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Chapter 17

Persistent Organic Pollutants: Occurrence and Health Risks in Australia

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Abstract

The major persistent organic pollutants (POPs) in Australia are the persistent organochlorine pesticides (OCPs). The adverse effects of DDT were first recognised in Australia in the late 1960s. Subsequently, the Australian Government introduced a policy of phasing out the registered uses of this major pesticide and related substances so that their use in agriculture ceased by 1987.

Australian foods are monitored on the basis of the National Residue Surveys and Australian Market Basket Surveys (now Total Dietary Survey). Only a low percentage of OCP residues have been detected in animal fats since 1996 and none in grains since 1992. Elevated intakes of residues (e.g. dieldrin and HCB) occurred in the early 1970s, which have since declined at approximately first-order kinetics to much lower and generally acceptable levels of intake in the 1990s.

Total intakes of OCPs from the Australian diet and environment have been estimated from the 1970s to the 1990s and used to evaluate human health risks for the Australian population. The Hazard Index (HI) was estimated for the general Australian population for the OCPs for 1996 at <1.0 in all cases. In addition, the USA-EPA model for occurrence of cancer has been applied to some of the persistent OCPs and found to be in the range of $>1\times10^{-6}$ to 4×10^{-5}. The level of health risk for the general Australian population was considered to be acceptable in 1996.

Some scenarios for high-risk groups in the Australian population were examined. These indicated that dieldrin intake from contaminated fish, contaminated soils, or in indoor air following termiticide treatment was a potential risk for exposed persons. Also the exposure of breast-fed infants to DDT during 1996 with a HI of 0.9 was questionable. However, extrapolation to 2005 using first-order

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kinetics indicated that the HI for all OCPs was below 0.1 and thus not a hazard to health of the Australian population.

The National Dioxin Program has extensively surveyed the levels of dioxins in Australian foods, human milk and the natural environment as well as identifying sources. As a result of this the risks to human health and the natural environment are considered to be minimal. Human health risks from exposure to POPs may continue to be an issue, because of increasing concerns about endocrine disruption and also the possible relationship between residues and specific cancers such as breast cancer.

17.1. Introduction

During the 1960s and 1970s, semivolatile and persistent chlorohydrocarbon pesticides, such as DDT, HCHs, dieldrin and chlordane, and industrial chlorohydrocarbons such as the PCBs and hexachlorobenzene (HCB), were detected throughout environmental compartments and were found to be bioaccumulative in lipid-containing tissues (Woodwell et al., 1971). These lipophilic substances, having environmental persistence and occurring widely in ecosystems, have been generally described as persistent organic pollutants (POPs). In addition, combustion products have been detected consisting principally of the polychlorodibenzodioxins (PCDDs) and the polychlorodibenzofurans (PCDFs), which share the persistent, lipophilic and bioaccumulative properties of the chlorohydrocarbons (Rappe et al., 1978). Thus, currently the following substances are usually considered to be globally important POPs: aldrin, chlordane, DDT, dieldrin, endrin, heptachlor, HCB, hexachlorocyclohexane (HCH including lindane), mirex, toxaphene, polychlorobiphenyls (PCBs), PCDDs, and PCDFs. The first nine substances in this list (from chlordane to toxaphene) are commonly referred to as the organochlorine pesticides, the OCPs. In this paper the term OCPs refers to the chlorohydrocarbon group alone and does not include such substances as 2,4,5-T and 2,4-D, which are not considered to be persistent in the environment.

In Australia several landmark reports were published regarding adverse affects of OCPs including major reviews by the Australian Academy of Science (1972) and the Victorian Government in 1966 (Victorian Pesticides Committee, 1966), and papers by Butcher (1965) and Bacher (1968). These publications have had a major influence in that they initiated a large monitoring program in Australia. As a result there have been many scientific and governmental enquiries into the usage of DDT and other chlorohydrocarbons. It is now clear that the use of DDT has caused a range of problems including direct lethal effects and sublethal

effects such as eggshell thinning in certain species of birds (Olsen & Olsen, 1979).

The OCPs have been the major POPs in the Australian environment and were introduced to Australia in the mid-1950s. The major uses of OCPs were crop and livestock protection from insects and parasites. Meaningful data on the actual amounts of OCPs that were used in Australia are not readily available. A steady increase may be assumed from Australian pesticide sales, which increased from a value of 15 million dollars in 1975 to 130 million dollars by 1990 (Short, 1994). Herbicides, however, account for the largest proportion of pesticides sold in Australia (Rayment & Simpson, 1993). The use of OCPs in Australia spans approximately 40 years from the 1950s to 1990s. By 1975, there were many pesticide products containing DDT, HCH and dieldrin, a lesser number for lindane and chlordane, and some for aldrin. During this period, one or more of these pesticides was registered for use against pests in a broad range of crops. OCPs were also used for termite control and around livestock quarters.

