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Australian water quality criteria for organic compounds

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AUSTRALIAN WATER QUALITY CRITERIA FOR ORGANIC COMPOUNDS

by
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ABSTRACT

Australian water quality criteria for organic substances.
Nicholson, B C

Presents water quality criteria for a wide range of organic compounds. Criteria are defined as maximum safe levels and derived from scientific data. They are considered for: environment protection, effect on fish for human consumption, drinking water, and water for industrial, agricultural and recreational uses.

Standards (Water Quality); Human Beings; Standards (Drinking Water); Water Use; *Standards (Health)*; Toxicology; Organic Compounds: Pesticides; Aquatic Organisms; Weedkillers; Aquatic Macrophytes; Aquatic Environments; Australia

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PREFACE

The objective of this project is to update the 1974 document "A Compilation of Australian Water Quality Criteria" by B.T. Hart with regard to organic compounds and to produce a monograph on "Australia Water Quality Criteria for Organic Compounds". It is one of several projects being carried out under the auspices of the Australian Water Resources Council aimed at updating the various sections of the original report on Australian water quality criteria. The project has been funded by the Australian Water Resources Council Water Research Fund in association with the Engineering and Water Supply Department, South Australia. It was carried out in the State Water Laboratories of the Engineering and Water Supply Department. The project leader was Mr D.B. Bursill and the project officer was Dr B.C. Nicholson.

GLOSSARY OF TERMS AND ABREVIATIONS

ADI	Acceptable daily intake
EC50	The concentration at which an effect is elicited in 50% of the organisms under the conditions of the test
EEC	European Economic Communities
EIFAC	European Inland Fisheries Advisory Commission
FAO	Food and Agricultural Organisation (United Nations)
IARC	International Agency for Research on Cancer
LC50	Median lethal concentration (Concentration which is lethal to 50% of the organisms under the conditions of the test)
LD50	Median lethal dose (Dose which is lethal to 50% of the animals under the conditions of the test)
MATC	Maximum acceptable toxicant concentration
NAS	National Academy of Sciences (United States)
NH & MRC	National Health and Medical Research Council (Australia)
PAH	Polynuclear or polycylic aromatic hydrocarbon
PBB	Polybrominated biphenyl
PCB	Polychlorinated biphenyl
PCDF	Polychlorinated dibenzofuran
PCN	Polychlorinated napthalene
PCT	Polychlorinated terphenyl
THM	Trihalomethane
US EPA	United States Environmental Protection Agency
WHO	World Health Organisation
WRC	Water Research Centre (United Kingdom)

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SUMMARY AND SCOPE OF THE PROJECT

There is an urgent need for the provision of water quality criteria for organic compounds because of the rapidly increasing production and use of synthetic organic materials, and the toxicity of a number compounds in common use.

This report presents water quality criteria for a wide range of organic compounds and should provide assistance to people with an interest in water quality and environmental matters.

Problems associated with increasing manufacture and use of chemicals are briefly reviewed in the first part of the introduction. A brief review of the occurrences of organic chemicals in the aquatic environment, and the problems which they can cause forms the second part of this introductory chapter. The remainder is a discussion of the concept of "criteria" and how they relate to standards. Criteria are defined essentially as maximum safe levels, and are derived, by definition, solely from scientific data. Because of the assumptions in the derivation process, there will be uncertainties in the resultant criteria. However the procedures used are considered the best available given our current knowledge. It is stressed that criteria should not be used as standards. Standards should be legally enforceable limits, arrived at by a consideration of criteria but tempered by other considerations, e.g. benefit - risk assessment.

A number of water uses are identifiable as important and perhaps requiring different criteria for a particular compound. In this report criteria have been considered for:-

- the protection of the aquatic environment
- the production of fish suitable for human consumption
- drinking water (the protection of human health and aesthetic acceptability)
- water for industrial uses
- water for agricultural uses
- water for recreational uses

The procedures available for deriving criteria, having regard for the various water uses were reviewed, and these are discussed in Chapter 2. The procedure which was considered as most realistic for a particular type of compound, and for a particular water use, has been adopted and used consistently throughout this report.

For the protection of the aquatic environment, criteria are derived from toxicity data using application factors chosen on the basis of the tendency of the compound to accumulate in exposed organisms. Derived criteria are then compared with chronic toxicity data to test their suitability.

For the protection of human health, procedures differ depending on the toxic effect elicited by the compound. For non-carcinogens, criteria are derived from no-effect levels determined in animal experiments using safety factors. For carcinogens criteria are calculated from risk estimates, a lifetime risk of 1×10^{-6} being considered as acceptable. The effects of a compound on the aesthetic quality of drinking water is also considered where relevant.

The remaining uses of water are relatively unimportant in relation to criteria for organic compounds and receiv only minor attention.

At the commencement of the project, attempts were made to determine priorities for compounds for which criteria were to be derived based on data relating to manufacture and usage of chemicals, especially in Australia. However Australian data are limited due to the confidentiality of records and this approach was relatively unsuccessful. These data are included as an Appendix.

On the basis of compounds with high toxicity and frequent detection in the aquatic environment, the following groups of compounds were finally chosen for detailed investigation.

Insecticides - organochlorine
- organophosphate
- carbamate
- miscellaneous

Herbicides
Fungicides
Fumigants and other pesticides
Polychlorinated biphenyls (PCBs) and related compounds
Phenols
Polynuclear aromatic hydrocarbons (PAH)
Trihalomethanes

Detailed criteria for these compounds are discussed in subsequent chapters.

1. INTRODUCTION

1.1 MAN-MADE CHEMICALS IN THE ENVIRONMENT

The production of new and increasing quantities of chemicals is an important part of the scientific and technological development of the twentieth century. Chemicals create and feed technology, and new technology requires the production of new and increasing quantities of chemicals.

The rate of increase in scientific knowledge and technological development needs no emphasis. Our knowledge of chemistry and chemical technology measured in terms of scientific publications, is doubling every 8-10 years (Sklovsky, 1980). The growth in the number of chemicals known to man and the quantities produced is no less impressive and this is particularly true for organic chemicals (Appendix).

It is inevitable that some of these man-made (xenobiotic) chemicals, many of which are quite toxic, are contaminating the environment in increasing quantities. The adverse effects these chemicals may have on the environment and on human health are many and varied (Goodman, 1974; Vouk & Parizek, 1978). They are too numerous to discuss in detail here, but human cancer is one such effect which is of particular concern.

In 1900 cancer caused less than 4% of the deaths in the USA, whereas by 1975 it accounted for 18% of all deaths in that country (Epstein, 1978). Similarly in the United Kingdom it has been estimated that cancer is now responsible for nearly 25% of all deaths under 75 years of age (Doll, 1977). The death rate due to some cancers has also increased markedly in Australia (the death rate due to some has, however, decreased) (Rohan & Christie, 1980). As cancer is predominantly a disease of old age, these increases are no doubt influenced by the increases in life expectancy in western society this century. Better diagnosis may also account for some of the increase.

Of the overall cancer incidence and mortality, it has been estimated that between 60% and 90% is primarily attributable to environmental factors (Boyland, 1969; Doll & Peto, 1981). Dietary factors (high fat, low fibre, caloric excess, etc.), cigarette smoking and alcohol consumption are the predominant "suspects" within the contemporary western environment. However, chemical contaminants in the occupational and general environment are also implicated since a number of chemicals, mainly organic, and chemical substances containing organic chemicals have been shown to cause cancer in man. A larger number have also been shown to be carcinogenic in animals (Tomatis, 1976).

It is impossible at the present time to accurately estimate the increase in cancer due to the various factors. For example, estimates of the proportion of cancer having occupational causes vary between 1-3% (Higginson, 1976) and 40% (Douglas, 1978).

Nevertheless there is a high incidence of cancer and the possibility of a significant proportion arising through exposure to carcinogenic chemicals cannot be ignored. The increase in the number and uses of chemicals with adverse effects on man and the environment is causing concern at the highest level. World Health Organisation (WHO) is actively involved in assessing the effects of chemicals on health (Vouk & Parizek, 1978), and a number of other international organisations are involved in similar activities (Kappeler, 1979).

The problem of environmentally hazardous chemicals in Australia has been investigated by the Australian Environment Council and a number of recommendations for their control made (Australian Environment Council, 1977; Crawford, 1978).

1.2 ORGANIC CHEMICALS IN THE AQUATIC ENVIRONMENT

The number of different organic compounds detected in water has increased considerably in the past 10 years. In 1970 only 66 organic compounds had been identified in freshwater (Davis et al., 1970). A 1973 report (Webb et al., 1973) listed over 200 organic compounds identified in water, mainly industrial effluents, using gas chromatography/mass spectrometry. By 1975, 423 compounds had been reported in various waters, 394 had been positively identified and 325 had been reported in drinking water (Junk et al., 1975). Another report published in 1975 identified and quantified 289 organic compounds in waters; 114 had been detected in drinking water, and 15 of these were considered to be very toxic (WHO, 1975). By the end of 1976, 1 259 were identified in waters including effluents (Shackleford & Keith, 1976). Most of the chemicals listed in these reports were synthetic and a number were carcinogens (US EPA, 1975a; Kraybill, 1976, 1977, 1978; Borneff, 1978).

Increasing pollution by these substances is strongly suggested, the increasing power of analytical techniques allowing the extent of this pollution to be more and more appreciated. With the production of organic chemicals still increasing rapidly (Appendix), further pollution of the aquatic environment seems likely.

Chemical pollution of water can have many sources. The more obvious such as pesticides and herbicides from agricultural practices, chemicals from effluent discharges and accidental spillages have been documented. Less obvious and more widespread sources have recently been identified and will assume more importance in the future. These include aircraft and motor vehicle exhausts, and the formation of trihalomethanes and other chlorinated organics during the disinfection of water.

Organic chemicals in the aquatic environment may cause direct adverse effects on aquatic life or life utilising the water for drinking purposes (Fielding and Packham, 1977; Windle-Taylor, 1978; Pendygraft et al., 1979; Stander, 1980). Adverse effects may also be caused indirectly by accumulation of these chemicals, or their metabolites or decomposition products in the aquatic life used as a food source by other life forms. Even with stricter pollution legislation being enacted in countries such as the USA, it is considered that pollution still poses a major threat to the water supplies of that country (Dickson, 1980).

Although the number of organic compounds identified in waters is increasing rapidly, the compounds being identified fall, almost exclusively, into the molecular weight range below 500. These compounds generally comprise a small proportion (5%) of the total organic content of waters. The remainder would appear to be mainly the fulvic and humic acids, naturally occurring higher molecular weight compounds formed by the degradation of plant material. These are polyhydroxy phenolic compounds and little is known of their structure. Nevertheless, this more readily identifiable group of compounds contains a number of potentially toxic compounds and the health and environmental effects of these materials at low concentrations must be established.

1.3 THE CONCEPT OF CRITERIA

If the entry of chemicals into the aquatic environment cannot be prevented absolutely, then information on the effects of levels of particular chemicals on the users of the water, the flora and fauna of the aquatic environment, or the consumers of aquatic life from that environment is essential. It is also essential to know levels below which it may be considered safe for a particular chemical to be present. Without this information there is no basis for the control of the entry of chemicals into waters and effective water resources management and environmental protection is not possible. Objective levels based on factual data, i.e. criteria, are needed.

The UN Conference on the Human Environment defined criteria as "observed qualitative or quantitative data that predict the change or magnitude of effects caused by an agent or agents on a defined receptor under specified circumstances defined by environmental variables and receptor variables" (Hart, 1974). Such a broad definition encompasses data for all possible situations and would provide the information necessary for water resources management and the protection of the environment.

In practice this information is never complete. It is physically impossible to obtain exposure - effect data for all life forms and for all pollutants under all conditions of temperature, salinity, pH, dissolved oxygen levels, etc. A complete literature survey could uncover all the currently available data but in most cases the experimental information would have been collected under conditions that are not identical or even similar to conditions in practice. In addition a complete compilation of data alone does not provide a convenient guide for water resources management. An identification of safe or no-effect levels for individual compounds having regard to the various water uses, is the most useful of the information.

The US Environmental Protection Agency in its document "Quality Criteria for Water" (US EPA, 1976a), describes a criterion as a "designated concentration of a constituent that when not exceeded, will protect an organism, an aquatic community, or prescribed water use or quality with an adequate degree of safety". Thus criteria represent maximum safe levels and are obtained solely from scientific data. In most cases they require derivation from the available data and are therefore 'derived' criteria. They are identical in concept to the 'derived working levels' of Hart (1974). The US EPA description of criterion has been adopted as a definition for the preparation of this report on "Australian Water Quality Criteria for Organic Compounds".

Criteria derived in such a way represent safe levels based on the best interpretation of the available data. However they may be controversial as introduction of the word 'adequate' in the definition of criteria leads to subjective bias in their derivation. What may be an 'adequate degree of safety' to one person or organisation may not be considered so by another. Such derived criteria are also rather simplistic but nevertheless useful. They allow a rapid judgement as to whether measured levels of a pollutant are likely to be harmful.

Criteria are not magic numbers below which there is no effect and above which disaster occurs. Because of the limitations and uncertainities in their derivation, they serve only as a rough guide to that level of chemical which can be reasonably tolerated. They should be a starting point in assessing the likely effect of a chemical on the suitability of water for a particular use. It is strongly recommended that derived criteria should also state the basis on which they are derived, and the effects which could be expected from exposure. The tables of criteria for organic compounds in the summary report of Drinking Water and Health (NAS, 1975) are good examples of the information that should be provided with the derived criteria so that the relevance of various assumptions made in the derivation process can be assessed in relation to actual situations. Consequently in applying the criteria detailed in this monograph, it is strongly recommended that the complete section concerning the chemical of interest be considered before determining the relevance of the derived criteria to actual situations.

1.5 THE RELATIONSHIP OF CRITERIA TO STANDARDS

Water quality criteria are not standards. It is generally accepted that water quality standards should be legally enforceable limits for a given substance in water (Hart, 1974; Wells, 1978). For drinking water they should ideally be identical to criteria to provide the maximum protection for drinking water. However standards are influenced by practical and political considerations and how far criteria can be tempered by such considerations often involves benefit-risk assessment.

Benefit-risk assessment of man's activities is a complex undertaking (Burton & White, undated; O'Riordan, 1977; Kletz, 1980). It is also difficult to define acceptable risk (McGinty, 1976) as perception of risk is not always related to the actual risk which exists, i.e. risk is not always equatable with fear (Lerch, 1980). The principles applied in setting of environmental standards in the United Kingdom is a good example of the many factors which must be considered in standard setting (Dept. of the Environment, 1977). In relation to water, other methods can be used for setting standards e.g. background levels and detection limits of analytical methods (Tate & Trussell, 1977; Commins, 1980), but they are generally not desirable for materials of public health significance.

The Australian maximum recommended level for aflatoxins in peanuts and peanut products is a good example of practical (economic) considerations in standard setting. The maximum recommended level was recently increased from 5 ug/kg to 15 ug/kg since a level of 5 ug/kg was not achievable. A major proportion of the peanut crop would have had to have been discarded had the recommended level of 5 ug/kg been maintained with a consequent financial loss to the grower (Anonymous, 1980). Raising the level increases the risk associated with consumption of this product and has generated a great deal of controversy because of the different opinions of acceptable risk.

Several countries and international organisations have promulgated water quality standards or are currently preparing standards. However very few organic compounds have been included. Countries and organisations that have published water quality standards or maximum levels for contaminants include USA (US EPA, 1975), Canada (Health and Welfare, Canada, 1978), South Africa (Wells, 1978), USSR (Stofen, 1973), India (Khirsagar, 1979), Australia, (Australian Department of Health, 1980), WHO (WHO, 1971), and the European Economic Communities (EEC) (Goodman, 1979;Commins, 1980).

The mechanism for enforcement is not clear for most of these "standards" and it is therefore doubtful that they are standards according to the generally accepted definition given above. Although it is difficult to withdraw from consumers a water supply which does not meet a standard, as occurs occasionally with sub-standard food, this recently occurred in the United States when a level of 649 ug/L trichlorethylene was detected in a public water supply (Anonymous, 1981).

Some water quality standards do have legislative support with a detailed course of action if standards are not met. For example, in the United States consumers must be notified in the event of standards not being met (US EPA, 1976b), and the EEC Directive on Quality of Surface Water for Abstraction of Drinking Water requires that certain treatment procedures be provided depending on the raw water quality (Goodman, 1979). The basis for published standards or maximum levels for contaminants is often not clear, which makes it difficult to judge their suitability. As no justification of a drinking water standard is usually given and susceptible individuals are not considered, drinking water standards have been widely criticised (Berlyne & Yagil, 1973).

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2. THE DERIVATION OF CRITERIA

2.1 PROTECTION OF THE AQUATIC ENVIRONMENT

2.1.1 General

The proliferation in the numbers and usage of organic chemicals is placing a heavy pollutional burden on the environment. The degree and variety of effects are extremely complex due to the immense number of physical, chemical and biological interactions that occur within an ecosystem (Crosby, 1975). The aquatic environment must be protected if it is to serve as a source of food or water for other purposes (drinking, and also other uses such as irrigation or industrial).

2.1.2 Criteria based on lethal levels

Safe levels, or criteria, have often been calculated from the simple relationship:

Criterion = application factor x LC50

where the LC50, the median lethal concentration (or the median tolerance level TL_m) is the concentration of the chemical which is lethal to 50% of the organisms under the conditions of the test.

Bioassay procedures (Sprague, 1969; Glass, 1973; APHA, 1975) and methods for calculating LC50 values (Stephan, 1977) have been well documented and are beyond the scope of this report. Bioassay tests are performed such that the LC50 value is determined for a given time period (e.g. 24 hours, 48 hours, 96 hours, etc.). Lethal concentrations for various percentages of the organisms under test can also be determined, e.g. LC10, LC75, etc. The incipient LC50, the level at which 50% of the test organisms survive indefinitely can also be determined (Sprague, 1969). Few long-term toxicity studies have been carried out, but since mortality was found to cease after 4 days in most of the cases studied, the 96 hour LC50 is generally considered a reliable approximation (Sprague, 1969). In calculating criteria which define safe levels for long term chronic exposure to chemicals, the LC50 for the longest exposure, i.e. incipient LC50 is used.

A number of application factors have been used in calculating safe levels. A value of 0.1 has been the most common but occasionally higher values have been used. With the realisation that a species survival may be impaired by events which take place over a long period of time lower and more conservative application factors of 0.05 or 0.01 have been used in deriving criteria for chemicals which have chronic effects on aquatic organisms. For example, persistent organochlorine insecticides can accumulate to high levels in organisms exposed to concentrations very much lower than lethal levels, with consequent deleterious effects, usually reproductive impairment, and application factors of 0.01 have commonly been used with these compounds.

The calculation of safe levels in this way from acute toxicity data obtained in laboratory experiments involves many uncertainties (Sprague, 1971; Lloyd, 1973; Brown, 1976; Hunter, 1978; Price, 1978; Buikema & Benfield, 1979; Cairns & Maki, 1979; Klapow & Lewis, 1979, Krenkel, 1979). Unfortunately, the word safe is subjective in itself, since absolute safety does not exist. Criticisms have been levelled at procedures involving the calculation of criteria from acute toxicity data and the use of application factors (Sprague, 1971, 1976). In particular, attention has been given to the basic difficulties in obtaining reproducible LC50 data (Brown, 1976; Hunter, 1978) and the uncertainties of extrapolating the information to field situations, where additional factors may not be readily apparent. Toxicity can be influenced by a number of factors such as life history of the organism (Buikema & Benfield, 1979), molt cycle (in Daphnia) (Lee & Buikema, 1979), diel variations (in fish) (Spieler et al., 1977), diet (Mehrle et al., 1977), temperature, hardness, pH, dissolved oxygen, salinity and other physical, chemical and microbiological characteristics of the water.

Different populations of a particular organisms may also have different susceptibilities to a toxicant. For example pesticide resistant strains of fish are not uncommon (Fabacher & Chambers, 1976). The different susceptibilities of different life stages to toxic chemicals is also well known, e.g. early stages such as larvae are more susceptible to toxic chemicals than adults (McKim, 1977).

The choice of bioassay technique can markedly affect the value of the LC50 determination. Whether static or flow-through (continuous or semi-continuous (Reynolds et al., 1977)) techniques have been applied is not always obvious from tabulated data, yet all these variations can affect the magnitude of the LC50 value obtained. A recent suggestion that data from static procedures be described with the terminology LC(I)50 (Lloyd & Tooby, 1979) would partially overcome this problem and allow a more objective evaluation of data where the nature of the bioassay technique has not been reported. Because of the large number of uncertainties in determining LC50 levels, these values are not such definitive characteristics as is often implied.

The greatest difficulty in determining criteria from acute toxicity data is the choice of the application factor. There is little scientific basis for application factors except that they are generally the result of careful judgement (Sprague, 1976), more conservative application factors being used for compounds which have long term detrimental effects. There is little evidence, in most cases, that the arbitrary value chosen is indeed the best choice i.e. whether a particular value for an application factor will provide 'adequate' protection and whether a less (or more) stringent value would be more appropriate. Procedures are available for experimentally determining the application factor, but these have been used in only a few instances (see Section 2.1.3). A large range of organisms have been used in bioassay tests. Sheepshead minnows (Cyprinodon variegatus) (Hansen & Parrish, 1977), pond snails (Lymnaea stagnalis) (Canton & Slooff, 1977), ciliates

(Persoonne & Dive, 1978), rainbow trout (Salmo gairdneri) (Wells, 1977), zebrafish (Brachydanio rerio) (Laale, 1977), Daphnia (Leeuwangh, 1978; Adelma, 1978), some crustacea (Lee, 1977; Nimmo et al., 1977) and copepods (Nitrocra spinipes) (Bengtsson, 1978) are among those that have been recommended for waters of various salinities. Some evaluations of their suitability in toxicity testing procedures have also been made (Fogels & Sprague, 1977; Kenaga, 1978; Kenaga & Moolenaar, 1979; Maki, 1979). In deriving a criterion for a particular organism, the toxicity data should ideally be available for that organism. To use data from another organism will introduce some uncertainties. In order to protect the aquatic environment as a whole, the most sensitive organism in that system must be protected and toxicity data should therefore relate to this organism in the derivation of criteria.

