years, i.e. many had attained the size at which males had matured and emigrated. Remaining males and females would be expected to emigrate within 12–14 years. Thus whilst some elimination of HCB and lower chlorinated CBs accumulated early in life might occur during the normal freshwater stage prior to spawning migration, this is not the case for the higher CBs. Metabolism of some CBs, such as CB 77 and 126, did appear to occur, as has been found in some other fish (e.g. in cod, Falandysz *et al.*, 1994) and marine mammals. De Boer and Hagel (1994) also suggested that the structural characteristics of CB 149 may render it more amenable to biodegradation. This, incidentally, would explain the relatively low concentrations of this CB found in eels from the Rhine and Meuse compared to sediment concentrations.

Other studies discussed by de Boer *et al.* (1994), de Boer and Brinkman (1994) and Spacie *et al.* (1995) suggest that elimination rates for HCH and CBs can be much faster in other species of fish. Some (e.g. guppies and goldfish) even show elimination of highly hydrophobic

compounds of log $K_{ow} > 6$. Inter-specific differences may exist but methodologies have varied between studies. Many have involved laboratory experiments on small fish, using different dosing methods (via water, food or by injection) and relatively short-time scales (20–350 days) for elimination. Thus intervals between uptake and residue analyses may well have been too short to allow equilibration within tissues. Small fish with relatively high surface area : volume ratios and metabolic and ventilatory rates (but low fat contents) would also be expected to take up and eliminate contaminants relatively more rapidly. Other authors have also not corrected for growth dilution in longer term experiments and in studies of natural populations. Losses during spawning also need to be taken into account. Annual spawners can lose up to 15% of body weight, and eels are at greater risk because they reproduce only once at the end of their life. The possible implications for eel reproduction and recruitment are discussed further below.

The study of Larsson *et al.* (1991) on eels in a Scandinavian lake yielded useful information on risks of bioaccumulation in body fat. DDE (at 0.02–1.0 and < 0.01 to 0.36 mg kg^{-1} in fat and whole body, respectively) comprised 22% of total organochlorine residues, compared to DDD at 6% and DDT at 1%. The range for total PCBs was 0.21–6.58 mg kg^{-1} fat (0.02–1.17 mg kg^{-1} whole body), the dominant congeners being the hexa-CBs 153 (11% of total organochlorines), 138 (11%) and 180 (7%). All residue concentrations were correlated with one another and with body weight, length and age up to about 11–12 years. Growth rates decreased with age (from 0.8 to < 0.2 cm $year^{-1}$ at age 12) and PCB and DDE residue concentrations were inversely correlated with growth rate. Thus rates of accumulation were partly off-set by growth dilution. Subsequent increases in residue concentrations after 12 years could then be explained by slowing of growth and hence of growth dilution effects. The authors point out that dietary biotransfer and biomagnification were also probably important. Older eels commonly switch from eating benthic macroinvertebrates to fish which have higher fat contents and hence residue burdens.

Larsson *et al.* (1991) showed that the body fat content of eels increased linearly (from 5 to 28%) up to about 400 g, 55 cm, age 11 years. PCB and DDE residue concentrations increased gradually with fat content but increased more quickly after 9 years of age. Larger eels would have been females and probably approaching maximum levels

of fat reserve accumulation prior to maturation and spawning migration. Thus rates of effective organochlorine accumulation in fat would have been diluted by enhancement of fat reserves in earlier life, up to the time of approaching maturation at $>9-11$ years. Larsson *et al.* (1990) have argued that attaining a critical fat content of over 20–25% is the major trigger for spawning migration. This would be adaptive in ensuring sufficient energy reserves for migration and reproduction, independent of the effects of temperature and productivity of waters on growth rates.

The importance of the findings of Larsson, de Boer and coworkers is that they confirm that eels are at risk of life-long accumulation of organochlorines of high K_{ow}, and that rates of depuration are generally low. Studies cited also confirm the suitability of eels as biomonitors of low levels of chronic pollution due to aerial deposition or biotransfer. Furthermore, there is no loss of organochlorines due to reproduction (which can exceed 15% of total body weight in other fish species).