These chemicals were most widely used during the mid-1970s but were largely phased out by 1990. The first restrictions occurred in 1961/1962 when OCPs were deregistered for use on food producing animals. Monitoring of residues in table ready foodstuffs was introduced in 1970. During the early 1970s, the Standing Committee on Agriculture introduced a policy of deregistering persistent OCPs in all agricultural uses as soon as alternatives became available.

Significant reductions in registered OCPs occurred during the late 1970s due to scrutiny by registration authorities and pressure from technical committees on agricultural chemicals including the National Occupational Health and Safety Committee, Australian Agricultural Council, National Health and Medical Research Council (NHMRC) and the Australian Environment Council. By the end of 1985, most pest/crop/chemical combinations in agricultural applications were deregistered. During 1987, the States and Territories commenced recall programs of OCP stocks and import of OCPs into Australia was prohibited. However mirex and lindane were registered for strictly restricted usage. In 2002, Connell et al. (2002) published a report on the environmental occurrence of the OCPs in which they report that the levels of these substances had fallen to low to negligible levels in the human diet, sewage inputs to the environment and in the environment in general.

In Australia there was little coordinated information on the dioxins and furans up until 2001. In that year a National Dioxins Program was set up by the Australian Department of the Environment and Heritage which carried out an extensive program of investigations of sources, air,

soils, aquatic fauna, human foods, blood and milk. The findings were released in 2004 and indicated that the levels of risk to human health and the natural environment were low (National Dioxins Program, 2004).

17.2. Sources of POPs in Australia

DDT had been imported or manufactured in Australia since 1966, but in early 1971, manufacture within the country ceased and all DDT since then was imported. The early use of DDT in Australia was summarised by the Australian Academy of Science (1972) in *The Use of DDT in Australia*. At that time it was estimated that about 900 tonnes of DDT were used annually. Total DDT annual imports or production in Australia peaked in 1973 at 3625 tonnes (Olsen et al., 1993). The cumulative total DDT usage in Australia up to 1993 was estimated to be 10,000 tonnes (Connell, 1981; Connell et al., 1999).

There are no clear estimates of the quantities of cyclodiene insecticides used in Australia. Investigations by Greenpeace indicated that Australia imported 272 tonnes of heptachlor and 95 kg of chlordane during 1987–1989 (Short, 1994). Estimates for aldrin indicate 364 tonnes were imported between 1989 and 1991. The lindane and the HCH group of perisistent-chlorinated pesticides have been extensively used in the Australian environment but data are not available on the quantities involved. However, the total lindane usage in Australia up to the end of 1993 was estimated to be 900 tonnes (Connell et al., 1999). HCB had limited manufacture as a fungicide the 1960s (Connell et al., 1996). However, its major sources are as a contaminant in a range of chlorinated compounds including pesticides and solvents, and as a waste product in a wide variety of chemical manufacturing processes.

Dioxins (PCDDs) occur as contaminants in many agricultural pesticides and can occur in the environment as a result of pesticide usage, although many other industrial sources and natural sources have been identified. The National Dioxins Program (2004) has revealed that major sources of dioxins in the environment are uncontrolled combustion sources such as bush fires and accidental fires which contribute 70% of the total to the air and 80% to the soil whereas waste disposal and land filling contribute 75% of the total to water.

17.3. Physicochemical and environmental properties

These substances share a set of common physicochemical properties that can lead to undesirable effects on living systems. There is a limited range

of bond types present in the POPs group. These are the C=C (aromatic) C=C, C–H, C–Cl with lesser numbers of C–C, C–O which all have zero or very low polarity. This means the compounds in this group tend to have low polarity and dipole moments. These properties result in compounds in this group being *fat soluble* or *lipophilic* and having a low solubility in water. While the solubility in lipid of these compounds lies in the order of grams per litre, the aqueous solubility ranges from 4.7 mg L^{-1} for lindane to 0.0032 mg L^{-1} for *p,p*[1]-DDT and lower for some PCDDs. The lipophilicity of these compounds is indicated by the octanol/water partition coefficient (K_{OW}) which lies between 4100 (log K_{OW} 3.61) for lindane and 3,200,000 (log K_{OW} 6.5) for aldrin and higher for some compounds. Lindane is the lowest molecular weight compound in this group and tends to fall at the extreme end of the properties of the group with properties such as the highest water solubility and lowest octanol/water partition coefficient.