2.1.3 Criteria based on sublethal effects

Since a population of organisms may be decimated by a chemical without the chemical having acute or even chronic lethal effects, e.g. by impairing reproduction, criteria can best be derived from data obtained from studies of chronic toxicity over the entire lifecycle of an organism. These effects on an organism which are not directly responsible for death are termed sublethal effects (Sprague, 1971; Koeman & Strik, 1975; Rosenthal & Alderdice, 1976; Anderson & D'Appolonia, 1978). The tendency to accumulate in exposed organisms (bioaccumulation) is an important property of some chemicals which may lead to these effects. For example organochlorine insecticides and polychlorinated biphenyls are resistant to degradation in the aquatic environment and have high fat solubilities. They therefore accumulate in the fat of exposed organisms and once solubilised, are particularly resistant to degradation or elimination. Further concentration also occurs through biomagnification along the food chain i.e. by ingestion of contaminated organisms. Accumulated residues have been shown to be responsible for deaths (Young et al., 1979) and reproductive failures in predatory birds (Rudd & Herman, 1972, Menzie, 1972) and fish (Menzie, 1972; Johnson & Ball, 1972), and can result in levels of contamination in animals which prohibit their use for human consumption.

Bioaccumulation factors, like LC50 levels, vary from species to species and are difficult to reproduce in the laboratory due to the influence of many compounding variables (Ernst, 1979). The relevance of some sublethal effects to the derivation of criteria is not always clear (Sprague, 1971; Lloyd, 1972). Effects such as impaired reproductive success, e.g. hatching and survival rates, mutagenic and teratogenic effects are obvious factors which affect the survival of a species. However, a number of other more subtle effects have been documented, for example, the impairment of feeding and swimming ability, changes in respiration rate (Sprague, 1971), alteration of choice of prey by predators (Farr, 1978), and changes in enzyme levels (Janicki & Kinter, 1971) have been identified but it is difficult to determine how critical these changes are to survival. Some effects such as impairment of food locating abilities may be important in the wild but may not be evident in laboratory based experiments.

In deriving a maximum safe level for a particular organism, data from an entire lifecycle toxicity study involving reproduction would therefore be the most suitable to use. Shorter tests with early lifestages have been reported to yield similar results (McKim, 1977; Macek & Sleight, 1977). Thus data from these tests which greatly simplifies the production of chronic toxicity data could be also used.

Studies utilising lifecycle tests for Daphnia have also produced data which correlate well with those generated in longer term experiments with fish (Maki, 1979), and since Daphnia are generally among the more sensitive of organisms to toxicants, chronic toxicity data for these organisms are generally important for criteria derivation. As Daphnia have very short lifecycles in comparison with fish, they represent an ideal subject for the determination of toxicities of chemicals for use in criteria derivation.

From chronic toxicity tests, a parameter called the maximum acceptable toxicant concentration (MATC) can be derived. This is the maximum level of the toxicant tested at which no adverse effects were determined, and corresponds to a safe level for that organism (Mount & Stephan, 1967; Eaton, 1973). The ratio of the MATC to the acute toxicity value (LC50) can provide an experimentally determined application factor since it relates the LC50 value to the no-effect (or 'safe') level. It has been found that application factors obtained with a particular toxicant with different species are reasonably constant (Eaton, 1973). Thus multiplying the LC50 value by the experimentally determined application factor will give safe levels for organisms for which a MATC has not been determined, but for which an LC50 value is available. For a few chemicals a number of such application factors have been experimentally determined and, if applied to the most sensitive species, will provide a safe level for that species which is also a criterion for the protection to the aquatic environment as a whole. Thus an objective water quality criterion based on acute toxicity and sublethal effects can be derived.

A similar approach to criteria derivation has been suggested by Canton & Slooff (1979). They have suggested that an ecological limit, which is very similar to a derived criterion, be established for pollutants. This require the determination of the no-toxic-effect level (NTEL) which is equivalent to the MATC. Also determined are the EC25 and EC50 levels, the concentration which relate to a particular effect in 25 and 50% of the organisms respectively. The ecological limit is defined as: NTEL x EC25/EC50

Since the NTEL and EC data may be available for a number of organisms and a number of effects, the lowest value of NTEL x EC25/EC50 is chosen as the ecological limit. Once again in order to protect the aquatic environment as a whole, this should apply to the most sensitive species. The EC data should also relate to the most marked effect, i.e. the effect elicited by the lowest concentration of toxicant.

There are still uncertainties in applying sublethal-effect data of this kind to the derivation of criteria since major differences occur between laboratory conditions and those in the natural environment. The toxicity and behaviour of chemicals in microcosms have been studied by a number of workers (Taub, 1976; Cripe & Livingston, 1977; Wilkes, 1978; Bourquin et al., 1979). Studies of this kind may lead to more realistic values of criteria if the microcosms can be made to duplicate the complex natural environment with its many and diverse interactions.

Another approach to describing the likely effect of a chemical on the environment which is essentially a criterion setting process is the 'rating' or 'profile' system (Weber, 1977; Korte et al., 1978, Freitag et al., 1979). The properties of a compound are quantitatively determined in a number of standard experiments. Properties such as bioaccumulation, metabolism, persistence, etc., are measured, the overall hazard estimated and the observed properties compared with those compounds for which environmental behaviour is better known. This procedure provides predictive evaluation of the likely adverse effects of a given chemical, and may become a useful screening tool. It has already been used in proposals to regulate the discharge of toxic chemicals. A similar computer based screening procedure has also recently been described (Neely, 1979).

2.1.4 Criteria based on field observations

In order to avoid the difficulties inherent in extrapolating data obtained in laboratory experiments to the field, criteria can also be derived from field observations. Although field observations would appear to provide more appropriate conditions for acquiring data for criteria derivation, there is seldom sufficient quantitative cause-effect data for this to be carried out.

The effects of pollution on the ecosystem can be monitored by studying the number and distribution of the organisms within the ecosystem. The study of the effects of pollution using diversity indices (Van Belle, 1977; Ghetti & Bonazzi, 1977) is outside the scope of this report, however suffice to point out that its usefulness for developing quantitative criteria is limited by the difficulty in determining the precise nature and level of the compounds responsible for the effects recorded, and the other variables involved.

Simpler procedures based on gross observations have been reported. These have involved the use of caged or confined fish, or natural populations for acute toxicity studies and monitoring of particular sublethal effects (Cairns et al., 1977; Price, 1978; Slooff, 1978; Kingsbury & Rees, 1978; Koeman et al., 1978). They have the advantage that pollution is detected rapidly whereas diversity indices generally take some time to become apparent. With sublethal responses it is again difficult to ascertain the significance of these responses. Nevertheless their presence is sufficient to warn that further action needs to be taken.

An increased tumour incidence in aquatic animals in polluted environments has been well documented (Rose, 1976; Mearns & Sherwood, 1976; Stich et al., 1976; Rose & Harshbarger, 1977; Windsor et al., 1977; Kraybill et al., 1977) and the detection of carcinogens in aquatic environment by monitoring tumour incidence has been suggested (Stich & Acton, 1976). Mutagens have also been monitored in mussels using the Salmonella microsome test on the tissue (Parry et al., 1976). If the significance of these environmental observations can be ascertained and quantitatively related to exposures to particular chemicals, then a useful water quality criterion could be calculated.

2.1.5 Approaches adopted

The derivation of criteria for all organisms under all conditions is an overwhelming task, and no single procedure is entirely satisfactory. Through a consideration of all the data available for a particular chemical (acute and chronic toxicity, sublethal effects, physical and chemical behaviour, etc.), safe levels for the most sensitive species can be estimated. The protection of the aquatic environment can best be assured by the protection of the most sensitive species. Derivation of criteria is thus essentially a predictive exercise since the available data will have rarely been generated under conditions identical to the system of interest.

How criteria are derived will depend somewhat on what data are available, and the relative importance of the various known effects. Because of the limited data in most cases, and the difficultly in interpreting its significance, the assumptions made in the criterion derivation process will often be suspect.

The US EPA has recently derived draft criteria for the protection of freshwater and saltwater aquatic life, and human health for a number of organic compounds (US EPA, 1979a, b, c). The procedure used for the protection of aquatic life was complex and highly formalised (US EPA, 1979a), based on both acute toxicity and subletnal effect data. Derived criteria consisted of two concentrations, a concentration not to be exceeded at any time, and a concentration as a 24 hour average. Although criteria were calculated in a formal manner by a set procedure there were still many assumptions in the calculations. It is therefore considered that such criteria have no more justification than those calculated by more simplistic procedures. Earlier, US EPA criteria were calculated by a number of procedures from various kinds of data as discussed by Sprague (1976). This was quite complex in some cases, e.g. the criterion for DDT in freshwater was based on an estimate of the no-effect level for DDE on reproduction of pelicans. This was extrapolated to a no-effect level in the food of ducks, which coupled with the bioaccumulation potential yielded the criterion of 0.001 ug/L (US EPA, 1976b).

Criteria based on field observations have also been reported. The European Inland Fisheries Advisory Commission (EIFAC) water quality criterion for phenols of 1 mg/L for salmonid fish is one such example. Waters with phenols at a concentration of 1 mg/L supported fish, including salmonids but at concentrations greater than 3 mg/L after an industrial discharge, salmonids were killed in these waters (EIFAC, 1973).

In this report criteria proposed for the protection of the aquatic environment have been derived on the following basis. From a compilation of the acute toxicity data for individual compounds judgement was made as to what is the most sensitive class of organisms (e.g. fish, crustacea, insects, etc.). If a number of the most sensitive class had been tested, the data were judged to be adequate for criterion derivation. The most sensitive species tested were then assumed to be the most sensitive species in existence. Although this can never be guaranteed because of the huge number of aquatic organisms, it was felt that such assumptions would be sufficiently valid to yield a reliable criterion estimate. Where LC50 data were available for a number of time periods for the most sensitive organism the 96 hour value was usually chosen, however in some cases, values for longer time periods were used if they appeared more appropriate.

The following application factors were chosen: 0.1 for compounds which do not accumulate and 0.01 for compounds which do. Where possible, this application factor was compared with application factors experimentally determined (i.e. the ratio MATC/LC50). A compromise was made if the agreement was poor.

Criteria were then calculated and compared to maximum no-effect levels determined in sublethal effects investigations. The criteria were sufficient to protect against these sublethal effects in nearly all cases. Where protection was not afforded, the criterion was lowered to the lowest maximum no-effect level reported.

No distinction was made between freshwater and saltwater environments, nor ecosystems of any particular type. Since criteria were felt to be rather crude numbers because of the approximations and assumptions involved, a distinction between different environments was not considered justified.

Although the approaches adopted have many uncertainties and are somewhat subjective, they are nonetheless most useful. The monitoring of pollutants and their effects on the aquatic environment can provide an indication of the accuracy of criteria and the derivation process. The usefulness of derived criteria can be compared to some degree with speed limits. Speed limits are derived from practical considerations with some limited knowledge of the benefits and risks arising out of any alternatives. Ideally there should be a limit for different cars on different roads under different weather conditions, time of day, state of repair of the car, etc. Clearly such a system would be unworkable, even if the limits for each situation could be scientifically evaluated. The current limits are a practical compromise; they guarantee a high degree of safety but not absolute safety.

Criteria should therefore be regarded as rough guidelines only. When all aspects of the behaviour of a chemical in an ecosystem can be described with the utmost accuracy, then a criterion which relates to that ecosystem can be obtained with some confidence. More work is required in evaluating the best procedures for determining derived criteria. The toxicity data for a number of compounds are very limited, and more work is required to provide these data. Information on the toxic effects of organic chemicals on Australian aquatic organisms is unfortunately almost non-existent, and represents one area where data are urgently required (Cairns, 1980). Since the variations in susceptibilities to toxic chemicals between organisms of any type (e.g. fish) are not large (Sprague, 1970), data in overseas experiments should however, be more or less transferable to the Australian situation. As suggested by Cairns (1980) this can easily be substantiated by tests on Australian species using a few of the more common toxicants.

2.2 PRODUCTION OF FISH SUITABLE FOR HUMAN CONSUMPTION

Criteria for compounds which accumulate in fish tissues may be considered sufficient to provide adequate protection for the fish, however, the accumulated levels in the fish may be unacceptable for human consumption.

The bioaccumulation and human consumption consideration have been used in deriving US water quality criteria for a number of compounds including aldrin/dieldrin (US EPA, 1976a). The resultant criteria were considered to relate to the protection of freshwater life but are really for water supporting fish for human consumption. Using a generally accepted application factor of 0.01 for compounds which accumulate, criteria established for protection of the aquatic environment, based on the acute toxicity of the most sensitive organism tested, (the 20 day LC50 for the stonefly) would be half that derived using the human consumption data and bioaccumulation factors. Thus two different criteria can be derived; the one based on human consumption and bioaccumulation data not being sufficient to provide for protection of the aquatic environment by the generally accepted derivation procedures. However the criterion for the protection of the aquatic environment would be adequate to provide for fish suitable for human consumption in this case. Where appropriate, criteria for water supporting aquatic life for human consumption were discussed in the sections on the individual compounds.

A similar approach to criterion setting has been adopted by the European Inland Fisheries Advisory Commission (EIFAC) for phenol (EIFAC, 1972). The criterion, which is the basis of an EEC directive (WRC, 1979), relates to the prevention of tainting of fish flesh rather than the protection of the fish themselves. Again such criteria relate more to a water use rather than protection of the fish. Similarly the US EPA criterion for phenol is loosely based on levels of 2-chlorophenol causing tainting of fish flesh (US EPA, 1976b).

Criteria for phenols based on the tainting of fish flesh are discussed in more detail in Section 4 (Detailed Criteria for Phenols).

2.3 DRINKING WATER (PROTECTION OF HUMAN HEALTH AND AESTHETIC QUALITY)

2.3.1 Criteria based on chronic toxicity

Although acute toxicity data are important in deriving criteria for the protection of the aquatic environment, they have little use in determining criteria for the protection of human health. This applies both to human acute toxicity data (some data are available from suicide cases although the dose is generally not accurately known), and acute toxicity data from animal studies. Criteria for health protection are generally based on data for the effects of long term exposures to small quantities of chemicals, i.e. chronic exposures. Little direct human data are available because of the ethics of human experimentation and animal data must therefore be extrapolated to generate human health criteria.

In general the following procedure is used for the derivation of criteria for health protection with respect to water quality:-
From the available data the maximum 'no-effect level' is determined. The maximum 'no-effect' level is the highest level at which 'no-effect' is observed (or what is generally considered to be 'no-adverse-effect'). This may be well below the true 'no-effect' level (if such a level exists) and will depend on the range of levels investigated. Again the problem arises of determining whether some of the measured effects are important for survival. As methods for detecting effects become more numerous and sensitive, the 'no-effect' level will generally decrease. This level is therefore subject to variation.

The 'no-effect' level is then converted to a daily no-effect intake or dose rate expressed in mg/kg/day if not already measured in this way. For compounds administered at certain concentrations in food, the dose rate can be calculated assuming typical weights of the animals and typical food intakes. The acceptable daily intake (ADI) for man is then calculated from the no-effect daily intake from the animal experiments using a safety factor. The magnitude of the safety factor is dependent on the amount of information available on the toxicity and the nature of the effects of a particular compound. For example a compound with well documented toxicity behaviour tested over a wide range of concentrations with a variety of animals and involving long periods of testing, would require a safety factor less conservative than that for a compound where only a few short term test results were available.

Until recently it was recommended that carcinogenic compounds be treated in the same way, using a very conservative safety factor, e.g. 1/5000 (Weil, 1972). However, as described in Section 2.3.2.1., more recent procedures utilise an estimation of risk as a function of the concentration since it is considered that no 'safe' levels exist for carcinogens. The safety factor procedure still finds use with carcinogens where quantitative information is inadequate for risk assessment to be made.

The use of safety factors is, like the use of application factors, rather subjective although they are based on scientific judgement. The extrapolation of data from animal experiments to safe levels in man is subject to many uncertainties (difference in susceptibilities between different species, adequacy of data, subjective nature of safety factors, etc.) (Loomis, 1975; Dixon, 1976; Sharratt, 1977).

Another approach to determining criteria, or 'safe' levels (Handy & Schindler, 1976) is that based on the earlier procedure of Stokinger and Woodward (1958). This procedure utilises the TLV (threshold limit value), which is the concentration of a substance in air to which workers can be continuously exposed during an 8 hour day, 40 hour week without adverse effect. As TLV data are not available for many of the compounds found in water, the available TLV data were related to their respective LD50 data by regression analysis. This allows 'safe' levels of compounds in water to be calculated from LD50 data if TLV data are not available. This approach, suffers from many uncertainties in extrapolation as well as long-term effects being generally ignored. Determining the TLV, especially where chronic toxicity occurs, is as difficult as determining the water quality criterion itself. As a result this procedure has not been extensively applied to the determination of water quality criteria.

2.3.2 Criteria for carcinogenic compounds

2.3.2.1 Extrapolation of animal data

The derivation of water quality criteria for compounds with carcinogenic properties is the most difficult and controversial area in criteria setting. The mechanism of cancer formation is complex and not well understood (Cairns, 1981). Some progress is being made in specific instances e.g., some compounds appear to be carcinogenic through reactive epoxide intermediates (Garner, 1979; Pullman, 1979). As a result of this lack of knowledge in the fundamental mechanisms, many assumptions and approximations must be made in the derivation process. The problems in establishing water quality criteria for the protection of human health, especially for carcinogens, have recently been reviewed in detail (NAS, 1977b) and will only be described briefly here. Other reports have described the more general problems of determining the relationship between chemicals in the environment and cancer rates (Epstein, 1974; Ember, 1975; Higginson, 1976; Wynder & Gori, 1977; Cairns, 1978; Fishbein, 1979a).

As previously indicated, toxicity data derived from laboratory experiments, by necessity, nearly always involves animals other than man. Thus carcinogenicity data also must be extrapolated from animals in an attempt to predict the effects in man. The two main areas of uncertainty in the extrapolation procedures are whether, for a particular chemical, man responds in an identical manner to the animal tested, and the quantitative aspects of extrapolation from high-dose short-term animal experiments to low-dose long-term exposures to which man is subjected. (Shubik & Clayson, 1976; Fishbein, 1979a; Falk, 1980).

Different animals respond in different biochemical and physiological ways to chemicals, including carcinogens. With carcinogens, different organs may be attacked in different species of animals and the extrapolation of data from animal experiments to man involves uncertainties arising from these differences in response characteristics.

Even within species of animals the results of carcinogenicity experiments may be difficult to duplicate. The results, as in LD50 and LC50 experiments, depend on many variables some of which are not easily identifiable (Garner, 1979). Whether a chemical is judged to be carcinogenic may well depend on the design of an experiment (Food & Drug Administration, 1971). Therefore it is not always easy to decide whether a chemical is carcinogenic or not. The International Agency for Research on Cancer (IARC) has an ongoing program of evaluating the carcinogenic risk of chemicals to man by assessing the available data (Tomatis, 1976).

All compounds known to be carcinogenic in man are also carcinogenic in at least one animal species (with the possible exception of arsenic) (Epstein, 1974; Shubik & Clayson, 1977). Whether the converse is true is an area which is being strongly debated. It is argued by some that evidence of carcinogenicity in animal experiments does not necessarily prove a chemical is carcinogenic in man, i.e. there can be species specificity. This argument is supported by the fact that different organs may be attacked in different species, and that large variations in susceptibilities to carcinogens occur between various species. For example DDT causes cancer in mice but not in other animals tested (Shubik & Clayson, 1977). 2-napthylamine produces cancer in man, monkeys and dogs but not in rats, mice, guinea pigs and rabbits (Epstein, 1974). Aldrin and dieldrin are good examples where prolonged legal processes have been involved in deciding whether a compound is a human carcinogen. Aldrin is rapidly converted to dieldrin in animals, so the behaviour of both can be considered together.

A comparison of six carcinogenicity studies for these insecticides carried out on rats and mice showed three to be positive for tumour formation and three negative (Ritper, 1979). This indicates experimental dosing may influence the results, with the result that a decision on the carcinogenic nature of the chemical may be difficult to make, even with animals. Furthermore, aldrin and dieldrin appear to show species specificity for tumour formation (Epstein, 1974; Van Raalte, 1977; Wright et al., 1977) and currently they are considered to be carcinogenic in the USA but not in Britain (Gillespie et al., 1979).

Epidemiological evidence has also been used to argue the case for species specificity. Phenobarbital has been widely used as a treatment drug for epilepsy, but has recently been shown to be carcinogenic in mice. It produces tumours identical to those produced by dieldrin. Epidemiological studies have shown no increased cancer rates in epileptics treated with phenobarbital (McLean, 1979).

Another consideration in applying animal carcinogenicity data to man is the relatively short life span of animals (approximately 2-3 years for mice) in comparison with man. It has generally been assumed that the latency period for tumour production is directly related to lifespan, although this requires verification. It is difficult, therefore, to ascertain how this difference in lifespan should be treated in the derivation of human carcinogenicity risk estimates from animal data.

In the interests of human safety, a conservative approach would appear to be preferred. A chemical shown to be carcinogenic in animal studies must be assumed to be carcinogenic in man since a risk to health may exist (Tomatis, 1976). It is, in fact, through the adoption of this approach that the discovery of the three most important carcinogens since 1970, i.e. stibesterol, bis-(chloromethyl)ether and vinyl chloride, was made (Maltoni, 1976; Tomatis, 1976).

The use of high dose rates for animal experiments is necessary in practice for the positive identification of carcinogenic compounds. Statistical procedures require an incredibly large number of animals (megamouse experiments) in order to reliably detect low occurrences of cancer with low exposure rates (Schneiderman et al., 1976). Such large scale experiments are not feasible. Even if they were, they would still apply only to the species tested and would provide no better verification of human carcinogenicity (Weil, 1972). Alternatively a negative result in animal experiments does not necessarily prove a compound to be noncarcinogenic (Food & Drug Administration, 1971,; Maugh, 1978a) as it merely means that carcinogenic activity may still exist but is below a certain level defined by that particular experiment.