With regard to the distribution of absorbed organochlorines between body compartments, the only detailed study published is that of Hodson *et al.* (1994). Concentrations of various CBs and total DDT varied between tissues of emigrant female eels in the St. Lawrence River but differences were not significant. Average concentrations were, however, higher in the ovaries than in the carcass, viscera or head, correlating with the higher lipid content (about 40% of ovary and 30% of carcass wet weight). The total burden in muscle must, however, have been higher because it comprised 83% of the whole body weight, compared to 5% for the gonads. Mobilisation of fat reserves from muscles during over-wintering quiescence or during periods of low food availability might be expected to occur. However, Boetius and Boetius (1985) have demonstrated preferential utilisation of muscle tissue, liver and visceral glycogen and fats. Ovary organochlorines residues, according to the Hodson studies, could have been reaching toxic levels. Concentrations of PCBs and DDT were as high as 8.8 and 2.8 $mg\,kg^{-1}$ respectively. There are no data for effects on eels but such levels can affect the reproductive success of salmonids (Evans *et al.*, 1990; Mac and Edsall, 1991). Giesy *et al.* (1986) demonstrated increased egg and larval mortality in chinook salmon (*Oncorhynchus tshawytscha*) from Lake Michigan at concentrations in eggs of about 1–10 $mg\,kg^{-1}$.

A critical TEQ of 65 $\mu g\,kg^{-1}$ for PCBs is sometimes quoted for chronic reproductive effects, based on these data (Hodson *et al.*, 1994). Risks for eels might be exacerbated by mobilisation of muscle lipid stores during migration. Critical loads could well be exceeded as lipids are mobilised and transferred to eggs as maturation is completed prior to spawning. Transfer of critical loads to larvae could then follow.

RISK EVALUATION

Acute and Chronic Toxicity

To fully evaluate risks, data are needed on acute and chronic impacts in relation to actual environmental and dietary hazard levels. Few LC50s have been measured for eels and some tests have not been well-controlled. Brusle (1991) cites values for yellow eels ranging from as high as a 24 h LC50 of 350 $\mu g\,l^{-1}$ (for lindane in freshwater aquaria) to as low as a 96 h LC50 of 0.6 $\mu g\,l^{-1}$ (for endrin in water of 24 ppt salinity). From data for other fish species, 96 h LC50s would be expected to be in the range of 1–100 $\mu g\,l^{-1}$ (NRA, 1995b). Such ambient concentrations would only be attained in large-scale spill situations. Indeed, it is only in more recent years that methods have been developed that can accurately determine levels in the lower $ng\,l^{-1}$ range.

As discussed earlier, high levels of organochlorine contamination were suggested by some authors to have caused the eel kills in the St. Lawrence River in the 1960–70s. Organochlorines were claimed to have affected gill membranes and hence gas exchange, with enhanced mucus secretion, desquamation and lamellar fusion (Dutil *et al.*, 1987). Such pathologies are observed in response to other stressors, such as heavy metals, because they help to reduce rates of entry across the gill membranes and into the blood stream. Dutil *et al.* (1987) also noted disturbances in ionic and osmoregulatory functions of the gills of moribund eels in the St. Lawrence River. Hypertrophy and hyperplasia of chloride cells led to sodium and chloride ion loss and a reduction in serum osmolality. DDT can also affect intestinal osmoregulation during adaptation to sea water (Janicki & Kinter, 1982). Skin congestion and haemorrhaging and changes in liver function have also been noted in eels exposed to organochlorines. Dutil and Lallier (1984) attempted to link bacterial infections, mortalities and

organochlorine burdens in St. Lawrence eels. However, as with other studies quoted above, cause-effect relationships could not be proven. Synergism between various natural environmental stresses (e.g. due to low DO and salt-water adaptation) and other pollutants (e.g. specific CBs and dioxins and furans) could not be ruled out.

Sublethal and chronic effects on reproduction and early eggs and larvae have been suggested as possible causes of falls in recruitment of *A. anguilla* to Europe in the last 10–15 years (Knights *et al.*, 1996; Moriarty, 1996). This possibility has also been advanced to explain similar falls in recruitment of *A. rostrata* in North America. Studies reviewed by Castonguay *et al.* (1994) did not reveal any histological abnormalities in the ovaries of emigrant silver eels from the St. Lawrence River but eggs and larvae could have been affected by lipids laid down in egg yolk. For example, the geometric mean DDT concentration in eel gonads was $0.37\,mg\,kg^{-1}$ and similar concentrations reduce the viability of eggs and larvae in salmonids, as discussed earlier. Castonguay *et al.* (1994) point out, however, that there was a mismatch between the timing of major pollution and declines in recruitment. Serious organochlorine problems occurred in Lake Ontario and the St. Lawrence River in the 1950s but emigrant mortalities did not occur until the early 1960s, peaking in the early 1970s. Any lack of breeding success/mortalities of eggs and larvae should have affected recruitment soon after, but major declines were not noted until the 1980s. Knights *et al.* (1996) also noted that glass eel catches of *A. anguilla* in Europe fell from a peak in the late 1970-early 1980s, again a long while after organochlorine pollution first became a major problem. Instead, these authors (and Castonguay *et al.*, 1994) suggest that changes in North Atlantic current systems could have affected transoceanic migration of leptocephalus larvae. Long-term time series data for glass eel catches in the Netherlands and, for the Japanese eel (*A. japonica*), in the Far East, also suggest large changes have occurred independent of anthropogenic factors (Knights *et al.*, 1996).