The limited ranges of bond types present in the POPs are generally relatively resistant to attack by abiotic or biotic agents in the environment. As a result environmental degradation proceeds at a relatively slow rate. Most compounds in this group persist for long periods in the environment and often exhibit half-lives of many years as illustrated by the data in Table 17.1.

Bioconcentration in aquatic organisms occurs as a result of partitioning between the organism lipid and the surrounding water. If a substance is *lipophilic*, then equilibrium occurs with a relatively high *Bioconcentration Factor* (K_B), or the organism/water partition coefficient. For example, the K_B value of *p,p*[1]-DDT is 79,400 (log K_B 4.9), heptachlor, 7900 (log K_B 3.9)

Table 17.1. Properties of some persistent organic pollutants

Substance	Log K_{OW}	$T_{1/2}$ (days) (freshwater)	Log K_{OC} adsorption sediments	Aqueous solubility (mg L^{-1})	Log K_B
p,p′-DDT (4,4)	6.2	7–350	4.8 (est)	0.0032	4.9 (est)
Total chlordane	5.54	<1400	2.4–4.2	0.08	4.22 (est)
Dieldrin	4.32	175–1100	3.87–4.08	0.17	4.1
Heptachlor	5.27	0.9–5.4	4.48	0.18	3.8–3.9
γ-BHC (lindane)	3.61	22–692	3.34 (est)	4.7	2.3 (est)
Hexachloro-benzene (HCB)	5.86	31–41	NA	0.006	4.1
Dioxins and furans	3.7–8.2	~720	NA	$4.4 \times 10{-}2$ to $1.3 \times 10{-}9$	2–6

Sources: Chemical Evaluation Search and Retrieval System (1995); Howard (1991); Howard (1991).

dieldrin, 13,600 (log K_B 4.1), lindane 200 (log K_B 3.3). The K_{OW} value and lipophilicity of various compounds are outlined in Table 17.1. Mammals have the capacity to induce oxidative enzyme systems particularly the Mixed Function Oxidase system including Cytochrome P-450, which reduces the bioaccumulation of the OCPs. Thus the concentrations observed in mammals is relatively low. The cyclodiene subgroup, e.g. dieldrin and aldrin, tend to have higher mammalian toxicity than other members of the chlorohydrocarbon family.

17.4. Occurrence of POPs in human foods

In the early 1960s, the Commonwealth (Australian) Department of Primary Industry began testing agricultural products, such as meat, for organochlorines to meet international trade requirements. This program has continued to the present as the National Residue Survey (NRS) now controlled by the Bureau of Rural Resources.

In the case of domestic foodstuffs, State Health and Primary Industry authorities have followed up with testing of selected food groups and the NHMRC initiated market basket surveys of residues in foods based on the normal Australian diet. Since 1990, these surveys have been managed by the Australian New Zealand Food Authority.

The NRS results show that Australian produce has relatively few samples that contain chemical residues above acceptable limits. In 1997, out of 26,161 samples analysed, 64 contained residues above the relevant maximum residue level (MRL) for POPs. Within the meat-testing program, only one sample in 5895 tested for organochlorines was above the MRL. This was a residue of dieldrin in a cattle sample (beef fat). In the other programs (egg, grains and horticulture), organochlorine residues were negligible. From 1993 to 1997, the NRS has detected a low incidence of organochlorine residues in raw food groups. There has been a progressive decline in the percentage of organochlorine residues found in animal fats during the 1990s as shown in Fig. 17.1, while organochlorine residues have not been detected since 1992 in grains. However, there remains continued surveillance of sheep and cattle by National, State and Industry Programs such as the National Organochlorine Residue Management Program. No organochlorine residues have been detected in selected seafood samples (abalone, scallops and southern bluefin tuna) taken in 1995 or freshwater crayfish (yabbies and marrons) sampled in 1996.

An extensive array of foods have been analysed for the dioxins and furans. As expected foods high in lipids were the highest in the lipophilic dioxins and furans which were estimated as one value based on a

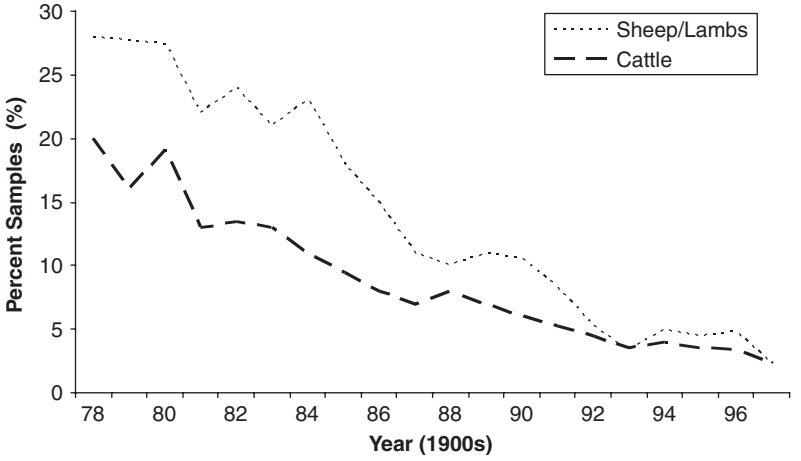

Figure 17.1. Sheep and cattle samples with detectable DDT and organochlorine residues.