If it is assumed that observations of carcinogenesis in animals can be extrapolated to man, the next consideration is the extrapolation process. Inherent in this process is the controversial concept of thresholds. The high-dose short-term animal carcinogenicity experiments have been criticised on the basis that the high dose levels overwhelm the normal detoxification and/or repair mechanism of the organism (Stokinger, 1977; Pendygraft et al., 1979) and therefore a level may exist for a particular compound below which carcinogenesis does not occur (i.e. there is a threshold to cancer induction, the threshold being equivalent to a safe level).

Both sides of this argument have been hotly debated (Bingham, 1971; Kotin, 1976) and the merits of both approaches discussed (Weil, 1972; Truhaut, 1977; Maugh, 1978b; Fishbein, 1979b). Proponents of either conviction seem to be able to offer positive proof of their point of view, often applying the same data.

The significance of low levels of chloroform in water is a good example of this controversy (Stokinger, 1977; Tardiff, 1977) and at the present time neither point of view can be proved or disproved. It may not even be possible to unequivocally establish the existence of thresholds due to statistical limitations. For determining criteria involving human health, the more conservative approach must be adopted at the present stage of knowledge, i.e. there is no threshold or safe level for a carcinogen and the exposure to any carcinogen at any level poses a finite risk, although possibly very small. Human exposure to any carcinogen, whether a proven human carcinogen or not, should, ideally, be eliminated as no safe levels for such exposures can be shown to exist.

Various models for the carcinogenic process have been proposed to provide mathematical and statistical methods for the calculation of risk (Mantel & Schneiderman, 1975; Mantel et al., 1975; Hoel, 1976; Cornfield, 1977; NAS, 1977b; Hartley & Silken, 1977; Guess et al., 1977; Guess & Crump, 1978; Whittemore & Keller, 1978; Crump, 1979). These involve a number of assumptions and extrapolation must be made over a wide concentration range. Also many other factors, not considered in the risk estimate calculations have been shown to influence carcinogenicity (Bingham et al., 1976; Fishbein, 1979c). For example laboratory experiments have shown some compounds to inhibit (Falk et al., 1964), and some to promote (Bingham & Falk, 1969) carcinogenesis. Carcinogens also appear to be additive or synergistic (Schmanl, 1976). Inhibitors or promoters may be present in the environment and therefore affect man's response. In addition, cancer induction may be affected by a host of other biological factors (Bingham et al., 1976) including genetic factors, and the exposure of pregnant females to some carcinogens may lead to cancer in their progeny (Doll, 1977).

Another approach to the quality criteria problem is that involving testing the water itself for carcinogenicity, rather than isolating and identifying the individual components responsible. Water extracts have been shown to be carcinogenic in rats and mice (Truhaut et al., 1979), indicating the possibility of water being a cause of cancer in man. The problem of quantifying these effects, associated with the long-term testing requirements render such an approach impractical at present. However approaches utilising short-term tests for mutagenicity (and indirectly carcinogenicity) as described in the following section have been developed but the results of these tests also need to be quantified and the hazards predicted.

2.3.2.2 Epidemiological Studies

Epidemiological evidence should be the test of whether the risk estimates are accurate. This kind of evidence has been important in identifying occupational exposure to carcinogens (but not always the causative chemical) where the exposures are generally high and the cancer rates also high amongst a small and well identified group of people (Saffiotti & Wagoner, 1976). Although important, few specific chemicals have been identified as possible human carcinogens by this procedure (Tomatis, 1976) and for even fewer has the evidence demonstrated proof of carcinogenicity (Maugh, 1978c).

Studies have been made in an attempt to determine whether there have been excess cancers due to low levels of carcinogens in drinking water (Cantor, 1978; Cantor & McCabe, 1978; Wilkins et al., 1979). Some correlation between cancer rate and polluted water was found in Louisiana (De Rouen & Diem, 1975; Page, 1976;

Wade, 1977), Ohio (Kuzma et al., 1977) and in New York (Alavanja et al., 1978) in the USA. An epidemiological study of the relationship between cancer rate and trihalomethane levels has also been carried out (Cantor et al., 1978). In some cases there is correlation but it is far from convincing, and only time will tell if these studies can be refined to provide more conclusive evidence.

Thus epidemiological evidence has yet to be useful in determining whether the predictions of an increased cancer risk due to exposure to low levels of carcinogens in the environment, e.g. drinking water, are correct. Because the exposures to environmental carcinogens are low, the number of excess cancer deaths is not expected to be very large. It is therefore very difficult, if not impossible, to determine whether a measured slight increase in the cancer death rate is due to the environmental carcinogen in question or due to the host of other factors which may affect it, (Muir et al., 1976; Coulter, 1979; Peto, 1979) e.g. nearby petrochemical industries. Further, since information on the drinking water consumption of individuals is difficult to obtain (in contrast to cigarette smoking and occupational exposures) only descriptive (i.e., population-based) epidemiological studies have been possible.

2.3.3 Criteria based on mutagenic and teratogenic effects

The large number of xenobiotic chemicals in the environment to which we are now exposed includes mutagens and teratogens (Drake, 1975; Tuchmann-Duplessis, 1979; Sullivan & Barlow, 1979). Mutagens may act on the genetic material of the germ cells, thereby producing effects which are passed on to subsequent generations. Mutagens may also act on the genetic material of cells not involved in the reproductive process in which case the result may be cancer (WHO, 1972; Renner, 1979).

The more obvious effects of mutagens and teratogens are deformed progeny which may be so severe that the foetus dies before birth. However, mutagens may have more subtle effects. It has been suggested that mutagens are contributing to the decay of the human species where modern medicine contributes to the survival of mutants and to their reproduction, i.e. there is a relaxation of natural selection (Drake, 1975; Vogel, 1979).

A number of rapid screening tests for the detection of mutagens have been developed and their merits discussed (Ames et al., 1975; Stich et al., 1975; Bridges, 1976; Purchase et al., 1976; Evans, 1978; Devoret, 1979; Glatz, 1979). Ames and co-workers found that 90% of the compounds shown to be mutagenic in their particular test were also known carcinogens (McCann et al., 1975). A more recent survey showed, however, that the correlation depended on the classes of chemicals selected. Some classes had much better correlations than others. Of 465 known or suspected carcinogens 58% had been tested in the Ames test with an overall correlation of 77% (Rinkus & Legator, 1979). Thus these tests are also considered as screening tests for possible carcinogens and their usefulness in this regard has been specifically discussed (Bartsch, 1976a,b).

Work is now being directed at the quantitative aspects of these short term tests and in particular the quantitative correlation between carcinogenicity and mutagenicity. One report has been published which shows a good correlation but is based on selected compounds (Meselson & Russel, 1977). This is an area which has already generated controversy (Ashby & Styles, 1978; Ames & Hooper, 1978) and whether quantitative predictions of carcinogenicity can be made with any certainty using the results of these short-term tests remains to be seen. They should be considered as preliminary or screening tests only and a more thorough evaluation involving animal experimentation required to give further evidence of possible carcinogenicity or mutagenicity, e.g. dominant-lethal testing for determination of severe mutagenic effects (Epstein, 1973; Renner, 1979). They can be considered the first level of a multi-tier approach necessary to obtain a more objective evaluation of the risk to human health (Bridges, 1974; Flamm, 1974; Bartsch, 1976a,b; Bochkov, 1976; Waters et al., 1980).

The future of these rapid screening procedures, especially the Ames test is promising, but it has been suggested that sensitivity may be gained at the expense of selectivity (Lawley, 1979). Not only can they be used to screen particular chemicals, but foods or water samples which contain mixtures of chemicals, i.e. real samples ingested as part of daily life, can be tested (Waters, 1978; Ames, 1979). Water quality could perhaps be assessed based on the response in one of these tests.

Several reports have already been made of the responses to these tests of water samples and their concentrates (Pelon et al., 1977; Dutka & Switzer-House, 1978; Chriswell et al., 1978; Glatz et al., 1978; Loper et al., 1978; Prein et al., 1978; Denkhaus et al., 1979; Gerin-Rose et al., 1979; Commins, 1980), and wastewater concentrates (Denkhaus et al., 1979; Rappaport et al., 1979; Neeman et al., 1980). Chlorination (Cheh et al., 1980) and ozonation (Gruener, 1978) processes, and the passage of water through the distribution system (Schwartz et al., 1979) have also been shown to increase the mutagenic behaviour of water as measured in these tests.

At the present time it is difficult to determine the significance of these results in terms of water quality. If these results could be quantified and a reasonable estimate of the risk made, a useful criterion of water quality could be available. Positive results in a screening test could indicate the need for water treatment procedures to be implemented in order to reduce the level of contaminants to a point where satisfactory results were obtained.

Mutagenicity data from animal experiments can be used in deriving criteria. The extrapolation of this data to man contains the uncertainties and assumptions involved in the extrapolation of any animal data to man, i.e. whether the animal responds in the same way as man to the chemical, and the quantitative aspects of the extrapolation process. 'Safe' levels can be derived using the maximum 'no-effect' levels and safety factors via the ADI in exactly the same way as with chronic toxicity data. Mutagenesis is thus one example of a chronic toxic effect. At the present time little information is available on the mutagenic properties of chemicals. Few appear to have been adequately tested in laboratory experiments.

Criteria for teratogens can be derived in the same way as for mutagens, i.e. using maximum 'no-effect' levels and safety factors. Teratogenesis is thus another example of a chronic toxic effect which results from exposure to a chemical. The assumptions and uncertainities in the extrapolation process are as great with this class of compounds as they are for most compounds for which chronic toxicity data are available. For example, the teratogen thalidomide has widely different teratogenic properties in different species of animals (Epstein, 1974). Man appears to be by far the most sensitive animal to thalidomide, a fact that would not be evident from data obtained with other species of animals. Fortunately there is no evidence of prolonged low level exposure to teratogens in drinking water. Drinking water was a suggested cause of reduced reproduction efficiencies in mice and rabbits, but no conclusive evidence was found (Staples, 1979).

It is very difficult to show an increased incidence of birth defects due to exposure to mutagens and teratogens using epidemiological data because of the fairly high background of birth defects in the human population. Thus it is almost impossible to prove that environmental exposures to mutagens and teratogens is occurring. This is an emotional issue at present with the herbicide 2,4,5-T and illustrates the complexity of interpreting the available information.

In the manufacture of 2,4,5-T, 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) is formed as an impurity. TCDD is very toxic (Lee, 1979; Murray et al., 1979), is mutagenic in short-term mutagenicity tests (Lee, 1979), and is a powerful teratogen in laboratory animals (Murray et al., 1979). The evidence is also strongly suggestive of TCDD being carcinogenic (Van Miller et al., 1977).

Large quantities of the defoliant Agent Orange, a herbicide mixture containing 2,4,5-T, were used during the Vietnam war. TCDD was thus suggested to be involved in the birth defects in children of soldiers exposed to Agent Orange and of people in areas sprayed with Agent Orange (Thomasson, 1979). Because of the properties of TCDD, a causal relationship may exist but as yet there is no conclusive evidence. Large quantities of chemicals, including TCDD were released into the environment at Seveso, Italy in 1976 following an explosion in a chemical factory (Reggiani, 1978). No increase in birth defects was reported in the years following this event, but more recently allegations of a cover up have been reported (O'Riordan, 1979). Because of the difficulties involved in carrying out epidemiological studies the safety of 2,4,5-T is still questionable.

2.3.4 Criteria based on aesthetic quality

Certain organic compounds, namely taste and odour producing (organoleptic) organics are very important in relation to the aesthetic quality of drinking water. Taste and odour problems in water supplies have been reviewed recently (Lin, 1977; Gerber, 1979), and will not be detailed in this report.

The odour of chemicals can be quantified in terms of the threshold odour concentration, the concentration at which 50% of the observers can still detect the odour. The derivation of criteria from these data has not yet been attempted on a scientific basis. Criteria could perhaps be derived from these concentrations using an application factor. A factor of 0.01 would result in approximately 5% of the population still being able to detect the odour (Cees *et al.*, 1974). A level sufficiently low to produce a response in 5% of the population, i.e. using threshold concentrations and an application factor of 0.01, could perhaps be considered a criterion.

Although criteria for individual compounds are not available, the threshold odour concentrations are useful alternatives. These levels for some naturally occurring compounds responsible for taste and odour that are produced by the filamentous bacteria actinomycetes are summarised in Table 1. Compounds responsible for odours, e.g. those produced by actinomycetes, can also taint the flesh of fish (Gerber, 1979). However insufficient data is yet available in order to derive criteria sufficient to prevent noticeable tainting of fish flesh by these compounds.

TABLE 1. THRESHOLD ODOUR CONCENTRATIONS FOR COMPOUNDS PRODUCED BY ACTINOMYCETES

COMPOUND	THRESHOLD ODOUR CONCENTRATION (ug/L)	REFERENCE
Geosmin	0.2	Cees *et al.*, 1974
	0.1	Piet *et al.*, 1972
	0.2	Calculated from Gerber, 1979
2-methylisoborneol	0.1	" " " "
Isopropylmethoxy-pyrazine	0.002	Reported in Gerber, 1979
Mucidone	3.3	" " " "

Xenobiotic compounds reported to cause tasts and odours include phenols, pesticides, petroleum chemicals and detergents (Lin, 1976, 1977).

Phenols are one particular class of compounds for which extensive investigations into taste and odour thresholds have been carried out since taste and odour thresholds are generally very low, especially with chlorinated phenols.

Some phenols may form chlorinated derivatives during the chlorination of water for disinfection purposes and thereby enchance the taste and odour characteristics of these waters. In general the threshold taste concentrations of phenols are considerably lower than the threshold odour concentrations which are in turn considerably lower than the median lethal concentrations (LC50) for fish (Dietz & Traud 1978). Criteria for the protection of the aquatic environment based on acute toxicities and application factors would not necessarily preserve the aesthetic quality with respect to taste and odour.

Criteria or standards relating to acceptable levels of contaminants in drinking water should not be based solely on organoleptic characteristics. Criteria based on health effects must assume the greatest importance and be the first consideration. Once criteria based on health effects have been established then they can be lowered by a consideration of organoleptic properties.

2.3.5 Approaches adopted

The derivation of water quality criteria for health protection is an extremely difficult and controversial process. In the USA attempts have been made to derive criteria on a scientifc basis for a relatively large number of organic compounds. The validity of many of the assumptions employed in these derivation procedures can be criticised but they, at least, provide a guide for use in water quality management.

The wide range over which laboratory data must be extrapolated in order to determine criteria for carcinogens is one of the basic deficiencies in current procedures. Nevertheless, at the present time, there is insufficient information on the effects of low level exposures to chemicals in water supplies to provide alternative procedures for establishing more accurate criteria. The current procedures are conservative and this approach is supported where human health is implicated.

For non-carcinogens the US National Academy of Sciences derived ADI data, and then criteria assuming a daily ingestion of 2L water, that 20% of the ADI of a chemical is ingested through water, and that the average person weighs 70 kg (NAS, 1977a).

For carcinogenic compounds drinking water quality criteria can be established using risk estimation procedures. For a number of carcinogens the NAS has calculated the risk associated with ingestion of these compounds in drinking water (NAS, 1977a). These were expressed as the probability of developing cancer in a lifetime (assumed to be 70 years) from daily ingestion of 1 litre of water containing 1 ug of the carcinogen, i.e. lifetime risks per ug/L. From this an acceptable risk could be decided upon or an exposure with a latency period longer than the normal lifetime, and a water quality criterion calculated assuming an average water consumption per day. However the NAS did not proceed this far.

The NAS risk estimates were calculated in the following way from lifetime animal studies involving oral administration (NAS, 1977a). A standard human dose was converted to a physiologically equivalent dose in the animal on the basis of relative surface areas (Hoel et al., 1975). The dose was then related to the effect using the risk model of Armitage and Doll (1961), the data being fitted to this by the procedure of Guess and Crump (1976). If data for more than one animal experiment were available, then the risk for each set of data was calculated e.g. DDT (NAS, 1977a). The NAS document is the only one so far available in which calculations of risks for ingestion of carcinogens in drinking water alone are available for a number of compounds.

Slightly different procedures for criteria derivation were utilised by the EPA in the USA for priority pollutants (US EPA, 1979). Exposures were assumed to arise through drinking water and through eating fish and shellfish grown in water with a pollutant level equivalent to that of the water consumed. Estimates of the levels of the pollutant in fish and shellfish were made from bioaccumulation factors and using the estimate of the average daily intake of this food (18.7g).

The procedures used by the NAS are considered to be the most valid for risk estimation with respect to exposure to chemicals in water at the present time. Procedures used by the US EPA in which other sources of the compound apart from water are considered in the derivation process appear to be less valid. There is little reason to assume that fish and shellfish consumed originate in waters with pollutant levels similar to those of drinking water.

Taste and odour thresholds of phenols have had some influence in the derivation of existing criteria. The US criterion for phenol in domestic supplies is 1 ug/L which is based on the threshold odour concentration of 2-chlorophenol and the level of this compound causing tainting in fish (US EPA, 1976d). The Russian list of maximum permissible concentrations for harmful substances in drinking water contains organoleptic limits for over 150 compounds (Stofen, 1973). However no basis for these levels are given.

In this monograph the following procedures have been used:

a) For non-carcinogens, ADIs were calculated from maximum 'no-observed-effect' levels and safety factors. The value of the safety factor was influenced by the nature and extent of data available from toxicological studies. In cases where relatively few studies had been carried out, a more conservative safety factor was applied.

In general the maximum 'no-observed-effect' levels cited by NAS were used to calculate the ADI, although in some cases the ADI recommended by the Food and Agricultural Organisation of the United Nations (FAO) was used. The safety factors were also generally consistent with those used by NAS, but occasionally these were altered where more recent data were available.

From the ADI, a water quality criterion was derived assuming a 2L per day consumption of water for a 70 kg individual with 20% of the ADI being ingested in water. This is consistent with US authorities (NAS, 1977a; US EPA, 1976).

It should be noted, however, that WHO (1971) assumed a 2.5L per day consumption for a 70 kg individual while the NH & MRC (undated) for pesticides assumed a 2L per day consumption for a 60 kg individual with water containing 20% of the ADI.

(b) For carcinogens, risk estimates for various levels of exposure were obtained, mainly from the NAS document but occasionally from US EPA draft criteria. The latter were adjusted for exposures to water only. These calculations involved data obtained from lifetime oral exposures of laboratory animals, and the risks were calculated on the basis of a daily consumption of 1L of water containing 1 ug of the carcinogen over a lifetime of 70 years. The lifetime risk associated with a particular level of a carcinogen assuming a more typical ingestion of 2L per day of water was than calculated. A criterion was then derived assuming that the risk of one additional cancer in 10^6 persons over a 70 year lifespan is considered acceptable. This figure of 1 in 10^6 for the acceptable risk is however arbitrary, but does not appear unreasonable.

(c) Criteria based on organoleptic properties were also considered where appropriate, e.g. phenols and tabled separately in this monograph.

Although there are many uncertainties in deriving criteria the present state of our knowledge does not allow predictions to be made with any greater confidence. Until many of the problems are solved, procedures are not likely to improve. Epidemiological evidence may help evaluate the reliability of these estimates in the future.

The derived criteria, as with criteria derived for protection of the aquatic environment, should be considered as rough guides or first approximations due to the assumptions involved in the extrapolation processes. Any major variations in these assumption should also be considered when applying these criteria to actual situations. For example, criteria calculated in this way apply to lifetime exposure to low levels. The occasional exposure to higher levels has not been considered in criteria setting in the past and it is unclear how such occurrences could be treated. Criteria which apply to lifetime exposures will, of course, be adequate to protect human health in these situations.

2.4 CRITERIA FOR INDUSTRIAL USES

Criteria for organic compounds for industrial uses of water are only of minor importance in comparison with those for environmental and health protection, and little attention has been paid to this area of criterion derivation. Such criteria would obviously depend on the industry and the use for which the water is intended. Each industry could therefore require individual criteria.

Water quality criteria for most industrial uses have generally been derived on the basis of the water being used for cooling or steam generation. As a result criteria are available for some metals, anions such as carbonate and hydroxide, and determinands such as alkalinity and pH (Hart, 1974a), since these water quality characteristics are most important in relation to corrosion and formation of scale and other deposits. Organic compounds, at the levels normally found in water, do not contribute to these effects.

Water quality criteria with respect to organic compounds are important in the food and drink industries. It would appear that criteria for drinking water should be applied to water from which beverages or other consumable liquid products are made. For other applications in the food and drink industry, such as washing, there seems to be little information available as to the desirable quality of the water used. However it appears reasonable that water which contacts any food, or any surface of equipment or containers into which the food or drink comes into contact, should be of drinking water quality in order to protect human health. NH & MRC (1977) has in fact recommended that water used in food industry conform to drinking water standards.

Because of the large number of different industries, with their widely different uses of water, and the relative unimportance of water quality with respect to organic compounds in most cases, it is impractical to expand on water quality criteria for organic compounds in relation to industrial uses apart from the recommendations made with reference to the food and drink industries.

2.5 CRITERIA FOR AGRICULTURAL USES

Water for agricultural uses includes irrigation and stock watering. For irrigation the most important factor affecting quality is salinity or total dissolved salts, the two terms being generally synonymous. Other important characteristics include the metal cations and certain anions such as chloride and bicarbonate (Hart, 1974b).

Organics in water may accumulate in or on irrigated crops, resulting in unpalatable produce or contamination which could be regarded as a health hazard. This possibility appears to have received little attention, greater emphasis having been paid to the possible health effects of using irrigation water with high levels of micro-organisms, e.g. sewage effluents. Maximum limits for pesticide and herbicide residues in crops have been formulated by the NH & MRC (1978) in Australia, but these cannot be related to the levels that may have existed in irrigation water. Therefore it is not possible to derive criteria based on contamination of food by unsatisfactory irrigation water.

The most important organic compounds which could be present in irrigation water are herbicides. Water used for irrigation which is contaminated with herbicides may have detrimental effects on irrigated crops. The possibility of herbicide residues in crops is, of course, implicit in the setting of maximum residue limits for herbicides in fruit and vegetables but these residues are expected to originate from the control of weeds.

Herbicide residues in water as a result of land spraying are generally low and are also infrequently detected (Frank, 1972). However, where irrigation channels have been treated for control of aquatic or bankside weeds, a serious threat to the irrigated crop could arise. The use of herbicides for aquatic weed control and their effects on aquatic organisms have been reviewed (Brooker & Edwards, 1975; Australian Department of Primary Industry, 1979). A number of studies of the persistence of aquatic herbicides have also been carried out with a view to minimising environmental effects (e.g. Bowmer & Higgins, 1976).