Biomarker studies

When discussing the hazards of organochlorine exposure, the possible value of metabolic responses as 'biomarkers' was mentioned. These could provide relatively cheap, rapid and sensitive screening tests for

critical contaminant levels. Eels have recently gained favour in laboratory and field biomarker studies but these have mainly focused on contaminants such as PAHs, heavy metals and organophosphate pesticides and induction of MFOs, conjugation reactions and effects on carbohydrate metabolism, e.g. Fenet *et al.* (1996) and Sancho *et al.* (1996). Others have concentrated on possible sensitive biomarkers for exposure to mutagenic and carcinogenic PAHs (van der Oost, 1994). Carbohydrate metabolism and haematological parameters have been shown to be affected by organochlorines (Ferrando *et al.*, 1991, Gill and Epple, 1993). However, the value of such relatively non-specific metabolic responses as biomarkers is debatable because they can be induced by so many other interacting stresses, both natural and anthropogenic. A more specific study was conducted by van der Oost *et al.* (1991) on eels from the moderately polluted Nieuwe Meer and less polluted Gaasperplas lakes near Amsterdam. In the former lake, hepatic MFO enzyme levels were raised, as were other liver enzymes associated with stress and these correlated with PCB and organochlorine tissue concentrations. It is difficult, however, to extrapolate from the responses seen to other mixtures of contamination in other ecosystems. Generally, biomarker data should be viewed very carefully and so-called critical contaminant concentration values obtained should not be accepted too readily for risk management decision-making. Clear evidence is needed first of the relationships between internal concentrations and whole organism and population effects, both in the short and long term.

RISK MANAGEMENT

The preceding discussions show that insufficient robust data are available for organochlorines to advise on specific water quality objectives and standards to protect eels. Instead, these have primarily been set for the protection of human health, as have critical levels of residues in eels that might be consumed by man. Hazards associated with organochlorines were first recognised in the late 1950s, with major inititiatives being spurred on by epidemiological evidence of biomagnification and potential health effects. Furthermore, because of their potentially widespread distribution, international initiatives had to be

taken. In Europe, for example, risk assessment approaches were used to classify organochlorines as List 1 (Black List) substances, requiring priority action, through the Dangerous Substances Directive (76/464/EEC) and subsequent daughter Directives and Regulations. Decisions were primarily made on the criteria of high acute aquatic toxicity (96 h LC50 $< 1\,mg\,l^{-1}$), mammalian toxicity (LD50 $< 50\,mg\,kg^{-1}$), persistence (half-life in water > 300 days), biological concentration factors (> 1000) and amounts produced (Agg & Zabel, 1990). Because of concerns about pollutant loads entering the North Sea and NE Atlantic by rivers and aerial routes, related voluntary risk management actions have also been proposed by bordering member states through the Paris Convention, overseen by the Paris Commission (PARCOM), and by the North Sea Conference Declarations (NSCDs) (NRA, 1995 a, b; Stanners & Bourdeau, 1995). The principal risk management approaches have been similar to those used in North America and other countries, i.e. controls on discharges in order to meet environmental quality standards, staged restrictions on use and eventual banning (e.g. in the UK, DDT was banned in 1984 and dieldrin in 1989) and, for PCBs, eventual regulated destruction of those in current use. Requirements have also been instituted to set environmental quality objectives (EQOs) and standards (EQSs) for surface waters (and groundwaters). In the UK, for example, EQS levels are typically equal to or less than $10\,ng\,l^{-1}$ (Table I), to allow a safety margin for high biomagnification risks. Monitoring studies are required, as are calculations and reporting of contaminant loads, i.e. the product of concentration and river or effluent flow to the sea (NRA, 1995a).