summation of the toxicity of the individual components, as toxicity equivalents (TEQ). For example fish fillets, 0.59–0.64 pg TEQ g^{-1} and butter, 0.028–0.270 pg TEQ g^{-1} were the highest observed (National Dioxins Program, 2004).

17.5. Australian market basket survey (now known as Total Dietary Survey)

The Australian Market Basket Survey (AMBS) was set up to monitor pesticides and contaminants in food and estimate their intakes in diets of Australians (ANZFA, 1998). The first total diet survey was conducted by the NHMRC in 1970 and surveys have occurred regularly since then. Currently, it is a biennial survey organised and published by the Australia New Zealand Food Authority (ANZFA, 1998).

Few foods contain residues of organochlorines. However some other pesticide residues (organophosphorus and synthetic pyrethroids) have increased in prevalence relative to organochlorines. Detectable levels of deregistered OCPs arise as a result of ongoing environmental contamination (ANZFA, 1998). A marked decline in residue levels (and intakes) occurred during the 1980s to 1990s as restricted uses, deregistration and regulatory controls on organochlorines progressed. Less persistent endosulfan and dicofol residues are evident in several foods (e.g. fruit and vegetables) in more recent surveys. No residues of 2,4-D and 2,4,5-T herbicides have been detected in targeted food surveys.

17.6. Occurrence of POPs in humans

17.6.1. Human lipid

The earliest Australian survey of human fats was published by Bick (1967). Biopsy specimens of human body fat were collected from 53 individuals of the general population. In this sample, the mean concentrations of total DDT equivalent and dieldrin stored were 1.81 mg kg^{-1} (ppm) and 0.046 mg kg^{-1} (ppm), respectively (100% positive).

Significantly higher levels of DDT and dieldrin were reported by Brady and Siyali (1972) in a survey of 75 human fat specimens. All the samples contained DDT and also HCB, which had not been previously reported in other surveys. An extensive NSW survey in 1988 was conducted by Ahmad et al. (1988) on 290 samples of human body fat obtained from the Westmead Hospital, Sydney. Western Australian levels of total DDT and dieldrin in body fats were shown to be elevated in the late 1960s. However by 1991 total DDT and dieldrin levels had decreased substantially (Stevens et al., 1993).

As well, HCH isomers, including lindane, occur infrequently in human fats. Levels of up to 2.6 mg kg^{-1} were reported in 1972 by Siyali (1972). More recently, Quinsey et al. (1995) detected low median levels (~0.1 mg kg^{-1}) of HCH isomers, with a measure of 4.4 mg kg^{-1}, in human milk fats for Victorian women. The presence of HCH isomers in all of the milk fats suggests a persistent 'background' level of contamination in body fats consistent with POPs profiles for total HCH.

17.6.2. Blood

In Australia, the most extensive set of data involves 4640 blood samples analysed for OCPs at the New South Wales Workcare Authority from 1987 to 1989. The vast majority of samples contained less than the occupational limits 50 ppb (μg L^{-1}) of dieldrin and less than 20 ppb (μg L^{-1}) of heptachlor. Of the 49 samples above the occupational limit 44 of these were from pest control operators (NHMRC, 1992). An earlier NSW study by Siyali (1972) compared whole blood levels of organochlorine insecticides from persons with and without industrial pesticide exposure. Levels of HCB, total DDT, dieldrin and HCH (BHC) were substantially higher in the exposed group, particularly for dieldrin.

DDT levels were tested in the whole blood of an agriculture usage area and unexposed residents during 1973 in NSW. The mean DDT levels in the exposed and non-exposed groups were 21.9 ppb and 16.7 ppb, respectively. The DDT levels in the unexposed groups from NSW were

comparable with UK and USA figures for blood levels of DDT and DDE (Ouw & Shandar, 1974).

Blood levels of aldrin in persons exposed to airborne aldrin in dwellings treated for white ants was investigated by Gun et al. (1994). The mean blood dieldrin (ppb or μg L^{-1}) increased from 0.75, prior to treatment, to 1.2; 3 months after treatment.