Criteria for aquatic herbicides for crop protection could perhaps be based on acute toxicity data and safety factors. However effects such as reduced crop yields, reduced quality, and the acceptable residue limits in crops are important considerations. Various effects could be used as a basis for criteria derivation, however, there are many variables to be considered including the species of plant, nature and concentration of herbicide, quantity of water applied, season of year, frequency of dosing (i.e. whether a single dose or multiple doses are applied over a period of time) and methods of application. This latter consideration, i.e. whether spray or furrow irrigated is very important since many herbicides are absorbed through the foliage but are adsorbed on soil and rapidly degraded.

Other difficulties arise because some herbicides actually increase crop yields at low exposure rates, e.g. 2,4-D and sugarbeets (Bruns et al., 1973). Thus criteria for aquatic herbicides in irrigation water cannot be derived because of the difficulty in applying available exposure - effect data. Some data involving levels at which various effects of aquatic herbicides on crops for single doses were observed, have been published (Bruns et al., 1972, 1973, 1974, 1976) since the compilation by Hart (1974) and these have been summarised, where relevant, in Section 3.3 dealing with herbicides. Some detailed data on the effects of aquatic herbicides on turf grasses have also been reported (Turgeon et al., 1972; Hiltibran & Turgeon, 1977).

Salinity and certain heavy metals and ions have been considered the most important factors in determining the suitability of water for stock drinking (Hart, 1974b). Little attention has been paid to organic constituents. With pesticides it has been recommended that the criteria for domestic water supplies be applied, since some of these compounds may accumulate in animal tissues and subsequently be consumed by humans (Hart, 1974b). In the absence of any further information being available, this recommendation appears reasonable. It could perhaps be extended to other organic compounds such as PCBs which also tend to concentrate in tissues.

Algal toxins can reach high levels in stock water supplies if these become nutrient enriched and stock deaths have been attributed to these compounds (Hart, 1974b; Collins, 1978). Human deaths have probably been prevented by the unpalatable nature of such polluted waters.

Algal toxins encompass a wide range of dissimilar compounds produced by various species of algae, mainly the blue-green types. This subject has been well reviewed recently (Collins, 1978). The review includes references to information on livestock deaths due to algal toxins and to the structures and toxicities of these compounds. However, at this stage the derivation of criteria for these compounds is not feasible.

2.6 CRITERIA FOR RECREATIONAL USES

Criteria for these areas of water use were discussed by Hart (1974c) in the "Compilation of Australian Water Quality Criteria" and were mainly concerned with microbiological characteristics. Little attention was paid to organic contaminants and no maximum desirable levels for specific compounds were recommended. Since the time of that publication no further attention has been paid to these areas of water use with respect to organic contaminants and criteria. Most concern in this area has been for microbiological characteristics (WRC, 1977), for example the EEC directive on quality of bathing water (Gameson, 1979). Recently the relationship between amoebic meningitis and swimming has received much attention although no criteria for this organism have been set. A study which attempted to relate risk of exposure to contaminants during recreational use was also directed towards microbiological contamination (Dudley *et al.*, 1976).

Recreational uses covers a diversity of pastimes such as boating, fishing, swimming, skiing, etc. The variety of activities and the possible different approaches to deriving criteria for each use make it impracticable to derive criteria in a quantitative manner, particularly as organic chemicals are not likely to contribute significantly to water quality for these uses. An exception may occur in the case of chlorinated swimming pools where trihalomethanes (THMs) have been shown to be present (Weil *et al.*, 1980). The amount absorbed through such exposures may be significant in comparison with that from drinking water (Beech, 1980).

Some general statements concerning water quality criteria in these areas can, however, be made. In general it may be assumed that a water quality sufficient to adequately maintain the aquatic environment should be sufficient for recreational uses, at least with respect to organic pollutants. Therefore water quality criteria for recreational uses can be considered identical to those for the protection of the aquatic environment. Criteria for water used for fishing are more complex and relate more to the production of fish suitable for human consumption (Section 2.2).

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3. DETAILED CRITERIA FOR PESTICIDES

3.1 GENERAL

Pesticides are compounds which are applied directly to the environment for the control of pests. As a result of their usage, pesticides can find their way into waters via accidental spillage, spray drift, agricultural run-off after rain or by air transport. Pesticides may also be introduced into waters from the wastes of factories engaged in their manufacture or use. Air transport, surprisingly, is a very effective means of dissipating pesticides (Woodwell et al., 1971; Kraybill, 1977; Wells & Johnstone, 1978) and is primarily responsible for the occurrences of the more persistent organochlorine pesticides, e.g. DDT (Woodwell et al., 1971; Kraybill, 1977) even in the remote areas of the world.

The production of pesticides in general is following the strong growth pattern of other organic chemicals (Appendix) although the quantities represent only a small proportion of the total production of organic chemicals. Approximately 1500 individual pesticide compounds are estimated to be in current use throughout the world (Maugh, 1978). This contrasts markedly with the small number of insecticides (slightly in excess of 100) of which 15-18 accounted for the majority of sales in 1955 (Nicholson, 1959).

Pesticides find extensive and important uses in Australia as evidenced by estimates of their additional costs to various industries; 1.3% in the sheep and wool industry, and up to 18% in cotton growing (Snelson, 1974). However detailed information on pesticide production and usage in Australia is limited. Some information, e.g. values of pesticides produced and for some pesticide chemicals the quantities imported is available from Australia Bureau of Statistics publications but data on the quantities produced are generally confidential. In relation to pesticide usage, data concerning quantitative aspects are very scant. A survey of pesticide usage was carried out but yielded information only on a small scale due to geographical limitations (Henderson, 1972). The lack of detailed information is a matter of concern considering the toxic nature of some of these chemicals with their high risk potential. Production figures and usage patterns are important data in assessing the hazard of chemicals (Stephenson, 1977).

In Australia pesticides are subject to registration under various Acts on a state basis, which provides some control. Recent legislation in NSW is aimed at controlling all aspects of pesticide use in order to minimize hazards, e.g. labelling, sale, use, exposing of others to the pesticide, etc. (Byrnes, 1978; NSW Dept. of Agriculture, 1979). Some aspects of commercial usage are controlled by the Public Health Act in that state (Health Commission of NSW, undated).

That the misuse and abuse of pesticides can have detrimental effects both on the environment and human health is well known.

However pesticies do have positive beneficial effects as crop losses due to pests may be substantial. Estimates of 35% worldwide, with up to 50% in developing countries (Snelson, 1977), and of 15-30% worldwide with 5% in Europe and 20% in Asia (Campbell-Smith, 1972) have been made. Pesticides are also extremely useful in the control of disease. In Australia annual losses due to cattle tick and sheep blowfly have been estimated at 40-50, and 20 million dollars respectively (Bacher, 1971). DDT has been extremely useful in the control of malaria, a disease which increased dramatically in Sri Lanka after the use of DDT was banned (Boden, 1979).

Pesticides are a very necessary part of modern life. They are vital in increasing food production and fighting disease. As with many aspects of modern life there are risks associated with these benefits. Sensible use will minimise the residues in crops and the surrounding environment. The establishment of criteria for pesticides is essential to assess the importance of any contamination of water supplies and other aquatic resources. Steps have already been taken to develop an approach for determining the hazard associated with pesticide chemicals based on their toxicological, physical and chemical properties (Weber, 1977; Korte et al., 1978; Freitag et al., 1979).

Numerous reports dealing with various aspects of pesticides have been published. Although they contain information relevant to the derivation of water quality criteria, they are too numerous and detailed to discuss here. They include the toxicology of pesticides (Hayes, 1975), pesticides and microorganisms (Hill & Wright, 1978), and fate in, and effects on the environment (Matsumura et al., 1972; Edwards, 1973, 1975a; Beynon, 1975; Haque & Freed, 1975; Moriarty, 1978; Hascoet et al., 1978). Pesticides and the aquatic environment in particular have also been discussed (Bacher, 1971; Faust, 1972; Livingstone, 1977). Compilation of the toxicities of pesticides to aquatic organisms and bioaccumulation factors have been made (US EPA 1972), and some anti-pesticide publications contain useful information (Van Den Bosch, 1976, Conservation Council of Victoria, 1979).

Existing criteria and standards for pesticides in relation to water quality in Australia are relatively limited. The review by Hart (1974) gives criteria for a few compounds for various water uses. Recently the Australian Department of Health (1980) has recommended maximum residue limits for a number of pesticides in potable water. These were based on ADI data and assumed a 60 kg individual drinks 2L/day of water with 20% of the ADI in the water (NH & MRC, Pesticide and Agricultural Chemicals Subcommittee, undated).

A problem with deriving criteria for pesticides is that many are not used as the pure active ingredients but as formulations. These may be mixtures of more than one active ingredient or of ingredients with chemicals to modify their action. For example, the pyrethroid insecticides are usually formulated with the synergist piperonyl butoxide which inactivates the enzymes that normally metabolise and deactivate the active ingredient. As a result the toxicity is enhanced.

Thus criteria for the active ingredient may not accurately reflect safe levels for the formulation. Similarly toxicity data of pesticide formulations, some detailed information on which is available (Tooby et al., 1975), cannot easily be used in deriving criteria for the individual ingredients. As criteria for pesticides are derived for the pure active ingredients (except in some cases, where the active ingredient is a mixture of isomers, e.g. BHC), the use of the derived criteria should be tempered by a consideration of the effects of other components of formulations.

With regard to insecticides, the majority fall into three classes; - organochlorine, organophosphate and carbamate.

Historically the most important are the organochlorine insecticides of which DDT has been in use since the 1940's. They have found widespread use due to their low cost, relative safety for non-target higher-level organisms and resistance to degradation. This latter property, the ability to resist degradation by chemical and biological means is particularly attractive since activity is retained for some time and infrequent applications are required. However the persistence of organochlorine pesticides also results in a greater chance of contact with non-target organisms. Because their fat solubilities are much higher than their aqueous solubilities they tend to accumulate in fatty tissues and in milk. They are not readily eliminated or metabolised by organisms or animals, with a resultant accumulation. In addition to accumulation through direct contact with water, organochlorine pesticides can also bioaccumulate along the food chain. Their persistence and accumulative properties has resulted in residues being frequently detected in the environment. Although originally considered a desirable property, the stability of these chemicals has now resulted in pressure for their uses to be terminated in order to avoid harmful effects arising from accumulated residues. The persistence of organochlorine insecticides, coupled with their extensive use has resulted in the widespread occurrence of residues in air, soil, water and fauna. Certain organisms are now being used as indicators of pollution by these compounds (Goldberg et al., 1976; Phillips, 1978).

Although widespread occurrences of residues in the environment have been well documented only in a few cases has it been demonstrated with any certainty that the presence of residues at low levels has resulted in adverse effects. Impairment of reproduction is the main effect that has been demonstrated as arising from organochlorine insecticide residues, especially DDT. The decline in predatory birds due to thinning and breakage of egg shells has been shown to be due to organochlorine residues (Cooke, 1973). This effect has also been shown to be occurring in Australia (Olsen & Olsen, 1979). Reproduction failures in fish have similarly been attributed to organochlorine residues (Johnson & Ball, 1972).

Reports show that the organochlorine insecticides can be associated with particulate matter in water (Weber, 1972; Picer et al., 1977). It has also been shown that sediments and soils can act as sources of organochlorine insecticides long after the original source has been removed (Young et al., 1977; Yamato et al., 1980). Comparison of levels determined in water with derived criteria in order to assess safety may give misleading results unless the contribution from sediments is also considered.

Concern for the persistence and accumulation of organochlorine insecticides has resulted in their usage being severely curtailed or even banned in developed countries. More recent findings that some are carcinogenic in animals (IARC, 1974a) has added further pressure for their withdrawal from use. However, as far as developing countries are concerned organochlorine pesticide use is continuing since the benefits still far outweigh the risks. As a result, production of these insecticides is not substantially decreasing in countries such as the USA, because increasing quantities are being exported (Edwards, 1975b). Data from Iran (Sodergren et al., 1978) and India (Edwards, 1975b) reflect this trend.

In contrast the organophosphate and carbamate insecticides tend to be labile. They generally degrade readily and do not accumulate. They also tend to be less toxic to fish than the organochlorines, but their very high mammalian toxicity results in a high health risk both to man and animals in the immediate environment during application. The low toxicities or organophosphate insecticides to fish in comparison with the organochlorine insecticides is related to their less ready absorption and accumulation from water (Yang & Sun, 1977).

Organochlorine insecticides act on the central nervous system. They are cumulative poisons, several small doses over a period of time can therefore constitute a lethal dose. Both organophosphate and carbamate insecticides act on the cholinesterase enzymes, the enzymes involved in the transmittance of nerve impulses. Organophosphates inhibit these enzymes by phosphorylation. Acetylcholine subsequently accumulates, the nervous system is disrupted and death can ensue. The effects of exposures are mostly reversible with sublethal effects in humans including eye and vision disturbances (Plestina & Piukovic-Plestina, 1978). Environmental exposures are considered not to be sufficient to cause these problems in man (Plestina & Piukovic-Plestina, 1978). They are also cumulative poisons if enzyme levels do not have the opportunity to recover between exposures.

The mechanism of action of the various classes of insecticides on insects has been reviewed (e.g. Casida, 1973) as has their toxicology (e.g. Hayes, 1975). Similarly an evaluation of organochlorine insecticides for the setting of ADI levels, important in deriving drinking water quality criteria, has also been reviewed (Vettorazzi, 1975).

3.2 ORGANOCHLORINE INSECTICIDES

3.2.1 Aldrin (HHDN)

General

Aldrin is one of the group of cyclodiene insecticides, a group which comprises the majority of the organochlorine insecticides and which are formed by the Diels-Alder reaction of hexachlorocyclopentadiene with various olefinic type compounds. The other cyclodienes are chlordane, dieldrin (the epoxide of aldrin), endosulfan, endrin and heptachlor.

Aldrin is still used extensively worldwide for the control of insects (Snelson, 1977), but as with most of the other organochlorine insecticides, its use is being curtailed due to its persistence.

It is mainly used for the control of soil insects in crops, with a significant quantity used for control of termites (IARC, 1974b). In the USA concern regarding the undesirable properties of aldrin has resulted in only 40% of the peak of 1966 being used in 1972 (Edwards, 1975b), with a ban being imposed in 1974 (NAS, 1977). It is also banned in other countries (IARC, 1974b), including Finland (Hattula et al., 1978a).

In Australia aldrin was used extensively in the dipping of sheep and for control of sheep blowfly strike. However, resultant unacceptable residue levels in the fat of treated animals has resulted in its use being discouraged for these purposes. It is still being widely used for control of soil pests in crops including sugarcane, corn, potatoes and tobacco and in termite control in buildings (Pesticides Section, Australian Department of Primary Industry, 1975). The quantities used in Australia and the usage trends are not readily available.

Residues in the aquatic environment

Aldrin residues have been widely found in the aquatic environment. The determinations have been infrequent in comparison with dielrin into which aldrin is rapidly converted, either photochemically in the environment, or biologically in soils or animals.

Dieldrin residues in water, sediments and organisms of the Des Moines River, Iowa, were associated with the heavy use of aldrin for the control of soil insects in neighbouring cropland. Some levels in catfish exceeded the maximum level permitted in human food (Kellogg & Bulkley, 1976). More recent determinations are shown in a Table 2.

TABLE 2. RECENT DETERMINATIONS OF ALDRIN IN THE AQUATIC ENVIRONMENT

LOCATION	DETERMINED IN	REFERENCE
CANADA	water	McNeil et al., 1977 Gummer, 1978
FINLAND	fish	Hattula et al., 1978a
	plankton	Sarkka et al., 1978a
	aquatic plants	Sarkka et al., 1978b
IRAN (Caspian Sea)	fish	Hashemy-Tonkabony & Langaroodi, 1976
THE NETHERLANDS	water	Wegman & Greve, 1978
USA (Mississippi)	water	Rihan et al., 1978
(South Carolina)	water	Sandhu et al., 1978
MEDITERRANEAN SEA	various organisms	Amico et al., 1979

Aquatic toxicology and criterion for the protection of the aquatic environment

The US EPA has reviewed most of the available data on the effects of aldrin and dieldrin on aquatic organisms (acute and chronic toxicity and bioaccumulation) in its 1976 criteria document (US EPA, 1976a) and more recently in a document relating to proposed criteria changes (US EPA, 1978a). Because of the relative ease of conversion of aldrin to dieldrin the properties of dieldrin were considered in these procedures. Since dieldrin is as toxic as or slightly more so than aldrin for most organisms, the criterion was based on the presence of either dieldrin or aldrin or the sum of both. The 1976 criterion calculated (0.003 ug/L) was based on a consideration of bioaccumulation factors and acceptable levels in fish for human consumption. As discussed in Section 2.5 this is not considered relevant to the protection of the aquatic environment. The more recent proposed criterion of the US EPA is 0.0019 ug/L as a 24 hour average and 1.2 ug/L not to be exceeded at any time for freshwater aquatic life. The corresponding values for saltwater aquatic life are 0.0069 and 0.16 ug/L respectively (US EPA 1978a, 1979a).

Based on the toxicity to the most sensitive species tested with aldrin or dieldrin (30 day LC50 of 0.2 ug/L for the stonefly Acroneuria pacifica (Jensen & Gaufin, 1966)) and an application factor of 0.01 for compounds which bioaccumulate, a more relevant criterion for the protection of the aquatic environment for aldrin/dieldrin of 0.002 ug/L is, however, derived in this report.

A consideration of tabulated toxicity data for aldrin and dieldrin (US EPA, 1972) shows aldrin to be somewhat less toxic than dieldrin in most cases. Although the criterion based on dieldrin will therefore also suffice for aldrin, it is possible to derive a separate criterion for aldrin. The most sensitive species tested with aldrin is the stonefly Pteronarcys californica which has a 96 hour LC50 of 1.3 ug/L (Sanders & Cope, 1968). (The 96 hour LC50 has also been independently determined as 180 ug/L (Jensen & Gaufin, 1966), indicating the variability which can be obtained in bioassays due to the number of indeterminable variables). Thus based on the toxicity datum of 1.3 ug/L and using an application factor of 0.01 a derived criterion of 0.01 ug/L for aldrin alone is obtained for the protection of the aquatic environment. However, care must be exercised in applying this criterion because of the ease by which aldrin is converted to the more toxic dieldrin.

The data available are deficient with respect to chronic toxicity, especially for studies involving reproduction. Highly desirable data such as MATC and hence more objective and less empirical application factors are not available. There is, therefore, no check on the appropriateness of the application factor of 0.01 chosen for the derivation of the criterion.

Bioaccumulation and the production of fish suitable for human consumption

Since it is dieldrin, rather than aldrin per se which accumulates in organisms (aldrin is rapidly metabolised to dieldrin (IARC, 1974b)), a criterion for aldrin which will provide acceptable levels in fish for human consumption should take this into account. Assuming aldrin accumulates to the same extent as dieldrin (a consideration of the data on bioaccumulation of aldrin and dieldrin shows the bioaccumulation factors to be of the same order of magnitude (US EPA, 1972)), then the criterion for dieldrin of 0.002 ug/L for the production of fish suitable for human consumption should suffice for aldrin.

Criterion for the protection of human health

Studies of the carcinogenicity of aldrin to animals have been few and the results inconclusive (IARC, 1974b). Similarly there is no adequate epidemiological data available to indicate that aldrin is carcinogenic in man. Dieldrin is carcinogenic in mice and may therefore be a human carcinogen. Thus aldrin may also be considered a possible human carcinogen (IARC, 1974b) and the criterion relating to drinking water quality for aldrin can be calculated on the basis of the carcinogenicity data for dieldrin. Therefore the derived drinking water quality criterion for aldrin can be considered to be the same as for dieldrin, i.e. 0.002 ug/L (see section on Dieldrin)

SUMMARY OF PROPOSED CRITERIA FOR ALDRIN

Protection of the aquatic environment - 0.01 ug/L for aldrin alone. The ready conversion of aldrin to the more toxic dieldrin must be considered in applying this criterion.

Production of fish suitable for human consumption - 0.002 ug/L.
Protection of human health (drinking water quality) - 0.002 ug/L.

3.2.2 BHC and Lindane

General

Free radical chlorination of benzene produces a mixture of isomers of 1,2,3,4,5,6 - hexachlorocyclohexane. The mixture of isomers is most often referred to by the trivial name of benzene hexachloride (hence BHC). It is also called HCH (hexachlorocyclohexane). The concentration of the various isomers varies depending on the conditions of manufacture; typical figures are α -isomer, 55-70%; β -isomer, 6-8%; γ -isomers, 10-18%; δ -isomer, 3-4%; ε -isomers, very small amounts (IARC, 1974c). Although the mixture possesses insecticidal properties and is used for this purpose, it is the γ -isomer (which is called lindane, gammexane, γ -BHC or γ -HCH), which is primarily responsible for insecticidal activity. However the α - and β - isomers are more toxic for mammals in chronic studies (NAS, 1977b). Since all isomers are persistent and accumulate, environmental problems can be greatly minimised by the application of lindane rather than BHC since for a given insecticidal activity, a much smaller quantity of lindane is required. Lindane is prepared from BHC by selective crystallisation.

BHC and lindane have found widespread worldwide use as insecticides in crop insect control due to their broad spectrum of activity and low mammalian toxicity (IARC, 1974c; Pesticides Section, Australian Department of Primary Industry, 1975). Lindane has also found use as a seed treatment, for control of pests on livestock and in agricultural premises and in public health pest control (Reuber, 1979).

In order to minimise environmental problems, BHC is being replaced by lindane in developed countries. BHC production in the USA fell from 53x10^{6} kg in 1951 to 8x10^{6} kg in 1963 (IARC, 1974c). BHC is voluntarily banned in the USA and its place taken by lindane (US EPA, 1978b) but is still used extensively in under-developed countries, e.g. on rice, due to its low cost (Snelson, 1977). In these countries, where food is scarce, the need for food must be balanced against pesticide residues and associated risks. Its use is banned or restricted in several countries (IARC, 1974c). Lindane also has undesirable properties and its use is being curtailed. In the USA as of 1973, it was still approved for use on a number of crops and for other uses such as control of pests on livestock. Its use too is now banned or greatly restricted in some countries (IARC, 1974c).