Biomonitoring has not been included in the above Directives or agreements, although this review has shown that eels could be very useful in such studies. Residue levels are indicative of mean concentrations in water and, especially, the risks associated with organochlorine sediment transport and sinks. Various countries have, however, instituted food residue monitoring programmes, which include eels where these are eaten in any quantity. Contamination levels are compared to recommended risk standards (as explained below). Residue levels found to exceed limits lead to restrictions on sales or advice on consumption levels. The benefits achieved by risk management approaches adopted can be illustrated by reviewing the results of temporal biomonitoring and of food surveillance programmes utilising eels.

(a) Evidence from Temporal Biomonitoring of Eels

There is no direct evidence for actual damage to eels or consumers of eels and, consequently, whether management actions have actually reduced risks. However, temporal biomonitoring has revealed reductions in environmental contamination. In North America, for example, Newsome and Andrews (1993a, b) found organochlorine residues had decreased in 11 species of common freshwater fish from the Great Lakes, following institution of pollution prevention measures in the 1970s. Contaminant levels fell less rapidly through the late 1980s, reflecting the long half-lives of persistent organochlorines (especially in sediments), plus diffuse emissions and aerial deposition. Eels, however, still had the highest residue levels of total PCBs ($0.75\,mg\,kg^{-1}$ wet weight compared to $0.63\,mg\,kg^{-1}$ in trout) and the second highest for other organochlorines ($0.061\,mg\,kg^{-1}$ compared to $1.40\,mg\,kg^{-1}$ in trout). The major PCB congeners (CBs) present were the more persistent higher (penta- and hexa-) chlorinated ones, DDE was the major component of other organochlorines. Other unidentified organochlorine compounds were also found. These could have represented metabolites of measured compounds or other organochlorines.

Analyses of emigrant eels caught in the St. Lawrence River have also shown that organochlorine residues fell between 1982–1990 (Hodson *et al.*, 1994). Patterns of decline were similar to those in other Great Lakes fish, but with a time lag of about 10 years. This would be expected because eels spend 8–16 years accumulating contaminants of high K_{ow} in fresh waters before emigrating down the St. Lawrence River. Concentrations of dioxins, furans, PAHs and most organochlorine pesticides were generally very low, many being below levels of detection. Other organochlorine residue levels were variable but means for comparable samples had decreased, e.g. by 68% for total PCBs (from 4.54 to $1.43\,mg\,kg^{-1}$) and by 56% for mirex (from 0.073 to $0.032\,mg\,kg^{-1}$). In 15.7% of eels, however, dieldrin still exceeded the Canadian Guidelines for Human Consumption of $0.1\,mg\,kg^{-1}$. This pesticide was still in restricted use in the 1980s, for control of insect pests of livestock. Residues of organochlorines with high K_{ow} and K_{oc} would still be expected in older eels, especially those from areas where sediments are still contaminated. Mirex was still found to

exceed Canadian consumption levels (Table I) in 29% of samples, compared to 52% in 1980.

In the Rhine delta, Hendriks and Peters (1993) showed similar downward trends had occurred, with tendencies to reach plateau levels later in the 1980s. This has been confirmed by the Dutch RIVO-DIO surveys, e.g. as shown for HCB and PCBs in Figures 3a and b. Dieldrin residue levels in 1995 (generally $< 0.15\,mg\,kg^{-1}$ wet weight, Figure 3b) have decreased > 10-fold since the 1980s. Gluck and Hahn (1995) also found reductions in eels from Lake Constance between 1982–3 and 1992–3. Residue levels were elevated downstream of the lake, however, indicating that low inputs were still being received as the Rhine flows through industrialised areas. Furthermore, some CB residues remained high, often exceeding statutory limits. For example, residue levels of CB 153 and 180 had actually tended to rise in the 1980s in Haringvliet-oost and at Lobith, where the Rhine enters the Netherlands, although they have fallen in the 1990s (Figure 2). The elevated Haringvliet levels may be due to disturbance of contaminated sediments or, as argued earlier, to discharges from waste dumps. The general trends in the Rhine and Meuse have been mirrored by those in eels from the River Elbe and its tributaries in Germany (Kruse *et al.*, 1994), as illustrated in Figure 4.