A survey of the occurrence of dioxins in 9000 human blood samples collected from individuals resident over the major parts of Australia was carried out in 2001–2003. Levels were found to increase consistency with age with adult of age >60 years having almost three times the levels of those of age <16 years. The mean level was 10.9 pg TEQ g^{-1} of lipid which is among the lowest recorded in the world.

17.6.3. Human milk

The excretion of organochlorine insecticides in human breast milk has raised questions about the safety of breastfeeding for infants. Human milks usually contain much higher levels of organochlorines than cow's milk. The main interest in breast milk studies emerged during the 1970s to early 1980s when food residue levels in Australia peaked and later declined following Federal Government phasing out of the OCPs. Nevertheless, follow-up studies appear to be limited (see Quinsey Some breast-fed infants have had daily intakes of organochlorine insecticides (e.g. total DDT, dieldrin and heptachlor) above acceptable daily levels for these insecticides during the early 1990s (Stevens et al., 1993; Quinsey et al., 1995).

Australian health authorities and researchers have conducted a number of studies into organochlorine insecticide residues and human milk that span three decades of organochlorine use, from 1969 to the early 1990s. Early Australian studies of human milk contamination (e.g. HCB, DDT and metabolites, HCH isomers and cyclodienes) cover the period from 1969 to 1972 and include Western Australia (Stacey & Thomas, 1975), New South Wales (Siyali, 1973), Victoria (Monheit & Luke, 1990) and Queensland (Miller & Fox, 1973). Total DDT was shown to occur in the parts per billion range (μg kg^{-1}) in whole milk and in the parts per million range (mg kg^{-1}) in milk fats.

A useful indication of the long-term changes in organochlorine pesticide residues in human milk is obtained from a summary of Western Australia levels from 1974 to 1991, as presented by Stevens et al. (1993) in Table 17.2. The results indicate that mean DDT and dieldrin levels have fallen to 20% or less of 1974 values with substantial decreases occurring between 1982 and 1990. HCB levels fell rapidly in the mid-1970s to 1980.

Table 17.2. Mean concentrations of organochlorine pesticides in breast milk fat, Western Australia (μg kg^{-1})

Pesticide	Years					
	1974	1978	1980	1982	1990	1991
DDT	3600	2000	1200	1100	360	800
Dieldrin	240	240	170	160	40	50
HCB	2600	750	150	NA	30	100
Heptachlor	NA	NA	140	80	20	20

Source: Stevens et al. (1993).

A low residual level appears to remain. Heptachlor levels were relatively low but have decreased further to under 20% of the 1980 value. Similar trends have been observed in other investigations, e.g., Quinsey et al. (1995).

During 2003 the National Dioxin Program (2004) carried out a survey of human breast milk using the WHO design which was used by that organisation during previous investigations. A range of metropolitan and rural regions throughout Australia was surveyed giving a mean of 9 pg TEQ g^{-1} lipid and no differences between regions. This was among the lowest reported by WHO for human breast milk.

17.7. Dietary exposure of the Australian population to POPs

The dietary intake of OCPs is considered the main source of exposure for the general population in Australia and in many other countries. Estimates of dietary intakes of OCPs from AMBS are summarised in Tables 17.3 and 17.4 for the years 1971 to 1996. The initial survey in 1970 estimated that excessive intakes of dieldrin and HCB could occur in the diets of 15–18 year old males. Dieldrin and HCB had estimated daily intakes from 0.4 to 1.8 (0.1) and 0.7 to 1.4 (0.6) μg kg^{-1} bw, respectively. For comparison the accepted daily intake (ADI) at the time is shown in parenthesis (NHMRC, 1971). By 1976, the estimated daily intake of dieldrin remained relatively high, particularly for infants (e.g. 0.1 μg kg^{-1} bw), while HCB residues in foods were too low to estimate a dietary intake.

Since the 1970s, the intake levels of DDT have decreased by a factor of 10 to 100 in the case of adults and children aged 12. However the levels in the diet of infants were a significant fraction of the ADI although below the ADI. Also the levels of the lower concentration pesticides, such as heptachlor and the HCHs were a small fraction of the ADI by the late 1990s. In contrast to OCPs, total endosulfan intakes increased between 1992 and

Table 17.3. Total DDT—estimated daily dietary intake ($\mu g\,kg^{-1}$ bw) for six age-sex categories

Year	Adult male	Adult female	Boy aged 12	Girl aged 12	Child aged 2	Infant 9 months
1976	0.457	0.345	0.543	0.506	0.898	0.737
1977	0.371	0.396	0.617	0.483	0.898	0.947
1979	0.286	0.241	0.444	1.368	0.653	1.158
1987	0.026	0.022	0.033	0.024	0.027	0.016
1990	0.001	0.001	0.001	0.001	0.003	0.546
1992	0.007	0.0054	0.0053	0.0051	0.0152	1.1035
1996	0.0063	0.0047	0.006	0.0167	0.0063	1.748