The uses of these insecticides in Australia are similar to those in other developed countries (Pesticides Section, Australian Department of Primary Industry, 1975). BHC usage is restricted to crop insect control. Some idea of the usage trends for BHC and lindane in Australia can be gauged from import statistics, these being two of the chemicals for which some data are available. Surprisingly lindane imports have fallen from a high of 177 509 kg in 1969-70 to 44 560 kg in 1977-78 while BHC imports have remained fairly steady (high - 903 017 kg in 1972-73; low - 410 661 kg in 1967-68; 637 043 kg in 1977-78: Data from "Overseas Trade, Part 1. Exports and Imports", Australian Bureau of Statistics, Canberra). This data does not reflect the reported trend of replacement of BHC by lindane, however locally manufactured lindane may now be used more extensively in Australia.

As with most organochlorine insecticides all isomers of BHC accumulate. However their bioaccumulation factors are low compared with other organochlorine insecticides and they are eliminated fairly rapidly on termination of exposure. This applies particularly to lindane. Therefore in comparison with other organochlorine insecticides such as DDT and dieldrin, residues tend to be less of a problem.

Residues in the aquatic environment

Residues of BHC isomers are frequently detected in the environment even though they are less persistent and more readily eliminated from organisms in comparison with those of some other organochlorine pesticides. This high frequency of detection of residues reflects their widespread use. The α - and γ - isomers are most frequently detected. BHC contains predominantly the α - isomer but conversion of lindane to the α - isomer in the environment has also been reported (Benezet & Matsumura, 1973).

The reported occurrences of residues are too numerous to document in this report. However some significant occurrences are described. The α - and γ -isomers have been found in rainwater (Wells & Johnstone, 1977), indicating that significant aerial transport of these insecticides may be occurring. The α - and γ -isomers have also been detected in surface waters (Gummer, 1978), and drinking water (McNeil *et al.*, 1977) in Canada. Data from Europe suggest that BHC is *a* significant environmental contaminant in this area. The α -, β - and γ -isomers have been found in water and fish from the Danube. The average concentration of the γ -isomer over the period 1971-74 was 0.12 ug/L (Sackmauerova *et al.*, 1977). The Rhine was found to contain levels up to 0.24 ug/L which is greater than levels harmful to *Daphnia* (Wegman & Greve, 1978). In 1974 levels of the α - and γ -isomers were in excess of LC50 levels for a number of organisms (Poels *et al.*, 1978). Contamination of water by BHC of domestic origin was found in Northern Ireland. The levels found were also demonstrated to be harmful to local aquatic organisms (Harper *et al.*, 1977). The presence of the β -isomer in herring gull eggs and fish from Lake Ontario (Norstrom *et al.*, 1978), reflects the pollution of this area by a number of pesticides.

Residues of the δ -isomer found in hatchery trout were a result of contaminated feed (Parejko & Wu, 1977). Other more recent determinations of BHC isomers in the aquatic environment are shown in Table 3.

TABLE 3. RECENT DETERMINATIONS OF BHC ISOMERS IN THE AQUATIC ENVIRONMENT

ISOMERS	LOCATION	DETERMINED IN	REFERENCE
$\alpha + \gamma$	GERMANY	Fish	Goerke et al., 1979
γ	IRAN (Caspian Sea)	Fish	Hashemy-Tonkabony & Langaroodi, 1976; Sodergren et al., 1978
γ	USA (Hawaii)	Water, sediments, oysters.	Tanita et al., 1976
γ	USA (Idaho)	Fish	Perry, 1979
$\alpha + \gamma$	Ligurian Sea	Organisms	Contardi et al., 1979
$\alpha + \beta + \gamma$	Mediterranean Sea	Organisms	Amico et al., 1979

Lindane residues have been determined in the aquatic environment of Australia. A survey of corals, fish and molluscs of the Great Barrier Reef for organochlorine pesticides revealed lindane to be by far the most common pollutant from this group of chemicals. The lindane residues appeared to be associated with agricultural usage on sugar cane (Olafson, 1978). Lindane residues were found in some birds in the Northern Territory but were absent from fish and other aquatic animals (Best, 1973). Aquatic animals from Tasmania were found to contain lindane (Bloom et al., 1979). BHC and lindane were also determined in Australian fish (Neuhaus et al., 1973). Trace levels of lindane (0.01 mg/kg) were found in oysters from the Tamar River, Tasmania (Sumner, 1978). Lindane was not found in fish (Solly, & Shanks, 1969) nor birds or mammals associated with the aquatic environment (Lock & Solly, 1976) in New Zealand, however. Recent work has shown that BHC, even though it is not as persistent as some other organochlorine insecticides, is still retained in soil for many years (Jackson et al., 1974). Contaminated soils were shown to act as sources of water pollution by BHC for several years after pesticide applications had ceased (Yamato et al., 1980).

Aquatic toxicology and criteria for the protection of the aquatic environment

Since the individual isomers have different toxicities criteria should be derived for each isomer. The US EPA (1976b) reviewed the data for lindane in their 1976 criteria document, and derived criteria for the protection of aquatic life in both fresh and marine waters. Criteria were based on the acute toxicities for the most sensitive species and application factors of 0.01. Several more toxicity studies have been reported since that time. A chronic lifecycle study involving reproduction showed midges to be the most sensitive of the organisms tested with an MATC of 2.2-5.0 ug/L and a derived application factor of 0.01-0.024 (Macek et al., 1976). Thus an application factor of 0.01 appears quite appropriate. The most sensitive organism for which data have now been reported is the shrimp, Penaeus duorarum, with a 96 hour LC50 of 0.17 ug/L (Schimmel et al., 1977a). Thus using this toxicity value and an application factor of 0.01, a criterion for the protection of the aquatic environment of 0.001 ug/L is derived for lindane.

More recently, the US EPA (1978b, 1979a) has formulated draft criteria for lindane. The criterion for the protection of freshwater aquatic life was 0.21 ug/L as a 24 hour average and 2.9 ug/L not to be exceeded at any time. For saltwater aquatic life the data were considered inadequate for criterion derivation. The value of 0.21 ug/L is greater than the 96 hour LC50 of 0.17 ug/L for Penaeus duorarum (a saltwater organism) indicating it is not suitable as a criterion for the protection of the aquatic environment in general. The criterion calculated in this document (0.001 ug/L) is considered more appropriate.

For BHC a 96 hour LC50 of 0.34 ug/L has been reported for Penaeus duorarum (Schimmel et al., 1977a). Using an application factor of 0.1 a criterion of 0.003 ug/L can therefore be derived for BHC.

Toxicity studies with the other isomers of BHC and aquatic organisms are few. The 96 hour LC50 of the α-isomer for Artemia salina was reported as 0.5 mg/L (Canton et al., 1978). Daphnia were more sensitive with an EC10 level (the level which caused a 10% inhibition of reproduction) of 5 ug/L (Canton et al., 1975). Based on this datum, and an application factor of 0.01, a criterion of 0.05 ug/L is derived for α-BHC. No recent data were found for the β-isomer.

Bioaccumulation and the production of fish suitable for human consumption

Bioaccumulation studies have been carried out on BHC (Schimmel et al., 1977a) and its α-(Canton et al., 1975, 1977, 1978; Sugiura et al., 1979), β-(Sugiura et al., 1979) and γ-isomers (Macek et al., 1979a, Schimmel et al., 1977a; Sugiura et al., 1979; Hansen, 1980). These studies indicated relatively low bioaccumulation factors with rapid elimination of residues on cessation of exposure where the latter studies were carried out.

It is therefore considered that residues in fish grown in water containing BHC and its isomers are not likely to approach anywhere near maximum residue limits. The Australian maximum residue limit for lindane in fish is 1 mg/kg with 0.3 mg/kg for BHC in meat fat (NH & MRC, 1978). The derived criteria for the protection of the aquatic environment for BHC and lindane of 0.003 and 0.001 ug/L respectively, allows for bioaccumulation factors of 100 000 and 1 000 000 respectively to meet these maximum residue limits. The bioaccumulation factors so far determined for fish fall well below these figures. Consequently criteria relating to this water use are not derived, the criteria for the protection of the aquatic environment will more than suffice.

Criterion for the protection of human health

Several discussions of the carcinogenicity of BHC and its isomers (IARC 1974c; NAS, 1977b; US EPA, 1978b), and in particular lindane (Reuber, 1979a), are available. The α, β, γ and $\delta + \epsilon$ isomers are carcinogenic in mice and therefore must be considered possible human carcinogens. The NAS (1977b) has reviewed the carcinogenicity data and calculated the lifetime risk for exposure to the BHC isomers in water as shown in Table 4.

TABLE 4. LIFETIME RISK OF DEVELOPING CANCER FROM EXPOSURE TO BHC ISOMERS IN WATER

COMPOUND	LIFETIME RISK (per ug/litre per litre of water consumed daily)	UPPER 95% CONFIDENCE LIMITS
α-BHC	-	$0.7 - 1.5 \times 10^{-6}$
β-BHC	$1.1 - 3.5 \times 10^{-6}$	$2.5 - 5.8 \times 10^{-6}$
Lindane	$3.3 - 8.1 \times 10^{-6}$	$5.6 - 13 \times 10^{-6}$

Taking the upper extreme and assuming a 2 litre/day consumption with an acceptable risk of 1×10^{-6} the criteria in Table 5 are derived.

TABLE 5. CRITERIA FOR BHC ISOMERS BASED ON NAS RISK ESTIMATES, A 2 LITRE/DAY CONSUMPTION OF WATER AND AN ACCEPTABLE RISK OF 1×10^{-6}

COMPOUND	CRITERION FOR PROTECTION OF HUMAN HEALTH (ug/L)
α-BHC	0.3
β-BHC	0.08
Lindane	0.03

The US EPA has recently proposed draft criteria for the protection of human health for α-, β- and γ-BHC and technical BHC based on the carcinogenicity data. The calculations assumed exposure through ingestion of water (2L/day) and consumption of fish and shellfish grown in that water (18.7 g/day). Of the exposure, 12% was calculated to arise from ingestion of drinking water, the remaining 88% coming from the fish and shellfish. The criteria corresponding to a risk of $1x10^{-6}$ are shown in Table 6.

TABLE 6. CRITERIA FOR BHC AND ISOMERS CORRESPONDING TO A RISK OF 1×10^{-6} AS CALCULATED BY US EPA (1978b, 1979a)

COMPOUND	CRITERIA (ng/L) Exposure through drinking water, fish and shellfish	 Exposure through water alone
α-BHC	1.6	13
β-BHC	2.8	23
γ-BHC	5.4	45
BHC	2.1	17

The criteria based on NAS risk estimates are however proposed throughout this document.

SUMMARY OF PROPOSED CRITERIA FOR BHC AND ISOMERS

Protection of the aquatic environment

α-BHC	0.05 ug/L
β-BHC	-
Lindane	0.001 ug/L
BHC	0.003 ug/L

Production fish suitable for human consumption - criteria for the protection of the aquatic environment will suffice.

Protection of human health

α-BHC	0.3 ug/L
β-BHC	0.08 ug/L
Lindane	0.03 ug/L

3.2.3 Camphechlor (Toxaphene)

General

The insecticide camphechlor is an extremely complex mixture of compounds formed by the chlorination of camphene. It contains 67-69% chlorine, and 26 compounds constitute approximately 40% of the product; the total number of compounds is over 177 (Holmstead et al., 1974). A number of components have been isolated and their toxicities determined (Casida et al., 1974; Holmstead et al., 1974; Khalifa et al., 1974; Turner et al., 1975). Ideally criteria should be derived for each component but the complexity of composition makes this impractical. It has also been suggested that the composition of camphechlor may vary depending on the method of manufacture (Vettorazzi, 1975).

Camphechlor is an extensively used insecticide. It has been reported as the most widely used insecticide in the USA and in 1971 it accounted for over 50% of the total organochlorine insecticides applied to crops in that country. Even greater quantities may be used in the future due to the severe restrictions on other organochlorine insecticides (Schimmel et al., 1977b). The uses of camphechlor include control of pests on crops and ectoparasites on animals in tropical countries. When combined with DDT it is most useful for control of insects on cotton (Snelson, 1977) although in the USA methylparathion has replaced DDT in camphechlor-DDT mixtures (Casida et al., 1974). In Australia camphechlor is used on cotton, tobacco, maize and tomatoes, usually in conjunction with DDT. It was also formerly used as a cattle dip (Pesticides Section, Australian Department of Primary Industry, 1975).

Residues are persistent in soil (NAS, 1977c), but in animals are metabolised and eliminated fairly rapidly in comparison with other organochlorine pesticides on termination of exposure (Vettorazzi, 1975). It is therefore not often found in animals. The analysis for camphechlor at low levels is difficult because of the number of compounds present and this may also account for the low frequency of detection despite its widespread use.

Residues in the aquatic environment

Some occurrences of camphechlor in the environment have been reported and include catfish (Crocket et al., 1975; Plumb & Richburg, 1977), and rainwater (Munson, 1976) from the USA.

Aquatic toxicology and criteria for the protection of the aquatic environment

The US EPA has reviewed most of the aquatic toxicity data on camphechlor in their establishment of water quality criteria for the protection of aquatic life (US EPA, 1976c), and more recently in a draft criteria document (US EPA, 1978c). The most sensitive organism for which data have been reported is the pinfish, *Lagodon rhomboides* with a 96 hour LC50 of 0.5 ug/L (Schimmel et al., 1977b). Based on this datum and an application factor of 0.01, a criterion of 0.005 ug/L was derived in the 1976 document. In the more recent draft criteria documents, a criterion of 0.007 ug/L as a 24 hour average and 0.47 ug/L not to be exceeded at any time was proposed for the protection of freshwater aquatic life. The corresponding values for saltwater aquatic life were 0.019 and 0.12 ug/L respectively (US EPA, 1978c, 1979c).

A long term study of the effect of camphechlor on brook trout established the 96 hour LC50 as 10.8 ug/L with a no-effect level of <39 ng/L for this species (Mayer et al., 1975). This data indicates an application factor of less than 0.004. On this basis it is considered that an application factor of 0.01 is too large and one of 0.005 is more appropriate. Applying this to the most sensitive species a criterion of 0.002 ug/L is derived for the protection of the aquatic environment in this report.

The US EPA has also indicated that 7 mg/kg of camphechlor in the food of mallards does not impair reproduction. In consideration of this, and the maximum bioaccumulation factor of 76 000 now reported for camphechlor in fish (Mayer et al., 1975), a criterion of 0.002 ug/L would also protect bird life. This is rather a gross simplication of the overall picture since available data show that not all the compounds in camphechlor accumulate to the same extent (Mayer et al., 1975). The more highly chlorinated are preferentially stored while the less chlorinated are more rapidly eliminated. It has also been shown that the more toxic components degrade faster in the environment (Lee et al., 1977).

Bioaccumulation and the production of fish suitable for human consumption

No maximum residue limit for camphechlor in fish for human consumption is stipulated in Australia. A criterion of 0.002 ug/L with a bioaccumulation factor of 76 000 would provide for a maximum residue limit of 0.15 mg/kg. This is well below the 3 mg/kg stipulated for the few crops for which maximum residue limits have been set (NH & MRC, 1978), and should provide for the production of fish fit for human consumption.

Criterion for the protection of human health

In relation to drinking water, several criteria for camphechlor have been reported. The 1976 US EPA drinking water criterion was 5 ug/L (US EPA, 1976c). Based on health considerations a criterion of 0.024 mg/L was derived. This was based on the highest level found to have minimal or no long-term effects on dogs, the most sensitive animal tested (10.0 mg/kg), and a 70 kg individual consuming 2 litres/day of water and adopting a safety factor of 500. However, as organoleptic effects had been reported at a much lower level (5 ug/L), this health based criterion of 0.024 mg/L was lowered to a final criterion of 5 ug/L.

The NAS (1977c) derived a drinking water quality criterion of 8.6 ug/L. This was based on a highest no-adverse-effect level of 1.25 mg/kg in the rat, a 70 kg individual consuming 2 litres/day of water and a safety factor of 1 000. At the time that both of these criteria were derived a number of chronic toxicity studies had been performed with camphechlor and various animals. A number of toxic effects had been observed, mainly liver damage, but no carcinogenic properties were evident. Some human toxicity studies had been performed but no adverse effects were observed (NAS 1977c).

More recently carcinogenic properties of camphechlor have been reported and the US EPA has proposed draft criteria based on the carcinogenicity data (US EPA, 1978c, 1979c). A criterion of 0.05 ng/L corresponding to a risk of 1x10^{-6} was derived assuming exposure through consumption of 2 litres of water and 18.7 g fish and shellfish per day. 99% of the exposure originates from the fish and shellfish and only 1% from the water. As discussed earlier, consideration of exposures through routes other than water is not considered justifiable in deriving criteria. Thus, based on exposure through water alone, a drinking water quality criterion of 8 ng/L is derived. The drinking water quality criterion proposed is that derived from carcinogenicity data, assuming an acceptable risk of 1x10^{-6} and exposure through drinking water only, i.e. 8 ng/L.

SUMMARY OF PROPOSED CRITERIA FOR CAMPHECHLOR

Protection of the aquatic environment (including production of fish suitable for human consumption) - 0.002 ug/L.

Protection of human health (drinking water quality) - 0.008 ug/L.

3.2.4 Chlordane

General

Chlordane is a cyclodiene insecticide, formed by the Diels-Alder reaction of hexachlorocyclopentadiene and cyclopentadiene followed by further chlorination. Pure chlordane consists of approximately 70% of the *cis-* or α - and 25% of the *trans-* or γ -isomers and is now available from improved manufacturing procedures (Martin & Worthing, 1977a). Technical chlordane, the basis of the earlier and some current insecticidal formulations containing chlordane, contains the α - and γ -isomers and at least 45 other compounds (Sovocool *et al.*, 1977), including a significant percentage of heptachlor which is itself toxic and carcinogenic. The proportion of the chlordane isomers in technical chlordane is not easy to determine as some reports give the proportion of the α - and γ -isomers as 60-75% (NAS, 1977d, Martin & Worthing, 1977a). The manufacturers give the proportion of the α - and γ -isomers as approximately 20% of each, but studies have been cited which suggest that the individual isomers may constitute only half this figure (Sovocool *et al.*, 1977). The mixture of isomers, variable composition and toxic impurities make it difficult to interpret some data where technical chlordane has been used.

Chlordane is a widely used insecticide for the control of soil pests, termites and ants (Snelson, 1977). In Japan it is also used for wood protection against ants and beetles (Miyazaki et al., 1980). In Australia the main uses are essentially the same (Pesticides Section, Australian Department of Primary Industry, 1975), although with pentachlorophenol it finds some use as a wood preservative (Department of Agriculture and Fisheries, South Australia 1978). As with most of the other organochlorine insecticides its use in being curtailed. It is now banned on agricultural crops in the USA (NAS, 1977d), and in 1977-78 only 67% of the peak of 1973-74 (115 000 kg) was imported into Australia (data from, "Overseas Trade, Part 1, Imports and Exports Australian Bureau of Statistics, Canberra"). As with all pesticides, data on the quantities manufactured in Australia are confidential so that the quantities used and the trends in usage cannot be ascertained.

Chlordane is typical of the organochlorine insecticides in that it is persistent, accumulates and is biomagnified up the food chain.

Residues in the aquatic environment

Chlordane has been widely detected in the aquatic environment. It has also been recognised recently that oxychlordane, one of the metabolites of chlordane, is more toxic (NAS, 1977d) and is the major terminal residue (Kearney, 1976; NAS, 1977d). Oxychlordane has also been determined in animals of the aquatic environment, e.g. herring gull eggs and fish from Lake Ontario (Norstrom et al., 1978). More recent reports of chlordane residues in the aquatic environment are shown in Table 7.

TABLE 7. RECENT DETERMINATIONS OF CHLORDANE IN THE AQUATIC ENVIRONMENT

LOCATION	DETERMINED IN	REFERENCE
CANADA	Water	McNeil et al., 1977
JAPAN (Tokyo Bay)	Fish	Miyazaki et al., 1980
MEXICO (Gulf of Mexico)	Oysters	Rosales et al, 1979
USA (Hawaii)	Water, sediment, oysters	Tanita et al., 1976
Great Lakes	Fish (salmon)	Moccia et al., 1978

Chlordane has also been determined in a municipal water supply in the USA where it was responsible for gross contamination (Harrington et al., 1978).

Aquatic toxicology and criterion for the protection of the aquatic environment

Most of the available data on the toxicity of chlordane to aquatic animals has been reviewed by the US EPA in its criteria documents (US EPA, 1976d, 1978d). In the 1976 criteria document, a criterion of 0.004 ug/L was derived for the protection of marine aquatic life based on the acute toxicity to the most sensitive species tested, the pink shrimp *Penaeus duorarum* (96 hour LC50 of 0.4 ug/L) and an application factor of 0.01. A criterion of 0.045 ug/L was calculated for the protection of freshwater aquatic life but was lowered to 0.01 ug/L due to uncertainity as to the true chlordane concentrations present in the toxicity test solutions.

In more recent draft criteria documents (US EPA, 1978d, 1979b) a criterion of 0.024 ug/L as a 24 hour average and 0.36 ug/L not to be exceeded at any time was derived for the protection of freshwater aquatic life. The corresponding values for saltwater aquatic life were 0.0091 and 0.18 ug/L respectively. These values were calculated using the EPA formalised procedures, and as discussed earlier are considered to have no more justification than more simplistic procedures. Thus it is suggested that the criterion of 0.004 ug/L be retained for the protection of the aquatic environment as it is based on the application factor of 0.01 used consistently in this document for compounds which accumulate.