With regards to contamination of marine environments, capillary GC analyses of chlorinated pesticides (including aldrin, endrin, lindane, HCB, and DDT) and six PCB congeners were carried out on 54 samples of eels from 11 European sites by Karl and Lehmann (1993). Levels had decreased since the 1970–80s, although they were still higher (especially in Baltic Sea eels) than in samples of farmed eels. However, mean contamination levels were less than 1–5% of German statutory limits and no individual samples exceeded limits. The authors concluded that residual levels did not pose a health hazard and that levels were decreasing following banning or tighter controls on the use and disposal and hence riverine transport into north-west European seas. This is borne out by similar changes in contaminant levels found in livers of cod. De Boer and Brinkman (1994) point out, however, that no significant decreases in HCB or recalcitrant CB concentrations had occurred since the early 1980s, mirroring the picture for freshwater and marine eels. This is because, although uses of HCB as a fungicide were banned in the 1970s, diffuse emissions still

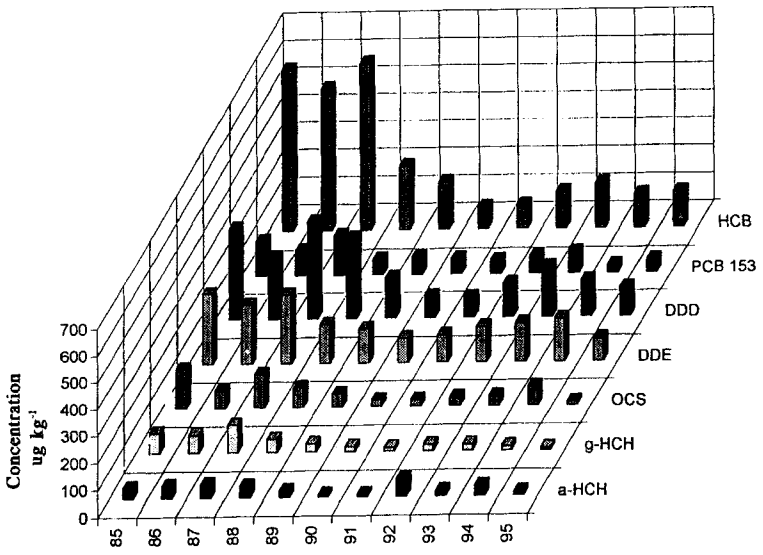

FIGURE 4 Changes in mean concentrations (g kg^{-1} whole wet weight) of residues of HCB, PCB congener 153, DDD, DDE, octachlostyrol (OCS), gamma-HCH and alpha-HCH between 1985 and 1995 in yellow eels from the River Elbe at Lauenburg. Adapted from Kruse *et al.* (1996).

occur from its uses (as with PCBs) in industrial applications. Releases of PCBs are still occurring and the large amounts still in use pose a threat for the future.

(b) Human Consumption Risk Assessment and Management

Absolute residue limits are difficult to derive because of the lack of direct evidence for critical levels in human food. Risks to man of consuming eels have not been clearly proven, although there is some indirect evidence. For example, it has been claimed that mothers who consumed fairly high quantities of PCB-contaminated fish from the Great Lakes tended to have children whose physical and behavioural development was retarded (Jacobsen & Jacobsen, 1993).

Generally, the approach to setting food residue limits has been to determine the effects of dose levels in laboratory test animals, such as mice and rats. A factor (typically of one thousandth of the no-observable effect concentration, NOEC) is applied to take account of possible

interspecific differences and as a safety margin. This is then extrapolated to predict the critical levels in food and/or daily food intakes. Critical values vary, however, between countries and in the ways in which they are expressed (Table I).

Particular problems arise in interpreting data for different PCB congeners. De Boer *et al.* (1993) measured concentrations of various PCBs, dioxins and furans in marine and freshwater fish and shellfish in the Netherlands. In all eels and eel liver samples (and shellfish), CB 126 made the major contribution to potentially toxic levels of contaminants and total PCB potential toxicities exceeded those of dioxins and furans. TEQs for yellow eels from the Rhine and Meuse (and cod livers from all parts of the North Sea) exceeded the Canadian dioxin tolerance limit of $20\,ng\,kg^{-1}$ (Table I).

In the studies of St. Lawrence River eels by Hodson *et al.* (1994), concentrations of CBs were highly variable between individual female emigrant eels and in tissues and some CBs could not be separated during analysis. The least concentrated CBs in carcasses were 28 and 137 ($< 0.01\,mg\,kg^{-1}$), the most concentrated were 118, 138 and 153 (range $0.052–3.033\,mg\,kg^{-1}$). By making assumptions about mixtures, the authors estimated the whole body TEQ to be $1885\,ng\,kg^{-1}$ using mammalian TEF values, $117\,mg\,kg^{-1}$ using values for fish. These exceeded the Canadian guidelines for human consumption of $20\,ng\,kg^{-1}$ and the threshold for chronic toxicity for fish of about $65\,ng\,kg^{-1}$ (based on the sensitivity of trout eggs and embryos, discussed earlier). The authors also suggested that such TEQ levels could pose a hazard for marine mammals consuming eels, such as the beluga whale (*Delphinapterus leucas*), as discussed below. De Boer *et al.* (1993) recommend that derivation of uniformly acceptable TEFs of PCBs are needed for estimating the potential hazards of different congeners to man.