Table 17.4. Dieldrin—estimated dietary intake ($\mu g\,kg^{-1}$ bw) for six age-sex categories

Year	Adult male	Adult female	Boy aged 12	Girl aged 12	Child aged 2	Infant 9 months
1976	0.043	0.0345	0.0494	0.046	0.082	0.102
1977	0.039	0.034	0.05	0.04	0.065	0.063
1979	0.038	0.034	0.0666	0.053	0.0898	0.105
1987	0.007	0.007	0.009	0.008	0.008	0.001
1990	0	0	0	0	0	0
1992	0.0048	0.005	0.0048	0.0048	0.0068	0.0041
1996	0.0015	0.0023	0.0017	0.0001	0.0019	0.0043

1996 surveys, which is a trend reflecting use. However the intake estimates do not take into account variation in consumption among localities, socioeconomic, ethnic or other groups. Whole dietary patterns may vary for the 'hypothetical diets' used in the AMBS (Harvey et al., 1998).

The dietary trends plotted for the AMBS data are illustrated in Fig.17.2 (dieldrin) and Fig. 17.3 (DDT). Since 1976, there has been an exponential decrease in DDT and dieldrin intake in the diet. However it is interesting to note that the pattern of decline with dieldrin is different for the different population groups of adult males, adult females and infants as shown in Fig. 17.2. The rate of decline in the adult males follows first-order kinetics ($r^2 = 0.90$) with a half-life of about 3 years as shown in Fig. 17.3.

For all age groups the monthly dietary exposure to the dioxins ranged from the highest for infants and toddlers at an average of 15.6 pg TEQ kg^{-1} bw $month^{-1}$ to 3.7 pg TEQ kg^{-1} bw $month^{-1}$ for adults. This is similar to the levels of dietary exposure in New Zealand at a median value of 11.1 pg TEQ kg^{-1} bw $month^{-1}$ but lower than that observed in other countries such as the United Kingdom.

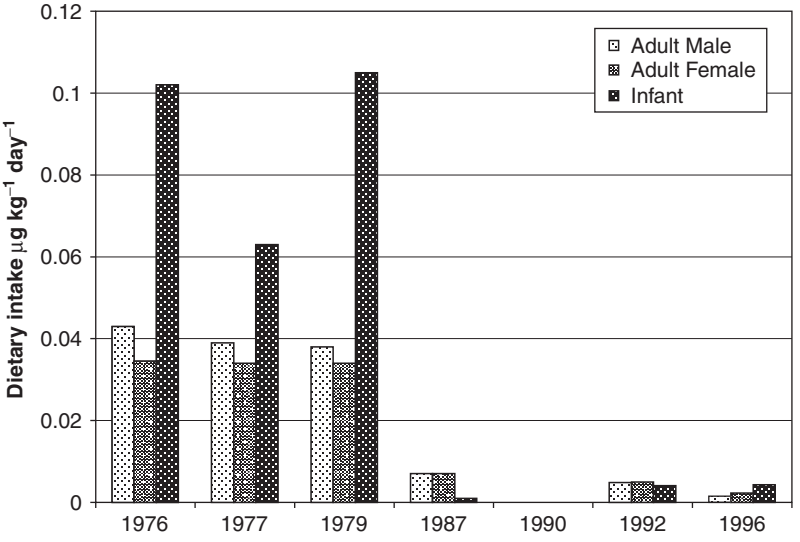

Figure 17.2. Estimated dietary intake of dieldrin for Australian adults and children (1976–1996).