A number of more recent studies of the sublethal effects of chlordane on aquatic organisms has been reported. Sublethal effects were observed at levels higher than the established criteria but in most cases no-effect levels cannot be determined. However the criterion will protect against these sublethal effects. Further acute toxicity studies have also been performed e.g. on bluegills (*Lepomis macrochirus*) and *Daphnia magna* (Randall *et al.*, 1979), *Saccobranchus fossilis* (Verma *et al.*, 1978), and sheepshead minnows (*Cyprinodon variegatus*) (Parrish et al, 1978). In all cases the organisms tested were less sensitive than those on which the criteria had already been based. The study with sheepshead minnows (Parrish *et al.*, 1978) also included a lifecycle test involving reproduction, however the purity of the chlordane used is open to doubt from the data given. From this study an MATC of between 0.5 and 0.8 ug/L was obtained with a derived application factor of between 0.04 and 0.06. This is in reasonable agreement with the empirical application factor of 0.01, indicating the validity of this selection. The study by Cardwell (1977) also produced application factors in better agreement for fish, although those for invertebrates were slightly higher.

Bioaccumulation and the production of fish suitable for human consumption

A maximum residue limit for chlordane in fish is not listed in Australia, but a limit of 0.05 mg/kg is stipulated for poultry fat (NH & MRC, 1978). Extending this to fish, a criterion of 0.004 ug/L allows for a bioaccumulation factor of 12 500.

Bioaccumulation factors of nearly 20 000 have been reported for fish, with higher factors for algae (Sanborn et al., 1976), Daphnia (Moore et al., 1977), and snails (Sanborn et al., 1977). The possibility of fish in a natural environment concentrating chlordane up to 100 000 times the water concentration has been suggested (US EPA, 1976d). Assuming a bioaccumulation factor of 100 000 is possible, a criterion of 0.0005 ug/L is derived which will provide for the production of fish to meet a maximum residue limit of 0.05 mg/kg.

Criterion for the protection of human health

A number of animal carcinogenicity experiments have been carried out with both technical and high purity grades of chlordane and the results were unequivocally positive for mice (Epstein, 1976). Chlordane can therefore be considered a possible carcinogen for man Chlordane residues have been detected in the adipose tissue of man (Atallah et al., 1977) and poisoning by chlordane in Australia has been reported (Barnes, 1967). Technical chlordane is also positive in the Ames mutagenicity test whereas pure chlordane is negative (Tardiff et al., 1978). The criterion for the protection of human health must therefore be based on carcinogenicity data. The toxicological data for chlordane, including carcinogenicity data, relevant to the derivation of a water quality criterion have been reviewed by the NAS (1977d), and the upper 95% confidence limits of the lifetime risk calculated as 0.96 - 1.8×10^{-5} per ug/L per litre of water consumed daily. Assuming a 2 litre/day consumption and an acceptable risk of 1×10^{-6}, a criterion of 0.02 ug/L is derived, based on the upper limit.

More recently the US EPA (1978d, 1979b) derived a criterion of 0.12 ng/L corresponding to a risk of 1×10^{-6} for the protection of human health. This was based on a consumption of 2 litres of water and 18.7 g fish and shellfish per day. 98% of the chlordane exposure arose from the fish and shellfish; only 2% arose from the water.

The NAS criterion of 0.02 ug/L is however proposed for chlordane.

SUMMARY OF PROPOSED CRITERIA FOR CHLORDANE

Protection of the aquatic environment - 0.004 ug/L.

Production of fish suitable for human consumption - 0.0005 ug/L.

Protection of human health (drinking water quality) - 0.02 ug/L.

3.2.5 DDT & Related Compounds (TDE & DDE)

General

Although DDT was first prepared in 1874, it was not manufactured for use as an insecticide until 1939. It consists primarily of the p, p'-isomer with some of the o, p'-isomer. TDE (sometimes called DDD) is related to DDT. It usually occurs as a metabolite of DDT, but small quantities are produced for drug use.

It, too, is highly toxic with accumulative properties. DDE is another metabolite of DDT with toxic and accumulative properties. The rates of metabolism of DDT to TDE and DDE vary depending on the animal concerned (IARC, 1974d). Thus organisms, after exposure to DDT can contain varying ratios of the three compounds. The proportion of metabolites in relation to unmetabolised DDT can also be used to estimate the time of exposure. Residue determinations are usually carried out for the three compounds and the result expressed as Σ DDT (DDT + TDE + DDE).

DDT is the organochlorine insecticide over which most controversy has arisen. It was extensively used in large quantities for many years before it was realised that it and its metabolites were extremely persistent and were accumulating throughout the environment. The concern for the accumulation of these residues and their ubiquitous occurrence has resulted in the use of DDT being curtailed and even banned in some countries (IARC, 1974d). In 1972 the use of DDT for agricultural uses was banned in the USA and is now limited to minor public health uses (NAS, 1977e). However, as already mentioned, DDT is still extremely valuable for insect control in third world countries, especially in the control of insect borne disease. Some bans have been imposed in Australia.

Although attempts are being made to reduce the usage of DDT, production in the developed countries has not been reduced greatly as the major part of production is now being exported to third world countries. Figure 5 in the Appendix shows US production of DDT up to 1970.

US production in 1972 (the year its agricultural use was banned in that country) of 20×10^6 kg was still approximately 25% of the peak of 82×10^6 kg in 1963 (IARC, 1974d). Figures for Iran (Sodergren et al., 1978) and India (Edwards, 1975b) indicate the trend for larger quantities of DDT to be used in third world countries. It was estimated that approximately 50% of world production in 1971 was used for prevention of malaria, yellow fever and sleeping sickness (IARC, 1974d). World production and usage has been discussed in some depth by the IARC (1974d) and Australian Academy of Science (1972).

Usage in Australia up to 1971 has been well reviewed qualitatively in terms of target insects and the problems they cause (Australian Academy of Science, 1972). The quantities used can be determined since there has been no production of DDT in Australia since 1971, and import figures are available (Australian Bureau of Statistics, Annual Publication). These have been compiled (Pesticides Section, Australian Department of Primary Industry, 1980a) and show that DDT imports are now below the peak of 1973-74. In that period 3 625 005 kg were imported compared with 995 712 kg in 1978-79; in 1975-76 only 8 586 kg were imported, however, reflecting rather erratic imports. Usage for a particular year may not exactly match imports for that year due to the possibility of supplies being carried out from the previous year's stocks. The main application would appear to be for the control of insects on cotton, tobacco, sorghum, lucerne and some vegetables (Pesticides Section, Australian Department of Primary Industry 1975).

Residues in the aquatic environment

DDT and its metabolites have been detected in environmental samples with great frequency and more recent occurrences are too numerous to list. Several of these do involve the determination of DDT residues in Australia and New Zealand. These are shown in Table 8.

TABLE 8. DETERMINATIONS OF DDT AND METABOLITES IN THE ENVIRONMENT OF AUSTRALIA AND NEW ZEALAND

LOCATION	DETERMINED IN	REFERENCE
AUSTRALIA		
MURRUMBIDGEE irrigation area	Water rats	Olsen & Settle, 1979
MORETON BAY, QLD	Oysters	Clegg, 1974
TASMANIA	Oysters	Sumner, 1978
TASMANIA	Various including aquatic animals	Bloom *et al.*, 1979
?	Fish	Neuhaus *et al.*, 1973
GREAT BARRIER REEF	Fish, coral	Olafson, 1978
BRISBANE RIVER ESTUARY	Fish, crabs	Thomson & Davie, 1974
NORTHERN TERRITORY	Various including fish	Best, 1973
PERTH, WA	Human milk	Stacey & Thomas, 1975
VICTORIA	Human milk	Newton & Greene, 1972
SYDNEY, NSW	Human milk, blood, etc.	Siyali *et al.*, 1974
WESTERN AUSTRALIA	Human body fat	Wasserman *et al.*, 1968
QUEENSLAND	Human milk	Miller & Fox, 1973
NSW	Blood of pest control operators	Siyali & Simson, 1973
NEW ZEALAND		
Various	Birds & mammals, including aquatic birds	Lock & Solly, 1976
Various	Fish, crustaceans, molluscs	Solly & Harrison, 1972
Three Lakes	Rainbow trout	Solly & Shanks, 1969

The Great Lakes of North America is an area where significant pollution by DDT is occurring and where most monitoring is being carried out (Frank et al., 1977, 1978a b; Waller & Lee, 1979). DDT residues in fish and water from Lake Ontario have exceeded existing criteria and objectives (Waller & Lee, 1979).

High residue levels are still being found in fish near a sewage outfall in Los Angeles, California although the input of residues had been greatly reduced several years ago (McDermott-Ehrlich et al., 1978). Contaminated sediments now appear to be acting as a souce of DDT in that area (Young et al., 1977; Smokler et al., 1979).

Environmental effects

Considering the ubiquitous nature of DDT and its metabolites, only a few adverse effects directly attributable to accumulated residues have been reported. These have involved mostly impaired reproduction. Mortalities in young trout have been fairly conclusively attributed to DDT residues (Menzie, 1972). There is also strong evidence that mortalities in salmon were due to DDT (Anderson, 1971; Johnson & Ball, 1972). In one case, death of the fry occurred on absorption of the yolk sac containing DDT residues (Johnson & Ball, 1972), an effect which can be demonstrated in the laboratory with trout (Menzie, 1972). In another case, death coincided with cold temperature. Laboratory experiments with salmon showed that DDT raised the lower lethal temperature, thereby decreasing their ability to withstand the stress of low temperature (Anderson, 1971).

The decline in predatory bird populations associated with DDT residues has caused great concern (Cooke, 1973). The decline can be attributed to reproductive failure through the thinning of eggshells, with consequential loss through increased breakages. Similar effects have been obtained in laboratory experiments with birds fed on a diet containing DDE residues (Longcore et al., 1971; Menzie, 1972).

Marine birds in the Los Angeles zoo died when fed for a period of time with fish containing accumulated DDE. The residue levels in the dead birds were similar to those which were lethal in laboratory feeding experiments (Young et al., 1979). The decline of the peregrine falcon in Australia shows some correlation with the usage of DDT (Olsen & Olsen, 1979).

Many other aspects of DDT residues in relation to the aquatic environment have been reviewed (Anderson, 1971; Johnson & Ball, 1972; Fishbein, 1974; Goodman, 1974; Addison, 1976; NAS, 1977e; US EPA, 1978e) but a detailed discussion is outside the scope of this document.

Aquatic toxicology and criterion for the protection of the aquatic environment

In relation to criteria for the protection of the aquatic environment, the US EPA has reviewed the relevant data on DDT in its criteria documents (US EPA, 1976e, 1978e). In the 1976 document a criterion of 0.001 ug/L was proposed for the protection of aquatic life. The data used for criterion derivation involved bioaccumulation factors and levels of DDE which caused reproductive failure in birds.

More recently the US EPA (1978e, 1979a) has proposed a draft criterion of 0.00023 ug/L as a 24 hour average and 0.41 ug/L not to be exceeded at any time for the protection of freshwater aquatic life. The corresponding values for saltwater aquatic life were 0.0067 and 0.021 ug/L respectively. These were calculated using the formalised procedures developed by the US EPA. Using the data for the most sensitive species tested (30% mortality of *Penaeus duorarum* after 56 days exposure to 0.05 ug/L DDT (Nimmo *et al.*, 1970)) and an application factor of 0.01 for compounds which accumulate, a criterion of 0.0005 ug/L can be derived. This is derived by a more simplistic procedure advocated in this document and would also protect birds consuming fish from that water (based on the rationale of the US EPA (1976e)).

Many more recent studies of toxic and sublethal effects of DDT on aquatic organisms have been reported. Most of these, which are too numerous to list, are unsuitable for inclusion in the criteria derivation process. The study of Jarvinen *et al.* (1977) on fathead minnows (*Pimephales promelas*) is useful, however, in that it involved a chronic lifecycle study including reproduction. An MATC of 0.9 ug/L was obtained with an application factor of 0.0188 for exposure to DDT in water alone. An MATC of 0.4 ug/L was obtained with an application factor of 0.0083 for exposure to DDT in both water and diet. These data indicate that the application factor 0.01 is appropriate and support the criterion of 0.0005 ug/L for the protection of the aquatic environment.

Bioaccumulation and the production of fish suitable for human consumption

The Australian maximum residue limit for DDT in fish stipulated by the NH & MRC (1978) is 1 mg/kg. The maximum bioaccumulation factor cited by the US EPA (1976e) was 2×10^6. More recent studies have yielded lower factors but of the same order, e.g. 690 000 for mussels (Riseborough *et al.*, 1976). Thus, based on the maximum residue limit of 1 mg/kg and bioaccumulation factor of 2×10^6, a criterion of 0.0005 ug/L is derived which will provide for the production of fish suitable for human consumption.

Criterion for the protection of human health

In man, the presence of DDT and its metabolites has not been shown to have any direct detrimental effects, even in workers involved in the manufacture or use of DDT where high exposures have resulted in high residue levels. Similarly, prolonged exposure of volunteers to relatively high DDT levels has resulted in high residue levels in various tissues, but no attributable effects (IARC, 1974d). DDT residues have been found in the human population of Australia (Springett, 1970) and a case of aplastic anaemia associated with high level exposure to DDT has also been reported in this country (Woodliff, 1966).

DDT and its metabolites are carcinogens in mice (IARC, 1974d) and therefore possible carcinogens in man. However in the Ames test DDE has been found to be negative (McCann et al., 1975). As DDT is a possible human carcinogen the criterion for the protection of human health should be based on carcinogenicity data. The NAS (1977e) has reviewed the available data, and the lifetime risk of developing cancer through DDT exposure in water was calculated as 0.18 - 13.0×10^{-6} per ug/L per litre of water consumed daily, with upper 95% confidence limits of 0.65 - 20×10^{-6}. Based on the upper limit, and an acceptable risk of 1×10^{-6} a criterion of 0.02 ug/L is derived for a 2L/day consumption. The US EPA (1978e), 1979a) derived a criterion for drinking water of 0.098 ug/L corresponding to a risk of 1×10^{-6}. This was based on a consumption of 2 litres of water and 18.7 g fish and shellfish per day. Over 99% of the exposure was calculated to arise from the fish and shellfish. As discussed earlier criteria derived from NAS risk estimates which assume exposure through drinking water alone are considered more relevant. A drinking water quality criterion of 0.02 ug/L is thus proposed.

SUMMARY OF PROPOSED CRITERIA FOR DDT

Protection of the aquatic environment (including the protection of aquatic predatory birds) - 0.0005 ug/L

Production of fish suitable for human consumption - 0.0005 ug/L
Protection of human health (drinking water quality) - 0.02 ug/L

3.2.6 Dicofol

General

Dicofol is an insecticide of similar structure to DDT but is much less persistent. Its main use appears to be in the control of mites (Snelson, 1977). Residues have been determined in California as a result of extensive usage there (Khorran & Knight, 1977). Because of its infrequent detection in the aquatic environment dicofol would appear to be a pollutant of minor importance. It is included in this document for completeness.

Aquatic toxicology and criteria for the protection of the aquatic environment

Little attention appears to have been paid to this compound in relation to the derivation of water quality criteria. A few toxicity studies have been performed. The 48 hour LC50 for the shrimp Crangon franciscorum was reported as 43.7 - 83.2 ug/L with an indication that earlier studies with other organisms were in that range (Khorran & Knight, 1977). Based on the lower estimate of the toxicity data, and an application factor of 0.01, a criterion for the protection of the aquatic environment of 4 ug/L is derived. A conservative application factor of 0.01 is chosen because of the lack of data in that other organisms could conceivably be more sensitive.

Criterion for the protection of human health

An ADI for dicofol of 0.025 mg/kg has been set by the FAO (Vettorazzi, 1975). Based on this, assuming a 70 kg individual with a 2 litre/day consumption and 20% of the total intake in water, a criterion of 0.1 mg/L is derived for the protection of human health.

SUMMARY OF PROPOSED CRITERIA FOR DICOFOL

Protection of the aquatic environment - 4 ug/L

Protection of human health (drinking water quality) - 0.1 mg/L

3.2.7 Dieldrin (HEOD)

General

Dieldrin, a cyclodiene insecticide, is prepared by the epoxidation of aldrin. It is the epoxide of aldrin and like the epoxides of the other cyclodiene insecticides, is more toxic and persistent than the parent compound (IARC, 1974e).

Dieldrin is still used extensively for insect control although the usage is diminishing as a result of concern for its persistent and accumulative properties. It is used mainly for control of soil pests and a significant proportion is used for control of termites, insects which transmit disease (IARC, 1974e; Snelson, 1977), and mothproofing of woollen clothes and carpets (IARC, 1974e). The discharge of effluents from factories involved in the latter use has been shown to be the source of dieldrin residues in Yorkshire, England (Brown et al., 1979). It is also a possible source of dieldrin residues in Tasmania (Bloom et al., 1979).

The uses of dieldrin on crops in Australia are essentially the same as for overseas. It was used in the dipping of sheep for control of blowfly strike but unacceptable residue levels in the fat of treated animals have discouraged this use (Pesticides Section, Australian Department of Primary Industry, 1975).

The concern for the persistent residues and toxic properties of dieldrin is reflected in the trends in usage patterns. It was estimated that in the USA in 1972 only 20% of the peak of 1956 was used (Edwards, 1975). It is now banned in several countries (IARC, 1974e), including Finland (Hattula et al., 1978a), and the USA (agricultural purposes) (US EPA, 1978d). The quantities used, and usage patterns cannot be determined for Australia because of restrictions on the data.

Residues in the aquatic environment

The frequency of detection of dieldrin residues in the aquatic environment is second only to DDT. This is a consequence of its widespread use, persistence and accumulative properties.

Part of the load is due to the use of aldrin which is readily converted to dieldrin in the environment. Residues of dieldrin in catfish have been shown to be a result of the use of aldrin (Kellogg & Bulkley, 1976).

Dieldrin residues are detected so frequently in the aquatic environment that it is not possible to document all reports. Dieldrin residues have recently been reported in rainwater (Wells & Johnstone, 1978) suggesting that aerial transport plays a large part in the widespread distribution of this compound. For example, dieldrin has been reported in offshore waters of the North Atlantic Ocean (Jonas & Pfaender, 1976).

Dieldrin residues have frequently been reported from the Great Lakes area of North America (Glooschenko et al., 1976; Frank et al., 1977, 1978a, 1978b, 1979a; Norstrom et al., 1978; Zabik et al., 1978; Waller & Lee, 1979), indicating extensive pollution of this area and intensive monitoring as a consequence. Levels of dieldrin in fish from Lake Huron have occasionally exceeded the 0.3 mg/kg residue limit set by US health authorities (Frank et al., 1978a). Similarly residues in water from Lake Ontario were found to exceed existing criteria and objectives (Waller & Lee, 1979).

Dieldrin residues have frequently been determined in the environments of Australia and New Zealand. These have usually been determined in association with DDT, determinations of which have been documented in Section 3.2.5 on DDT. Consequently they will not be listed here.

Aquatic toxicology and criterion for the protection of the aquatic environment

Most of the available data on dieldrin toxicity and effects on aquatic organisms has been reviewed by the US EPA in its criteria documents (US EPA, 1976a, 1978a). In the 1976 document a criterion of 0.003 ug/L was derived for aldrin/dieldrin based on bioaccumulation factors and acceptable levels of dieldrin in fish. Because of the ready conversion of aldrin to dieldrin in the environment, the two compounds are usually considered together in deriving criteria. The derived criteria then apply to aldrin, dieldrin or the sum of both. However, as discussed under aldrin (Section 3.2.1) separate criteria can be derived for each compound. Separate criteria have been derived in this document but the possibility of aldrin being converted to dieldrin should be considered in applying criteria.

As discussed earlier, a criterion based on accumulation in fish is not necessarily the best criterion for the protection of the aquatic environment as it strictly only applies to the production of fish suitable for human consumption.

A large number of acute toxicity tests have been performed with dieldrin and a wide range of organisms. Fish tend to be reasonably sensitive, for example, a 96 hour LC50 of 1.1 ug/L for the rainbow trout, Salmo gairdneri has been cited (US EPA, 1978a). The toxicity to invertebrates varies considerably with marine invertebrates being particularly sensitive. A 48 hour LC50 of 0.28 ug/L has been cited for the pink shrimp, Penaeus duorarum (US EPA, 1978a). Insects are particularly sensitive with a 96 hour LC50 of 0.50 ug/L having been reported for the stonefly, Pteronarcys californica (Sanders & Cope, 1966). In a longer term test with a stonefly, Acroneuria pacifica, a 30 day LC50 of 0.2 ug/L was determined (Jensen & Gaufin, 1966). Immobilisation EC50 values of 0.24 and 0.25 ug/L have also been cited for daphnids (US EPA, 1976a). Based on the lowest acute toxicity value and an application factor of 0.01 since dieldrin accumulates, a criterion of 0.002 ug/L is derived for the protection of the aquatic environment. This is much lower than the no effect levels determined in chronic studies (US EPA, 1978a), and should therefore afford these organisms some protection.

Recently the US EPA (1978a, 1979a) proposed a draft criterion of 0.0019 ug/L as a 24 hour average and 1.2 ug/L not to be exceeded at any time for aldrin/dieldrin for the protection of freshwater aquatic life. The corresponding values for saltwater aquatic life were 0.0069 and 0.16 ug/L respectively. However, the criterion of 0.002 ug/L calculated in this document from a more simplistic procedure is proposed as this procedure is considered more realistic.

Bioaccumulation and the production of fish suitable for human consumption

No maximum residue limit for dieldrin in fish is stipulated by the NH & MRC in Australia. A limit of 0.2 mg/kg is set for meat fat (NH & MRC, 1978). The maximum bioaccumulation factor for dieldrin in fish is 100 000, with some invertebrates accumulating dieldrin to a slightly higher degree. Assuming a bioaccumulation factor of 100 000 and extending the Australian maximum residue limit cited above to fish, a criterion of 0.002 ug/L is derived to provide for the production of fish suitable for human consumption.

Criterion for the protection of human health

Dieldrin is a liver carcinogen in mice (IARC, 1974c;Esptein, 1975a,b). However the failure to promote tumours in other animals has prompted the argument of species specificity with the argument that tumour induction in animals, especially mice is not evidence for possible cancer induction in man (Wright et al., 1977). This theory is supported by evidence that dieldrin causes tumours in mice identical to those caused by phenobarbital, a drug used for the treatment of epilepsy in man without any apparent adverse effects. By extrapolation dieldrin is therefore not a human carcinogen in the view of the supporters of this theory (Van Raalte, 1977; Wright et al., 1977). Dieldrin is not mutagenic in the Ames test (McCann et al., 1975).