Further complications arise in human risk assessment and management because different agencies are involved in setting standards, monitoring residues, interpreting data and advising consumers. One measure is the Acceptable Daily Intake (ADI), based upon toxicity data and assumptions about the quantities of contaminated food that might be consumed every day throughout an individual's life span. For dieldrin, for example, the ADI set by FAO/WHO is $0.1\,\mu g\,kg^{-1}$ of body weight per day, equivalent to meat residue limits of 0.2–100 μg

kg^{-1}. In the absence of firm toxicity data, the concept of Maximum Residue Level (MRL) is used by the EC. For a pesticide approved for use for a particular purpose, this is equivalent to the lowest residues achievable in foods whilst still achieving effective and reliable control of pests. The European Commission is attempting to harmonise maximum residue levels (MRLs).

Setting limits on food residues is obviously a good precautionary approach to risk management. However, a clear understanding of sampling, clean-up and analytical methods is needed. Similarly, an understanding of eel biology, fisheries and markets and human consumption factors are needed to reach objective risk management decisions. As an example, in the United Kingdom the Working Party on Pesticide Residues (WPPR), which coordinates monitoring of organochlorines in foods, has issued warnings in the past about high contaminant levels in eels from certain waters. In 1986, consumers in the London area were warned that some eels from the Thames contained high levels of dieldrin (0.4–0.6 mg kg^{-1} wet muscle). As this could exceed the FAO/WHO ADI (Table I), consumers were warned not to eat more than 35g of eel per week. The author (B. Knights) criticised this announcement because the eel sample sizes were very small and included large females from closed waters where no commercial fishing was allowed. Furthermore, no account had been taken of losses during cooking, nor the fact that many of the eels consumed in London were imported from Ireland and Australasia. Later studies by the WPPR have shown that dieldrin levels are lower in eels caught by commercial fishermen in the Thames and in imports and that there is a loss of residues on cooking (MAFF/HSE, 1996).

This example demonstrates the need for careful and standardised sampling and monitoring. It also strikes a cautionary note in that much publicity is given to contaminated eels from polluted waters but that the majority caught commercially for human consumption come from 'clean' waters. This is borne out by other European studies, e.g. that of Karl and Lehmann (1993) mentioned earlier. Incidentally, monitoring of residues in eels as part of food surveillance programmes has also been useful in further demonstrating the improvements achieved since rigorous controls on organochlorines came into force (e.g. see DoE, 1996; MAFF/HSE, 1996). However, the continuing

presence of organochlorines such as dieldrin emphasises their persistence in the natural environment and in eels.

(c) Risk Assessment and Management for Animals Consuming Eels

Concerns have been expressed that some predators have been affected by consuming contaminated eels. For example, relatively high levels of PCBs and organochlorine pesticides (including mirex) have been found in the blubber of beluga whales (*Delphinapterus leucas*) off the mouth of the St. Lawrence River. These were associated with an abnormally high incidence of tumours, lesions and immunosupression problems. It has been suggested that contaminated eels originating from the Great Lakes were important items in the whale's diet, leading to biotransfer and biomagnification (Hodson *et al.*, 1994). Fish-eating birds have also possibly been similarly affected, leading to deaths and deformities of embryos and chicks (Giesy *et al.*, 1994). Cormorants (*Palacrocorax carbo sinensis*) living near the polluted Lake Ketelmeer in the Netherlands, for example, were found to have high levels of organochlorines (and heavy metals) in their livers. Concentrations correlated with age and apparent dietary preferences for different fish species. It was suggested by Platteeuw *et al.* (1995) that residues were related to the relative proportions of eels in the diet.