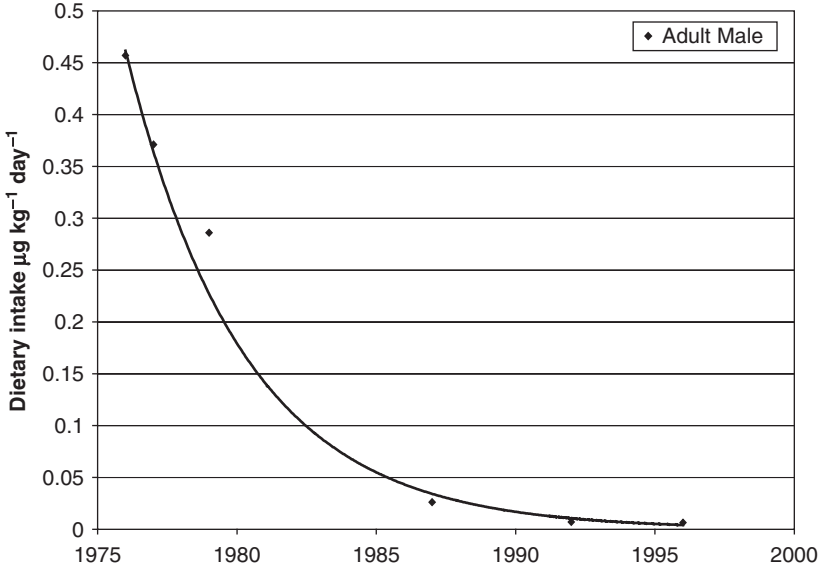

Figure 17.3. Trend in estimated dietary intake of total DDT for Australian adult males (1975–2000).

17.8. Risks to human health in Australia

Most POPs are readily absorbed (via the digestive system and inhalation) and accumulate in fatty tissue, including brain and adipose tissue and human milk. These substances can induce neurological effects and cause altered functioning of the nervous system as well as acting as endocrine disruptors.

ADIs are based on experimental data, which allows an intake level to be set below which adverse effects are not expected. The ADIs are recommended by the WHO/FAO Joint Meeting and Pesticide Residues (see Table 17.5). ADIs are not necessarily set values and can change based on reviews of toxicological information by WHO/FAO and Australian authorities. For example, the ADI for DDT and analogues has reduced by a factor of 10, from 20 to 2 $\mu g\ kg^{-1}$ bw day^{-1} (see Table 17.5) and in some cases where detection has not occurred in many years the residue limit has been cancelled, e.g. BHC and HCB.

To enable a comparison of hazards from chemicals a Hazard Index (HI) can be calculated as the ratio to the ADI. In addition to dietary uptake the possible pathways of entry of POPs for humans are

1. eating foods—dietry
2. contact with dusts or soils
3. drinking water
4. breathing air
5. absorption through skin—considered to be minor here but important for occupational or workplace exposures.

The amount of certain POPs which could enter the human body through contact with dusts or soils, drinking water and breathing air was calculated for the general population in Australia. Thus the total intake for POPs was calculated for 1996 and the risks to the general Australian population have also been calculated and the results are shown in Table 17.6. In addition the risks for specific population groups have been evaluated. These groups are breast-fed infants, high intake consumers of seafoods, persons living in houses treated for termite control, residents living on former livestock sites, persons consuming vegetables from gardens treated with biosolids and rural residents consuming tank drinking water.

From the selection of specific population groups as identified above, it was clear that the risks from being exposed to OCPs can vary for different groups of individuals in the community. In 1996 a variation of a HI of 3.3 for dieldrin for a hypothetical group of high intake seafood consumers to a small fraction for the other pesticides was estimated. Many persons

Table 17.5. Acceptable daily intakes for organochlorine pesticides

Pesticide	ADI μg kg^{-1} bw day^{-1}	
	NFA (1992)	ANZFA (1998)
Aldrin	0.1	0.1
Chlordane	0.5	0.5
Dieldrin	0.1	0.1
BHC (HCH)	20	No Australian ADI
DDT and analogs	20	2
Dicofol	2	1
Heptachlor and epoxide	0.1	0.5
HCB	0.6	No Australian ADI

Source: ANZFA (1998).

Table 17.6. Risks from total intake of organochlorine pesticides for the Australian population in 1996

Pesticide	Period	Persons	Daily intake (μg kg^{-1} bw day^{-1})	ADI (μg kg^{-1} bw day^{-1})	Hazard index	Lifetime risk estimate
Total DDT	1996	Adults	0.007	2	<0.1	$<1 \times 10^{-6}$
		Children	0.009		<0.1	$<1 \times 10^{-6}$
		Infant	1.7		0.9	
Dieldrin		Adults	0.003	0.1	<0.1	3.7×10^{-5}
		Children	0.004		<0.1	3×10^{-5}
		Infant	0.004		<0.1	
Total heptachlor		Adults	0.0006	0.5	<0.1	1×10^{-6}
		Children	0.002		<0.1	1×10^{-6}
		Infant	0.02		0.04	4×10^{-5}
Heptachlor epoxide		Infant	0.02	0.5	<0.1	

living in homes previously treated with organochlorine termiticides are likely to have been exposed to abnormal or excessive levels in indoor air (and household dusts) in the short term (months to years) with the HI for dieldrin up to 1.25. However for most people, intakes during 1996 indicate a safe level in terms of acceptable daily intakes. Lifetime carcinogenic risks (US-EPA model) were also estimated to be conservatively low (see Table 17.6), although uncertainties exist for persons exposed to higher intakes in the 1960s and 1970s.