Epidemiological studies of workers involved in the handling of dieldrin have provided no evidence of human carcinogenicity (Van Raalte, 1977). The carcinogenicity of dieldrin is a matter of controversy as dieldrin is regarded as a carcinogen in the USA but not in Great Britain (Gillespie et al., 1979). It is considered that prudence should prevail where human health is concerned and therefore dieldrin be considered a possible human carcinogen at this stage. The drinking water criterion for this compound was derived accordingly, i.e. from carcinogenic risk estimates.

The NAS (1977a) has considered the available carcinogenicity data for dieldrin and determined the lifetime risk of developing cancer through exposure to dieldrin in drinking water as 0.8 - 1.9×10^{-4} per ug/L per litre of water consumed daily, with upper 95% confidence of 1.9 - 2.4×10^{-4}. Based on the upper limit, and an acceptable risk of 1×10^{-6}, a criterion of 0.002 ug/L is derived for a 2 litre/day consumption.

More recently the US EPA (1978a, 1979a) derived a criterion of 4.4×10^{-3} ng/L corresponding to a risk of 1×10^{-6}. This was based on exposure through water and organisms grown in that water and assumed a consumption of 2 litres of water and 18.7 g fish and shellfish per day. A bioaccumulation factor of 4 500 was used in the calculation. 98% of the dieldrin exposure arose from the fish and shellfish while only 2% arose from the drinking water. The criterion based on NAS risk estimates is considered more relevant as it assumes exposure through drinking water alone and is therefore proposed.

SUMMARY OF PROPOSED CRITERIA FOR DIELDRIN

Protection of the aquatic environment - 0.002 ug/L.

Production of fish suitable for human consumption - 0.002 ug/L.

Protection of human health (drinking water quality) - 0.002 ug/L.

The presence of aldrin must also be considered when applying these criterion and if present, allowances must be made for its conversion to dieldrin.

3.2.8 Endosulfan (Thiodan)

General

Endosulfan is one of the few organochlorine insecticides with little tendency to accumulate in exposed organisms. It is a cyclodiene insecticide prepared from hexachlorocyclopentadiene and (Z)-1,4-diacetoxybut-2-ene followed by hydrolysis and reaction with thionyl chloride. It is a mixture of two isomers, the α -which constitutes approximately 70% and the β - the remaining 30% (Martin & Worthing, 1977b). The relative toxicity of the two isomers is not known (US EPA, 1978f).

It is used on a wide range of crops as evidenced by the large number of products where maximum residue limits have been established by the NH & MRC (1978). It is effective against many of the pests for which DDT was formerly used. It has a short residual life which, makes it useful for control of pests in pastures and forage crops. (Snelson, 1977). In the USA despite restrictions its use is still significant (US EPA, 1978).

Residues in the aquatic environment

Endosulfan is relatively rarely found in the environment because of its ready degradation and its non-cumulative properties (Vettorazzi, 1975; Martin & Worthing, 1977b). The α -isomer has however been found in low levels in animals from the Wester Estuary, Germany (Goerke et al., 1979), and the β -isomer in oysters from the Gulf of Mexico (Rosales et al., 1979). The α - and β -isomers have been found in sediments from the Great Lakes (Frank et al., 1977). Endosulfan has often been determined in the Rhine River (Greve & Wit, 1971; Greve, 1972; Wegman & Greve, 1978). In recent years levels in the Rhine tended to be lower and it was less frequently detected compared with 1969 when levels of up to 0.81 ug/L were recorded (Wegman & Greve, 1978). The high levels recorded in 1969 were greater than LC50 levels for some fish and were associated with massive fish kills during that year (Greve & Wit, 1971).

Aquatic toxicology and criterion for the protection of the aquatic environment

The toxicity data for endosulfan have been reviewed by the US EPA in its criteria documents (US EPA, 1976f, 1978f). It is highly toxic to fish which are in general more sensitive than invertebrates.

In its 1976 document, the US EPA derived criteria of 0.003 and 0.001 ug/L for the protection of freshwater and marine aquatic life respectively. The freshwater criterion was derived from the 96 hour LC50 for the rainbow trout (0.3 ug/L at 10°C) and an application factor 0.01. The marine criterion was derived from the 96 hour LC50 for the striped bass (0.1 ug/L) and the same application factor.

More recently a criterion of 0.042 ug/L as a 24 hour average and 0.49 ug/L not to be exceeded at any time was proposed by the US EPA (1978f, 1979c) for the protection of freshwater aquatic life. The data were considered inadequate for derivation of a saltwater criterion.

The most sensitive species so far tested is the harlequin fish, Rasbora heteromorpha with a 96 hour LC50 of 0.014 ug/L (Alabaster, 1969). Slightly higher acute toxicity values have been reported for other organisms, e.g. a 96 hour LC50 of 0.1 ug/L for the striped bass, Morone saxatilis (Korn & Earnest, 1974) and a 96 hour LC50 of 0.04 and 0.09 ug/L for the grass shrimp, Palaemonetes pugio and spot, Leiostomus xanthurus respectively (Schimmel et al., 1977c). Based on the 96 hour LC50 of 0.014 ug/L and an application factor of 0.05 a criterion for the protection of marine aquatic life of 0.0007 ug/L can be derived. An application factor of 0.01 appears low for endosulfan which does not accumulate and 0.1 appears too high because of the high toxicity to commercially important organisms such as fish. Hence 0.05 is considered a reasonable compromise. One long term study involving reproduction has been reported (Macek et al., 1976b). An MATC of 0.2-0.4 ug/L was determined for the fathead minnow. No acute toxicity data were reported so strictly no application factor can be calculated. However, from the data tabulated by the US EPA (1978f) an LC50 of approximately 2-5 ug/L could be expected and an application factor of 0.04-0.1 thus derived. The selected value of 0.05 is within this range.

Bioaccumulation and the production of fish suitable for human consumption

Since endosulfan has little tendency to accumulate, the derivation of a criterion for the production of fish suitable for human consumption is not warranted.

Criterion for the protection of human health

Endosulfan has not been shown to be carcinogenic in animal experiments (US EPA, 1978f, 1979c). A criterion for the protection of human health can therefore be calculated from the ADI data. An ADI for man of 0.0075 mg/kg has been established for this compound (Vettorazzi, 1975). Thus, based on this and assuming a 70 kg individual and a 2 litre/day consumption of water with 20% of the total intake derived from water, a criterion of 50 ug/L is derived for the protection of human health.

The US EPA (1978f, 1979c) has recently proposed a criterion of 0.1 mg/L for endosulfan in drinking water in order to protect human health. The data on which this was based was a no-observed-effect level of 0.4 mg/kg/day for the mouse. Applying a safety factor of 0.1, an ADI of 0.28 mg/day for a 70 kg individual was calculated.

Assuming a consumption of 2 litres of water and 18.7 g fish and shellfish per day with a bioaccumulation factor of 28, a criterion of 0.1 mg/L was derived. Based on exposure through water alone, a criterion of 0.1 mg/L is still derived. The criterion based on the FAO/WHO ADI is proposed (50 ug/L) however, to be consistent with procedures used in other parts of this document.

SUMMARY OF PROPOSED CRITERIA FOR ENDOSULFAN

Protection of the aquatic environment - 0.0007 ug/L

Protection of human health (drinking water quality) - 50 ug/L.

3.2.9 Endrin

General

Endrin is another of the cyclodiene insecticides prepared from hexachlorocyclopentadiene. It is a stereoisomer of dieldrin. It is an epoxide and like the epoxides dieldrin and heptachlor epoxide, is quite toxic. It is the most toxic of organochlorine insecticides to insects and to mammals, including man. It is approximately 100 times more toxic than DDT and has a similar toxicity to parathion (Conservation Council of Victoria, 1979).

The high toxicity of endrin has restricted its uses in Australia. Its more important uses include control of insects on cotton and tobacco, and in orchards (Pesticides Section, Australian Department of Primary Industry, 1975). Overseas, the uses of endrin are widely restricted and in the USA it is registered for use on only a few crops. Other important uses are for the control of birds and mice (IARC, 1974f). It is estimated that 70% of the endrin produced is used on cotton (Snelson, 1977).

Residues in the aquatic environment

Endrin is rapidly metabolised by animals and residues are not a great problem (IARC, 1974f; Vettorazzi, 1975). The data show that with aquatic organisms chronic exposures can result in accumulation (Grant, 1976; US EPA, 1976g, 1978g), with rapid elimination on termination of exposure (US EPA, 1976g, 1978g). The occurrences of endrin in the environment are not many in comparison with DDT and dieldrin, reflecting its smaller use and ready elimination and degradation. The occurrence of endrin in the freshwater environment has been reviewed by Grant (1976). Other more recent occurrences include mussels from the Dutch coast (Quirjins *et al.*, 1979), and oysters from Mexico (Rosales *et al.*, 1979).

Aquatic toxicology and criteria for the protection of the aquatic environment

Endrin is one of the most toxic pesticides to fish which makes it particularly important that criteria be available in this regard. Most of the available toxicity data for endrin and freshwater life have been reviewed by Grant (1976). The toxicity data for endrin and aquatic organisms in general have been reviewed by the US EPA in criteria documents (US EPA, 1976g, 1978g). The 1976 criterion was calculated from the acute toxicity data for the most sensitive organism for which data was available. This was the pink shrimp, *Penaeus duorarum* with a 96 hour LC50 of 0.037 ug/L (Schimmel *et al.*, 1974). The application factor used was 0.1, a compromise between the 0.01 usually used for compounds which accumulate and the 0.3 experimentally determined from the relationship between the safe and acutely lethal concentration. From these data a criterion of 0.004 ug/L was proposed. The experimentally determined application factor of 0.3 which was cited is unusually high for organochlorine insecticides, however, it was also obtained by Hermanutz (1978) in his study of endrin toxicity to the flagfish. Utilising the practice adopted in this document of rounding to the lower value, a criterion of 0.003 ug/L is derived from the same data. Lower LC50 levels have been obtained for insects over longer time intervals. These were a 30 day LC50 of 0.035 ug/L for the stonefly, *Acroneuria pacifica* (Jensen & Gaufin, 1966), and a 28 day LC50 of < 0.034 ug/L for the caddis-fly, *Brachycentrus americanus* (Anderson & DeFoe, 1980). Using a value of 0.034 ug/L as the lowest LC50 reported and an application factor of 0.1 a criterion of 0.003 ug/L is still derived.

In a partial chronic study, endrin concentrations of 0.14 ug/L affected survival of progeny of fathead minnows (Jarvinen & Tyo, 1978). An MATC of > 0.12 and < 0.31 ug/L was determined for the sheepshead minnow, *Cyprinodon variegatus* (Hansen *et al.*, 1977). Thus the criterion of 0.003 ug/L would certainly protect these species. In the latter study, a lifecycle study involving reproduction, an application factor of between 0.35 and 0.91 was experimentally determined. This appears to be the study from which the US EPA (1976g) obtained the value of 0.3 which it cited. In a life cycle test involving reproduction with the grass shrimp, *Palaemonetes pugio*, gonadal development and spawning were inhibited at 0.03 ug/L, the lowest concentration tested. An application factor of less than 0.08 was also determined (Tyler-Schroeder, 1977). Thus the criterion of 0.003 ug/L should afford some protection to this species since it is factor of 10 lower. The application factor chosen as most appropriate for criterion derivation is also in good agreement with the value determined in that study.

More recently a draft criterion of 0.002 ug/L as a 24 hour average and 0.1 ug/L not to be exceeded at any time was proposed for the protection of freshwater aquatic life. The corresponding values for saltwater aquatic life were 0.0047 and 0.031 ug/L respectively (US EPA, 1978g, 1979c). However criteria derived in this document using more simplistic procedures are considered more realistic and a criterion of 0.003 ug/L is thus proposed for endrin for the protection of the aquatic environment.

Bioaccumulation and the production of fish suitable for human consumption

A maximum residue limit of 0.02 mg/kg for endrin is stipulated by the NH & MRC (1978) for some food products in Australia. Although endrin residues are rapidly eliminated on termination of exposure, continuous exposure can result in biomagnification up to 26 000 times (US EPA, 1976g). Thus based on the maximum residue limit of 0.02 mg/kg and a bioaccumulation factor of 26 000, a criterion of 0.0007 ug/L is derived which will allow for the production of fish to meet this maximum residue limit.

Criterion for the protection of human health

The IARC (1974f) evaluation of the carcinogenic risk for endrin concluded that there was insufficient data to draw firm conclusions. Using no-effect levels, safety factors and the usual assumptions, the US EPA (1976g) derived a drinking water quality criterion of 0.2 ug/L for endrin.

A more recent review of later data considers endrin to be carcinogenic in the rat (Reuber, 1979b). It is negative for mutagenicity in all short term tests (Waters et al., 1980). The NAS (1977f) considered endrin to be a suspected carcinogen but risk estimates could not be performed due to insufficient data.

An ADI of 0.004 ug/kg/day can be derived from the maximum no-effect level (0.02 mg/kg/day) for the most sensitive animal tested (the dog) (US EPA, 1976g) using a safety factor of 1/5 000, the large safety factor being used because of the suspected carcinogenicity. This is the safety factor recommended by Weil (1972) and adopted in this document for such compounds. Based on a 70 kg individual with a 2 litre/day consumption of water and an intake of 20% of the ADI in water, a criterion of 0.02 ug/L can be derived. This is a factor of 10 less than that reported by the US EPA (1976). The difference lies in the use of a more conservative application factor because of the recent assessment of the possible carcinogenic potential of endrin.

The FAO/WHO ADI for endrin is 0.0002 mg/kg (Vettorazzi, 1975). Utilising the usual assumptions (a 70 kg individual consuming 2 litres/day water with 20% of the ADI from the water) a criterion of 1 ug/L can be derived.

Recently the US EPA (1978g) also reviewed the data for endrin. It was considered not to be carcinogenic and a no-effect level of 0.1 mg/kg was reported with a safety factor of 1/100 considered as being appropriate. Thus an ADI of 0.001 mg/kg was derived. A criterion of 1.87 ug/L was derived for exposure through consumption of 2 litres water and 18.7 g fish and shellfish per day, a bioaccumulation factor of 1 900 being used in the calculation. Assuming more simplistically that 20% of the exposure arises through drinking water, an approach used throughout this document, a criterion for the protection of human health of 7 ug/L can be derived.

Vastly different criteria for endrin can be determined depending on the data used. These values are influenced mainly by the safety factors employed which depend on whether endrin is considered a carcinogen. This would appear to be a contentious issue at present. As it is possible that endrin may be carcinogenic, a conservative application factor of 1/5 000 appears most appropriate. Thus a criterion of 0.02 ug/L is proposed for the protection of human health.

SUMMARY OF PROPOSED CRITERIA FOR ENDRIN

Protection of the aquatic environment - 0.003 ug/L

Production of fish suitable for human consumption - 0.0007 ug/L

Endrin residues are not likely to be a problem due to the relatively rapid metabolism in most cases.

Protection of human health (drinking water quality) - 0.02 ug/L.

3.2.10 Heptachlor

General

Heptachlor is a cyclodiene insecticide which places it in the same group as aldrin, dieldrin, endrin and chlordane. It also rapidly forms, either biologically or photochemically, a toxic epoxide, heptachlor epoxide (NAS, 1977g), which is also the major metabolic residue (IARC, 1974g). Heptachlor is also formed as a byproduct in the preparation of chlordane from chlordene and constitutes a percentage of technical chlordane (Sovocool et al., 1977). Technical heptachlor can also contain 22% trans-chlordane (Schimmel et al., 1976b). Heptachlor has a number of uses as an insecticide. It is very effective against soil insects and is used as a seed dressing (Snelson, 1977). It is also used against termites (Pesticides Section, Australian Department of Primary Industry, 1975). The main crops on which heptachlor is used would appear to be corn, sorghum, rice, pineapples and sugar cane (Snelson, 1977).

Heptachlor like most of the organochlorine insecticides is particularly persistent and accumulates in the environment. These undesirable properties have resulted in its use being restricted in many of the developed countries. In 1974 it was reported that in the USA its use was being constantly reviewed with tighter restrictions on its use being applied. It was finally banned in that country for use on agricultural crops in 1976 (NAS, 1977g). It is also banned in other countries (IARC, 1974g).

Residues in the aquatic environment

Because of the widespread use of heptachlor and the persistence of both heptachlor and its epoxide, residues of both are frequently detected in the environment. Heptachlor has recently been determined in water from northern Mississippi (Rihan et al., 1977), South Carolina (Sandu et al., 1978), and Hawaii (Tanita et al., 1976) in the USA. In the latter two cases heptachlor epoxide was also determined. The ready conversion of heptachlor to its epoxide is evidenced by its frequent determination in the tissues of animals from the aquatic environment, e.g. in fish and herring gull eggs from the Great Lakes (Norstrom et al., 1978), in fish from British Columbia (Albright et al., 1975), and the Great Lakes (Frank et al., 1978a, b), and various organisms from an aquatic environment in Idaho, USA (Perry, 1979). Heptachlor and analogues have also been found in lake trout as a result of contaminated feed (Parejko & Wu, 1977). Few reports of heptachlor in the Australian environment have been made. Heptachlor and its epoxide were found in fish and shellfish (Neuhaus et al., 1973). Corals and fish from the Great Barrier Reef occasionally contained heptachlor and heptachlor epoxide (Olafson, 1978).

Aquatic toxicology and criterion for the protection of the aquatic environment

The US EPA (1976h, 1978h) has reviewed most of the toxicity data for heptachlor in its criteria documents. In the 1976 document a criterion of 0.001 ug/L was derived for the protection of aquatic life based on the toxicity to the most sensitive organism for which data were available (the pink shrimp, Penaeus duorarum with a 96 hour LC50 of 0.11 ug/L) and an application factor of 0.01 since heptachlor accumlates.

Recently the US EPA (1978h, 1979b) proposed a draft criterion of 0.0015 ug/L as a 24 hour average and 0.45 ug/L not to be exceeded at any time for heptachlor for the protection of freshwater aquatic life. The corresponding values for saltwater aquatic life were 0.0036 and 0.05 ug/L respectively.

The toxicity of heptachlor to a number of organisms has been established. Like all organochlorine insecticides it is highly toxic to aquatic organisms with invertebrates being more sensitive than fish. Marine organisms are more sensitive than freshwater organisms. For example, 7.0 ug/L has been cited as the 96 hour LC50 for the most sensitive freshwater fish, the rainbow trout, Salmo gairdneri while the corresponding value for the most sensitive saltwater fish, the bluehead, Thallassoma bifasciatum is 0.8 ug/L (US EPA, 1978h). The stonefly, Pteronarcys badia was reported to have a 96 hour LC50 of 0.9 ug/L (Sanders & Cope, 1968).

The most sensitive organism is the pink shrimp, Penaeus duorarum with a 96 hour LC50 of 0.03 ug/L (Schimmel et al., 1976a). Based on this value and an application factor of 0.01 since heptachlor accumulates, a criterion of 0.0003 ug/L is derived. Some long term studies are available for comparison. An MATC of between 0.86 and

1.84 ug/L with an application factor of 0.07 has been reported for the fathead minnow (Macek et al., 1976b). For the sheepshead minnow in a partial lifecycle study an MATC was not well defined. It was possibly less than 0.71 ug/L with an application factor of 0.07 (Hansen & Parrish, 1977). The criterion of 0.0003 ug/L should protect both these species. The application factor of 0.01 may be a little low but the uncertainty in one of the tests justifies its retention. Thus a criterion for the protection of aquatic life of 0.0003 ug/L is proposed.

Birds are sensitive to low doses of heptachlor and this criterion would also be sufficient to protect aquatic birds consuming fish grown in this water (US EPA, 1976). The metabolite of heptachlor, heptachlor epoxide has similar toxicity to heptachlor (Schimmel et al., 1976a), so the criterion proposed would also suffice for heptachlor epoxide.

Bioaccumulation and the production of fish suitable for human consumption

The maximum residue limit for heptachlor, including the epoxide, for meat fat, stipulated by the NH & MRC (1978) in Australia is 0.2 mg/kg. Bioconcentration factors of greater than 20 000 have been reported for heptachlor (US EPA 1976h), but a significant amount of the epoxide may also be present. For example, fish exposed to heptachlor contained appreciably more heptachlor epoxide than heptachlor during the depuration part of the experiment (Schimmel et al., 1976b). A criterion of 0.0003 ug/L for protection of the aquatic environment will allow for a bioaccumulation factor of 670 000 before the maximum residue limit is reached. Therefore this criterion should more than suffice for the production of fish suitable for human consumption, even allowing for conversion to the epoxide.

Criterion for the protection of human health

The IARC (1974g) evaluation of the carcinogenic risk to man for exposure to heptachlor produced inconclusive results. Evaluations based on more recent data have shown heptachlor and its epoxide to be carcinogenic in mice (Epstein, 1976; NAS, 1977g; US EPA, 1978h). Therefore heptachlor must be considered a possible human carcinogen and the criterion of drinking water quality determined from a consideration of the carcinogenicity data. The NAS (1977g) has reviewed this data and established the upper 95% confidence limits of the lifetime risk as $3.5\text{-}4.8 \times 10^{-5}$ per ug/L per litre of water consumed daily. Taking the upper extreme and assuming a 2 litre/day consumption with an acceptable risk of 1×10^{-6} a criterion of 0.01 ug/L is derived for drinking water quality. The US EPA (1978g 1979b) has recently derived a drinking water quality criterion of 0.023 ug/L for heptachlor corresponding to a risk of 1×10^{-6}. The calculation assumed exposure through ingestion of 2 litres of water and 18.7 g fish and shellfish per day. A bioaccumulation factor of 5 200 was used in the calculation and 98%

of the exposure arose from the fish and shellfish with only 2% from the drinking water. As calculations based on exposure through consumption of drinking water alone are considered more relevant in Australia the criterion of 0.01 ug/L based on NAS risk estimates is proposed.

SUMMARY OF PROPOSED CRITERIA FOR HEPTACHLOR

Protection of the aquatic environment including production of fish suitable for human consumption - 0.0003 ug/L.

Protection of human health (drinking water quality) - 0.01 ug/L.