Otters would also be expected to face high risk from consuming contaminated eels. This might have contributed to past declines in populations and the distribution of European otters, *Lutra lutra* (Mason & Macdonald, 1993: Mason, 1995 a, b). Reductions in pesticide and PCB pollution may in turn be a factor in their current recovery – but the success of introductions and natural recolonisation in the future may be limited by continuing PCB contamination of eels. Cause-effect relationships have not been established directly. However, Mason (1995 a, b) quotes laboratory experiments that showed reproductive success in mink (*Mustela vison*) was reduced when they were fed fish spiked with certain levels of PCBs (especially potent being higher chlorinated congeners). From these data, assuming an average daily consumption of 0.5 kg of eels out of a total fish intake of 1 kg, the recommended critical concentration level for PCBs in eels can be extrapolated as $0.05\,mg\,kg^{-1}$. Dutch recommendations are

more conservative, at 0.025 mg kg^{-1}. There are, however, conflicting views on this. Mink could be more susceptible than otters. Furthermore, Kruuk and Conroy (1996) argue that field studies do not show patterns of impaired reproduction and reduced population densities and distributions that are clearly related to known or suspected patterns of PCB contamination in surface waters. They suggest other factors may be more significant, especially habitat loss or degradation, generally poor water quality and human disturbance. It appears, therefore, that whilst Mason's recommendations may be a useful precautionary guide, other factors need to be taken into account. It will be interesting to see whether otters and other consumers of eels show further recoveries as organochlorine contamination declines in the future. However, the potential risks of continued pollution by PCBs are possibly going to increase rather than reduce, as discussed further below.

CONCLUSIONS AND RECOMMENDATIONS

This review has emphasised the chronic hazards posed to eels by the persistence of organochlorines and by sediment transport and sinks and aerial transport. Risks of bioaccumulation to critical levels (and also of transfer of residues to consumers) are exacerbated by the high fat content of eels. Other key risk factors are their ubiquitous distribution, benthic habits, dietary preferences for benthic macroinvertebrates and/or fish and the fact that they only breed once.

Risk management strategies aimed at general environmental protection have involved the setting of EQOs and environmental loading limits, leading to derivation of EQSs and controls on emissions. Restrictions on uses, production and eventual bans (and plans for controlled destruction of PCBs) have had to be instituted because of the high hazards. In relation to potential risks to human consumers, critical residue limits in eels have been recommended. Each of these risk management approaches has important limitations.

Setting of standards which involve measurements of concentrations in water are limited by the low solubility of hydrophobic organochlorines. Concentrations in many samples fall below detection limits. Average levels cannot therefore be calculated and results can only be

expressed as percentages of sites failing EQSs. Sampling is also only carried out periodically and at a limited number of sites. For example, the European Commission (EC) EQS for dieldrin is set at an annual average of $10\,ng\,l^{-1}$. Surveys in England and Wales by the National River Authority found that organochlorine concentrations in the majority of samples fell below detection limits. Between 1975–79, 15% of sites failed their EQS, but none failed between 1990–4 (NRA, 1995b), implying falling contamination. The earlier results were, however, biased by localised high levels, for example in the River Aire in Yorkshire due to contamination from wool processing. Studies quoted earlier show that residues are still present in eels (and in sediments), emphasising that biomonitoring would be a useful supplement to water quality surveys.

Similar criticisms can be levelled at estimations of organochlorine loads based on water quality data. PARCOM and North Sea Declarations require 50% reductions by 1995 of total riverine and coastal inputs of priority pollutants to north-west European seas, relative to 1985 levels. Results, such as those obtained by the NRA (1995 a), show targets are being achieved. Lindane loads are still relatively high, however, reflecting their continued industrial production and emissions, especially via sewage works effluents. However, organochlorine levels in many samples are below detection limits and reliable base-line data for the 1980s are rarely available. For example, lindane loadings to the North Sea from England could have been as high as 0.4 tonnes in 1985, falling to 0.27 tonnes in 1993, but these calculations only include samples where lindane was found in measurable concentrations (NRA, 1995 a). Again, biomonitoring of residues in eels would provide useful additional information.

Biomonitoring should also be applied on a wider scale. For example, agricultural and industrial activity has been intensifying in eastern and Mediterranean countries in Europe and in the Far East. Thus problems from past, present and future uses of organochlorines will arise (Stanners & Bourdeau, 1995). Even in the more prosperous and regulated EU countries and in North America, PCBs still in use and in waste tips threaten to be a problem for years to come. Since the late 1950s, over 1.2 million tonnes of PCBs have been produced world-wide. Over 30% has since been released into the environment through accidental spillages and disposal to landfills, leaving a large amount still in use. For

example, 8000 tonnes are still in use in the UK in over 800 transformers and 45,000 large capacitors. The manufacture of PCBs ceased in the EU in 1978 and new uses have been progressively banned since 1980. In 1996, an EC Directive was agreed for the controlled collection and destruction of remaining sources (and any hazardous substitutes) by 1999. Conflicts are likely to arise, however, with PARCOM proposals for destruction by the year 2000. For example, the Directive differs from PARCOM in allowing derogations for PCBs in small transformers. Even small pollution incidents can pose severe problems, as those arising from the disposal of just 1200 litres of PCBs into the Vanajavesi catchment in Finland (Tulonen & Vuorinen, 1996). Problems will also arise, as discussed earlier, from sinks such as contaminated sediments and waste tips. These will not be easily detected by water quality studies. Biomonitoring of residues in eels would be a useful additional tool for risk evaluation and assessment.