Breast-fed infants were an unusually exposed group to DDT, with a HI of 0.9, and dieldrin at 0.04 in 1996. Human milk studies in Australia have shown a marked decline in residues but a significant proportion of

breast-fed children have been exposed to levels above the Acceptable Daily Intakes for total DDT and dieldrin and occasionally, heptachlor epoxide.

Similarly, a report produced by the NHMRC (1992) on cyclodiene insecticide use in Australia provides a summary of the toxicological and chronic health effects of aldrin, dieldrin, chlordane and heptachlor.

The levels of total DDT in the Australian environment can be considered to be represented by the data in Fig. 17.3 since this represents a number of food groups including fish, which are usually the most contaminated group with lipophilic substances. This data measured during the 1970s to 1990s follow first-order kinetics ($r^2 = 0.90$) with a half-life of about 3 years (Connell et al., 2002). This decline is due to cessation of usage, environmental distribution characteristics as well as degradation. Extrapolating from the 1976 to 1996 data indicates that the HI for breast-fed infants would be <0.1 and thus a low order of health hazard. Dieldrin in the Australian environment has been suggested to have a half-life of 1.1 to 1.5 years (Connell et al., 2002) and, at this rate of decline the levels present in 2005 would be 0.02 to 0.005 of the levels present in 1996. Thus breast-fed infants, high intake seafood consumers and residents of homes treated with dieldrin would be expected to have low levels of HI, <0.1, in 2005.

The National Dioxin Program (2004) estimated that the dietry intake of the substances as the TEQ value and then estimated the HI against the Australian Tolerable Monthly Intake (TMI) of 70 pg TEQ kg^{-1} bw $month^{-1}$. These values showed an increase with age with toddlers 2–4 years having HI of 0.1 to 0.5; individuals 4 to 15 years HI 0.05 to 0.35 and adults HI 0.05 to 0.25. This suggests that the dioxins are a low health hazard in the Australian environment.

Some toxicological effects, such as endocrine disruption, continue to be uncertain. For example recent studies of breast cancer patterns in women (e.g. USA and The Netherlands) have shown a statistical association with organochlorine residues, such as dieldrin (see Høyer et al., 1998). Again, there is considerable scientific and medical debate with other studies indicating no significant association (e.g. Krieger et al., 1994; Zheng et al., 1999). Plausible toxicity mechanisms exist (Shekhar et al., 1997) to the extent that this issue may be important for long-term health risks in Australian women, given a past history of elevated DDT, DDE and dieldrin exposures in the 1970s and 1980s.

In a preliminary Australian study, Taylor et al. (1999) found the levels of OCPs were higher in breast adipose tissue taken from women with breast cancer compared with women with benign breast conditions. DDE levels were significantly different between malignant and benign tissues. Studies of plasma levels of organochlorines and breast cancer risks have

not supported the hypothesis that exposure to DDT (or DDE) and PCBs increases the risk of breast cancer (e.g. Hunter et al., 1997).

17.9. Conclusions

The dioxins are considered to be at a low level in the Australian environment and constitute a low hazard to human health. The dietary intake of OCPs is considered the main source of exposure to POPs for the general population in Australia and in many other countries. From the 1970s, the AMBS of foods has shown a progressive decline in OCPs detected in food. This has occurred as a result of the banning of the OCPs in Australia, which was effectively complete by 1985. Between 1976 and 1996 the OCPs have declined in the environment in accord with first-order kinetics allowing extrapolations to 2005. The HI from all sources, dietry and others, was used to evaluate the level of risk to human health. This was estimated at <0.1 for all the individual OCPs in 2005.

However several POPs, particularly the OCPs and dioxins, remain at low levels in the Australian environment and several remain persistent at low levels in body fats and fluids of Australians. The levels reflect the past use and persistence of OCPs in the Australian environment, contamination of the food chain and the capacity of the body to metabolise and store in body fats. The dioxins remain due to the ubiquitous nature of their sources with combustion as a major source and their persistence. Future trends are likely to mean very low-level residues in human fats of DDE, cyclodienes, HCB, HCHs and dioxins in the long term. Their rate of decline will probably depend on removing HCB from chlorinated industrial chemicals and OCPs from the environment (e.g. remediation of contaminated soils) by hazardous waste treatment methods (e.g. physical, chemical and biological degradation or fixation) or secure landfill.

Some possible toxicological effects such as endocrine disruption continue to be uncertain, particularly for DDT, DDE, dieldrin and dioxin exposures. The history of environmental health shows that safe levels are uncertain and vary as knowledge improves. The long-term trend is often towards lower levels as uncertainties decrease. This should be considered when accounting for the public benefits of further reducing POPs residues and wastes in the Australian environment.

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