Biomonitoring and food residue studies have sometimes produced confusing results in the past and led to poor decision-making. To ensure reliability and comparability, the following recommendations are made:- (a) sampling should be undertaken at the same site every year in the spring, restricting analyses to pre-spawning eels of 30–40 cm. This will help avoid confusions due to sampling of older emigrant eels of unknown geographical origins. Ideally, stock origins and ages should be determined (see Knights *et al.*, 1996); (b) organochlorine analyses should follow carefully standardised methods, such as those used by de Boer and Hagel (1994). Standard reference samples should be analysed and inter-laboratory comparisons made to validate methods (see Kruse *et al.*, 1996); (c) to provide information on levels of environmental contamination and also possible hazards to animals and man, residue levels should be expressed in concentrations relative to whole body, edible muscle portion and lipid fresh wet weights. Any effects of processing or cooking (e.g. of jellied eels) prior to human consumption should also be taken into account; (d) uses of residue data in risk assessments and management should take into account the high variability between eel samples. For example, the exhaustive studies of de Boer and co-workers typically reveal 95% confidence limits to be between 25 and 50%.

Although this review has shown eels are useful in biomonitoring and biomarker studies, further work is needed to answer some key

questions. More information is needed on cause-effect relationships between residue levels and chronic effects to clairfy risks to man and animals consuming eels. Similarly, more quantitative data and long-term studies are needed of uptake, elimination, bioaccumulation and chronic effects on eels themselves. Risk assessments for different life-stages are required. In view of possible relationships between organochlorines and stock-wide recruitment failures, there is a particularly pressing need for studies of impacts on reproductive systems in wild silver eels and/or in long-term controlled dosing experiments.

ABBREVIATIONS

ADI	Acceptable Daily Intake, maximum daily routine consumption recommended by WHO
BCF	Biological Concentration Factor, ratio of concentration of a contaminant in biota relative to the concentration in water at equilibrium
BSAF	Biota: Sediment Accumulation Factors, ratio of concentration of a contaminant in biota relative to the concentration in sediments, calculated on a normalised body lipid to total organic carbon basis
CB	An individual congener form of PCBs, numbered according to the standard IUPAC system
DDT	p, p-DDT, dichlorodiphenyltrichloroethane. Total DDT includes DDT and its derivatives, DDD and DDE
DO	Dissolved level of oxygen in free water bodies
EIFAC	European Inland Fisheries Commission
FAO	Food and Agricultural Organisation of the United Nations
EQO	Environmental Quality Objective
EQS	Environmental Quality Standard
H	Henry's law constant
HCH	gamma-hexachlorohexane (commercial preparation is lindane). Total HCH includes alpha, beta and gamma forms
HCB	Hexachlorobenzene
K_{oc}	*n*-octanol:carbon partition coefficient

K_{ow}	*n*-octanol:water partition coefficient
MAFF	Ministry of Agriculture Fisheries and Food (UK)
MFO	Mixed-function microsomal monoxygenase enzyme.
MRL	Maximum Residue Levels based on FAO/WHO recommendations for human diet
NRA	National River Authority (UK), now the Environment Agency
PARCOM	Paris Commission
PCB	Polychlorinated biphenyl
PBB	Polybrominated biphenyl
QSAR	Quantitative Structure-Activity Relationship
TCDD	2,3,7,8-tetrachlorodibenzo [*p*] dioxin
TEF	Toxic Equivalency Factor, the ratio of the relative toxicity of TCDD to a PCB congener (measured via the ability to induce hepatic MFOs)
TEQ	TCDD Toxic Equivalent for a PCB congener, derived by multiplying the TEF by the concentration found in tissues. Total PCB TEQ can then be found by summing the values for all (or the most potent) congeners. present
WPPR	Working Partly on Pesticide Residues, UK interdepartmental body which monitors pesticide residues in food

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