3.2.11 Methoxychlor

General

Methoxychlor is an insecticide of similar structure to DDT but, unlike DDT, does not accumulate to any great extent. It is comprised mainly of the *p,p'* -isomer with some of the *o,p'*-isomer. It is used (based on US data) on a wide range of crops (including several types of seed), on animals and in agricultural premises (IARC, 1974h). Other applications in the USA include its use as a blackfly larvicide and for control of beetles involved in Dutch elm disease (NAS, 1977h). Its very low mammalian toxicity makes it an attractive insecticide and also leads, in part, to its widespread use. It is often used a a replacement for DDT.

Residues in the aquatic environment

Due to the rapid degradation and metabolism of methoxychlor, residues are not often found in the environment (IARC, 1974h). However methoxychlor has been determined recently in drinking water in the USA (Sandhu *et al.*, 1978). Residues detected recently in fish (Lockhart *et al*, 1977), and in fish, water and sand (Freeden *et al.*, 1975) in Canada, were associated with deliberate introduction of methoxychlor into streams for control of blackfly larvae. The lack of residues found in a watershed receiving widespread applications of methoxychlor, a situation expected from its ready degradation, demonstrated the low potential for this compound as a water pollutant (Sullivan & Atchison, 1977).

Aquatic toxicology and criterion for the protection of the aquatic environment

The US EPA (1976i) has reviewed most of the relevant toxicity data and established a water quality criterion for methoxychlor. The criterion for the protection of marine aquatic life of 0.03 ug/L was calculated from the 96 hour LC50 for the most sensitive species tested, the pink shrimp, *Penaeus duorarum* and application factor of 0.01. The criterion of 0.03 ug/L for the protection of freshwater aquatic life was a factor of 10 lower than the level causing effects on invertebrate populations in a 1 year dosing of a natural stream. Since methoxychlor does not accumulate to any great extent, the application factor of 0.01 appears too conservative and an application factor of 0.1 appears more appropriate.

A long term chronic toxicity study involving reproduction has recently been reported for sheepshead minnows, Cyprinodon variegatus. From this study an MATC of between 12 and 23 ug/L was determined for this species with an application factor of between 0.24 and 0.47 (Parrish et al., 1977). This data supports the use of an application factor of 0.1 for deriving criteria for methoxychlor rather than the 0.01 previously used.

The most sensitive species for which data were available to the US EPA was the crayfish, Orconectes nais with a 96 hour LC50 of 0.5 ug/L. A more recent study with crabs does not provide LC50 data, but the data indicated that levels near 0.5 ug/L were acutely toxic to the larvae (Bookhout et al., 1976). Crab zoeae were recently shown to be very sensitive with a 96 hour LC50 of 0.42 ug/L and in long term studies 0.05 ug/L had effects on the survival of zoeae (Armstrong et al., 1976). The 28 day LC50 for the isopod Asellus communis was also determined as 0.42 ug/L (Anderson & DeFoe, 1980). A study involving the continuous dosing of a small stream at 0.2 ug/L methoxychlor showed only minor effects (Eisele & Hartung, 1976). Thus based on the 96 hour LC50 for the most sensitive species and an application factor of 0.1, a criterion of 0.04 ug/L is derived for the protection of the aquatic environment. It will afford some protection to crab zoeae.

Bioaccumulation and the production of fish suitable for human consumption

Although it is generally considered that methoxychlor is considerably less bioaccumulative in aquatic organisms than other organochlorine pesticides (US EPA, 1976i), the degree of accumulation would appear to depend on the nature of the organisms. Toads exposed to methoxychlor had bioaccumulation factors of only 2.8, presumably because of rapid elimination in this animal (Hall & Swineford, 1979). Fish do not accumulate methoxychlor to any great extent (bioaccumulation factors of several hundred appear to be typical) but accumulation by crabs (Armstrong et al., 1976; Bookhout et al., 1976), and snails (Anderson & DeFoe, 1980) is several thousand times the water concentration. Even though invertebrates accumulate methoxychlor, residues are rapidly eliminated on termination of exposure.

A maximum residue limit of 3 mg/kg for methoxychlor in meat fat has been set by NH & MRC (1978) in Australia. Applying this limit to fish, the criterion of 0.04 ug/L for the protection of the aquatic environment allows for a bioaccumulation factor of 75 000, well above anything reported. Thus the criterion of 0.04 ug/L will also allow for the production of fish suitable for human consumption.

Criterion for the protection of human health

The IARC (1974h) evaluation of the carcinogenic potential of methoxychlor to man considered the data inadequate. A more recent evaluation considers the data to be negative for tumour induction in rats (NAS, 1977h). Methoxychlor is non-mutagenic in short term tests and is also negative in recently reported animal carcinogenicity experiments (Waters et al., 1980). Therefore methoxychlor can be considered to pose no carcinogenic risk to man. On the basis of the highest no-observed-effect level of 10 mg/kg/day in the rat and a safety factor of 100, an ADI of 0.1 mg/kg was calculated and, assuming a 70 kg individual with a 2 litre/day consumption of water and that 20% of the total intake was from water, a criterion of 0.7 mg/L was derived (NAS, 1977h). This criterion is proposed.

SUMMARY OF PROPOSED CRITERIA FOR METHOXYCHLOR

Protection of the aquatic environment (including production of fish for human consumption) - 0.04 ug/L.

Protection of human health (drinking water quality) - 0.7 mg/L.

3.3 ORGANOPHOSPHATE INSECTICIDES

General

This group of insecticides comprises a large number of compounds with widespread use. In South Australia alone over 30 compounds are registered for use. Their usage is increasing as they replace the persistent organochlorine insecticides. By 1972 malathion was reported to be the most widely used insecticide in the USA (Bourquin, 1977). They have the advantage of being labile, i.e. residues are relatively non-persistent on crops and soils.

Organophosphate insecticides generally have low toxicity to fish compared with organochlorine insecticides. Crustacea and aquatic insects tend to be much more sensitive than fish. Their use, as opposed to organochlorine insecticides, does offer some added protection for fish which may be considered as the most important organisms in the aquatic environment. Their high mammalian toxicity, however, makes their application a hazard.

The relative ease with which organophosphate insecticides are degraded in the environment, and residues metabolised in exposed organisms, lessens pollution to some extent. However, their widespread use can result in significant short-term pollution of the aquatic environment near the point of application. A number are also used for insect control in the interests of public health, e.g. for control of mosquitoes and midges, and as such are applied directly to the aquatic environment.

Residues in the aquatic environment

Residues of organophosphate insecticides are rarely found in the aquatic environment because they degrade readily, especially under alkaline conditions, e.g. malathion (Wolfe et al., 1977) and dimethoate (EL Beit et al., 1978). Also they do not accumulate to any great extent in exposed organisms, a fact which has been demonstrated in a number of laboratory experiments, e.g. diazinon by fish (Kanazawa, 1978; Goodman et al., 1979), malathion by fish (some metabolites were determined) (Cook et al., 1976), and fenitrothion by clams and mussels (McLeese et al., 1979).

Organophosphate residues are not biomagnified up the food chain and therefore do not reach the sometimes high levels of organochlorine insecticides with their associated adverse effects.

The compilations of determinations of organic chemicals in the aquatic environment referred to in Section 1.1 contain few organophosphate insecticides. More recently a survey of Ottawa, Canada drinking water failed to detect 16 organophosphate insecticides for which a procedure had been developed with detection limits for each compound of 1 ng/L (Lebel et al., 1979). Several organophosphate insecticides were detected in estuarine fish during a routine monitoring program (Butler & Schutzmann, 1978). Fenitrothion was detected in water (Mallet et al., 1978), and in water and aquatic plants (Moody et al., 1978) immediately after aerial spraying for insect control. In the latter case fenitrothion rapidly disappeared indicating its low persistence. Low levels of temephos residues were detected in milkfish, Chanos chanos after treatment of ponds for control of chironomids (Tsai, 1978). Temephos was also determined in snails from a salt-marsh immediately after spraying for mosquito control (Fitzpatrick & Sutherland, 1976).

The River Rhine is monitored regularly for cholinesterase inhibitors (Greve et al., 1972; Wegman & Greve, 1978; Poels et al., 1978). The procedure used measures the inhibition of the enzyme acetylcholinesterase which is a property of organophosphate insecticides. Significant cholinesterase inhibitor levels are frequently recorded and diazinon, dimethoate, malathion and parathion have been specifically identified by mass spectrometry. Carbamate insecticides also have the property of inhibiting acetylcholinesterase and are also determined by this procedure (Greve et al., 1972). Levels of cholinesterase inhibitors as high as 56 ug/L (as paroxon equivalents) were recorded. The concentrations in the Rhine were reported to have increased since 1972 and significantly in 1975 (Wegman & Greve, 1978).

Aquatic toxicology and criteria for the protection of the aquatic environment

With organophoshate insecticides chronic exposure of the aquatic environment would require continuous contamination since in general these compounds are rapidly degraded. As pollution is more likely to be sporadic, criteria for the protection of the aquatic environment were derived in relation to acute exposures and therefore were based on acute toxicity data.

For frequent exposures which may approximate chronic conditions such criteria must be used with caution. Organophosphate insecticides act by inhibiting the enzyme acetylcholinesterase (AChE). Although residues of the insecticide itself may not be readily detectable in an exposed organism and therefore indicate exposure, AChE may be inhibited for some considerable time after exposure (Coppage & Mathews, 1975; US EPA 1976j; Thirugnanam & Forgash, 1977; Verma et al., 1979). Thus repeated exposures can have cumulative effects if the AChE level does not have time to recover between exposures (Coppage & Mathews, 1975; Thirugnanam & Forgash, 1977). Criteria cannot be derived taking this factor into account, because the resultant effect will depend not only on the dose but also the frequency of exposure.

Since death can be associated with a minimum AChE level, the concentration of AChE could perhaps be used as a criterion of water quality. It would appear that inhibition by more than 35-40% could result in damage to aquatic organisms for some organophosphate insecticides (US EPA, 1976k).

The cumulative effect of these insecticides has been demonstrated in the results of same chronic lifecycle or part lifecycle toxicity studies that have been carried out. The very low MATC levels for diazinon (Allison & Hermanutz, 1977; Goodman et al., 1979), and azinphos-methyl (Adelman et al., 1976) in comparison with the LC50 levels are indicative of this effect. Two studies with malathion however, have reported data which indicate that the ratio of the MATC to the LC50 which is the experimentally determined application factor, is nearer the expected value of 0.1 for compounds which do not accumulate (Parrish et al., 1977; Hermanutz, 1978). The use of an application factor of 0.1 for malathion leads to a criterion greater than some recorded no-effect or chronic toxicity levels (Table 9). However the criteria in Table 9 relate more to acute exposures which are considered more realistic for this class of compound.

Organophosphate insecticides at low concentrations have been reported to increase the nitrogen fixation and growth of algae (Wurtsbaugh & Apperson, 1978). This effect, which appears to be partly due to the removal of grazing organisms, could occur in the natural environment with consequent detrimental effects. Such data should be incorporated in the criterion derivation process if possible, however, data are inadequate for this at the present time.

Because of the relatively low priority for criteria for organophosphate insecticides, coupled with the large number of compounds, individual compounds will not be discussed. Derived criteria for the protection of the aquatic environment are simply summarised in Table 9. Criteria were based on the acute toxicity to the most sensitive species and an application factor of 0.1. For many compounds the data were scant, i.e. acute toxicity levels had been established for only a few organisms. Thus it was necessary to determine whether the data available were likely to include the most sensitive species. From a compilation of toxicity data for aquatic organisms (US EPA, 1972) it was evident that crustacea and aquatic insects were much more sensitive to organophosphate insecticides than fish. From the compiled data it was also evident that Daphnia were among the most sensitive organisms. Thus data were considered to be adequate for criteria derivation if a number of organisms, including Daphnia had been tested, otherwise the following assumptions were made. It was indicated that, for most organophosphate insecticides, the LC50 levels for the most sensitive species were in the range 0.1-1.0 ug/L (US EPA, 1976k). Thus for most of the compounds where

TABLE 9 ACUTE TOXICITIES FOR MOST SENSITIVE ORGANISMS AND DERIVED CRITERIA FOR ORGANOPHOSPHATE INSECTICIDES

COMPOUND	ORGANISM	ACUTE TOXICITY (LC50)		REFERENCE	DERVIED
		Hours	(ug/L)		CRITERION (ug/L)[m]
Azinphos-methyl	Gammarus fasciatus & G. pseudolimneaus	96	0.10	US EPA, 1972	0.01[c]
Azinphos-ethyl	Daphnia pulex	48	3.2	"	0.3
Carbophenothion	Palaemonetes kadiakensis	96	1.2	"	0.01[a]
Chlorpyrifos	Palaemon macrodactylus	96	0.01	"	0.001
Chlorothion	Daphnia magna	48	4.5	"	0.01[a]
Coumaphos	Gammarus fasciatus	96	0.07	"	0.007
Crotoxyphos	Gammarus fasciatus	96	11	"	0.1[b]
Demeton	Gammarus fasciatus	96	27	"	0.1
Diazinon	Daphnia pulex	48	0.90	"	0.09[d]
Dichlorvos	Daphnia pulex	48	0.07	"	0.007
Dioxathion	Gammarus fasciatus	96	8.6	"	0.1[b]
Disulfoton	Pteronarcys californica	96	5	"	0.1[b]
EPN	Penaeus duorarum	96	0.29	Schimmel et al., 1979	0.01[a]
Ethion	Gammarus lacustris	96	1.8	" "	0.01[a]
Fenchlorphos	Pimephales promelas	96	305	" "	0.01[a]
Fenitrothion	Culex tarsalis	24	0.5	cited in McLeese, 1976	0.01[a,l]
Fenthion	Simocephalus serrulatus	48	0.62	US EPA, 1972	0.06
Leptophos	Penaeus duorarum	96	1.88	Schimmel et al., 1979	0.01[a]
Malathion (maldison is recommended name)	Gammarus fasciatus	96	0.76	US EPA, 1972	0.07[e,f,g]
Methamidophos	Cyprinus carpio	96	68 mg/L	Chin & Sudderuddin, 1979	0.01[a]
Methidathion	Homarus americanus	96	10	McLeese & Metcalfe, 1979	0.01[a]
Mevinphos	Daphnia pulex	48	0.16	US EPA, 1972	0.01
Naled	Daphnia pulex	48	0.35	"	0.03
Oxydemeton-methyl	Pteronarcys california	96	35	"	0.1[b]
Parathion	Orconectes nais	96	0.04	"	0.004[h]
Parathion-ethyl	Palaemonetes pugio	96	2.8 (EC50)[i]	cited in Farr, 1977	0.01[a,j]
Parathion-methyl	Crangon septemspinosa	96	2	US EPA, 1972	0.01[a,k]
Phorate	Gammarus fasciatus	96	0.6	"	0.01[a]
Phosmet	Gammarus pseudolimnaeus	48	2.4	Julin & Sanders, 1977	0.02
Phosphamidon	Gammarus lacustris	96	2.8	"	0.3
Quinalphos	Saccobranchus fossilis	96	1.55 mg/L	Verma et al., 1978	0.01[a]
Temephos	Penaeus japonicum	72	1	Tsai, 1978	0.1
TEPP	Gammarus lacustris	96	39	US EPA, 1972	0.1[b]
Thiometon	Saccobranchus fossilis	96	11.0 mg/L	Verma et al., 1978	0.01[a]
Trichlorphon	Daphnia pulex	48	0.18	US EPA, 1972	0.02

(a) Data considered inadequate. Acute toxicity of the most sensitive species assumed to be 0.1 ug/L and used in the derivation process.

(b) Data considered inadequate, but data available indicate compound not as toxic as most organophosphate insecticides. Acute toxicity of 1.0 ug/L used to derive criterion.

(c) MATC of 0.33-0.51 ug/L for sheepshead minnows (Pimephales promelas) in lifecycle test involving reproduction (Adelman et al., 1976). Criterion is therefore sufficient to protect this species.

(d) MATC of 0.55 ug/L for brook trout (Salvelinus fontinalis) (life cycle test involving reproduction) (Allison & Hermanutz, 1977), and 0.47 ug/L for sheepshead minnows (Cyprinodon variegatus) (partial lifecycle) (Goodman et al., 1979). Criterion should be adequate for the protection of these species.

(e) Parathion is markedly synegistic with malathion (Macek, 1975).

(f) Based on the toxicity to the most senitive species and an application factor of 0.1, a criterion of 0.07 ug/L is derived. This is higher than the 30 day LC50 for Gammarus pseudolimneaus (0.023 ug/L) and higher than the no-effect level (0.008 ug/L) for this organism (US EPA, 1976j). However the criterion relates to acute exposures. For chronic exposures a criterion of 0.01 ug/L would be more appropriate.

(g) 1 ug/L is reported to have an effect on survival of carp egs (Prokopenko et al, 1976). An MATC of $> 4 < 9$ ug/L has been determined for sheepshead minnows (Cyprinodon variegatus) (Parrish et al., 1977). A no-effect level of 10.8 ug/L has been reported for lifecycle exposures to flagfish (Jordanella floridae) (Hermanutz, 1978). A criterion of 0.01 ug/L will therefore be sufficient to protect these species. The no-effect level for effect on temperature selection response of common shiners, (Notropis cornutis) is reported to lie between 0.05 and 0.25 ug/L (Domanik & Zar, 1979). A criterion of 0.01 ug/L would also protect against this effect.

(h) Malathion is markedly synergistic with parathion (Macek, 1975).

(i) The EC50 is for death or loss of equilibrium.

(j) Grass shrimp (Palaemonetes pugio) exposed to ethyl parathion at levels as low as 0.1 ug/L were more susceptible to predation (Farr, 1978). A criterion of 0.01 ug/L should protect against this.

(k) Grass shrimp (Palaemonetes pugio) exposed to methyl parathion at levels as low as 0.024 ug/L were more susceptible to predation by the gulf killifish, Fundulus grandis (Farr, 1978). A criterion of 0.01 ug/L should protect against this.

(l) 10 ug/L is the threshold of avoidance by goldfish (Scherer, 1975). The threshold for the effect of fenitrothion on swimming velocities of brook trout (Salvelinus fontinalis) is near 150 ug/L (Peterson, 1974). The lethal threshold for the lobster, Homarus americanus was reported as 0.015 ug/L (McLeese, 1974). A criterion of 0.01 ug/L would protect against these effects.

(m) A number of other reports have documented other sublethal effects, mostly with fish, which had no-effect levels much higher than the derived criteria. In these cases references to these reports have not been made.

data were considered inadequate for criteria derivation in the usual way, an LC50 of 0.1 ug/L was assumed for the most sensitive species. Using this value and an application factor of 0.1, criteria of 0.01 ug/L were derived. This assumption can apply until such time as adequate data are obtained.

For some organophosphate insecticides included in Table 9 the data were considered inadequate, but what data were available suggested that these compounds were not as toxic as most of the other organophosphate insecticides. In these cases an acute toxicity level for the most sensitive species of 1.0 ug/L was assumed. Using this value and an application factor of 0.1, criteria of 0.1 ug/L were derived.

For the organophosphate insecticides not listed in Table 9 indicating no data were found, an acute toxicity concentration for the most sensitive species of 0.1 ug/L can be assumed pending adequate data. A criterion of 0.01 ug/L is thus suggested for these compounds. As already discussed, all these criteria relate to a single acute exposure.

The US EPA (1976k) has reviewed the data for 4 organophosphates insecticides, azinphos-methyl, demeton, malathion and parathion and derived criteria of 0.01, 0.1, 0.1 and 0.04 ug/L respectively for the protection of aquatic life. These appear to be the only criteria for specific organophosphate insecticides in relation to the aquatic environment yet published. They are similar or identical to those derived in this document except for parathion for which the US EPA criterion is a factor of 10 higher.

Criteria for the protection of human health

Drinking water quality criteria for organophosphate insecticides for the protection of human health have received little attention in the past. The NAS (1977i) has reviewed in depth the relevant data for azinphos-methyl, diazinon, disulfoton, malathion, parathion, parathion-methyl and phorate and have formulated criteria. Maximum acceptable concentrations for diazinon, parathion and parathion-methyl have been recommended by Canadian authorities (Health & Welfare, Canada, 1978). The latter criteria were based on ADI data of the WHO or US EPA.

In all cases considered by NAS, criteria were based on chronic toxicity data. Chronic exposure to organophosphate insecticides in drinking water would require continuous introduction of these compounds into water supplies because of their ready degradation in this environment. Such a situation is not very likely but it does represent the most dangerous type of exposure and should therefore be considered in formulating criteria. Criteria based on acute toxicity data would lead to criterion levels sufficiently high that human health could be endangered if by chance, chronic exposures occurred. Once again conservatism must prevail where human health is concerned.

The drinking water quality criteria derived by the NAS (1977i) are summarised in Table 10.

TABLE 10. DRINKING WATER QUALITY CRITERIA FOR ORGANOPHOSPHATE INSECTICIDES DERIVED BY NAS (1977i)

COMPOUND	DERIVED CRITERION (ug/L)
Azinphos-methyl	88
Diazinon	14
Disulfoton	0.7
Malathion	160
Parathion	30
Parathion-methyl	*
Phorate	0.7

* Data considered inadequate.

For many of the organophosphate insecticides ADI levels have been recommended by WHO/FAO and are generally based on adequate long term animal studies, i.e. chronic toxicity data (Vettorazzi, 1975). Assuming 20% of the ADI is derived from water, a water quality criterion for the protection of human health could be derived using the further assumption of a 2 litre/day consumption of water for a 70 kg individual.

The NH & MRC has utilised this approach in deriving drinking water quality criteria for a number of pesticides (NH & MRC, Pesticides and Agricultural Chemicals Subcommittee, undated). They used NH & MRC (or FAO) ADI data, assumed a 2 litre/day consumption of water, but assumed a 60 kg individual consuming the water. Table 11 shows derived criteria for organophosphate insecticides using their ADI data but assuming the average weight of an individual to be 70 kg. 70 kg appears to be the most commonly accepted value and is used extensively overseas. Thus criteria derived using this value of 70 kg are proposed as being the most suitable.

A number of organophosphate insecticides have been recently found to be mutagenic in short term tests. Some animal experiments have also yielded evidence suggestive of carcinogenicity (Waters et al., 1980). These results indicate that some of these compounds require further investigation and if any are judged to be carcinogens, criteria will need to be re-established using risk estimate procedures.

SUMMARY OF PROPOSED CRITERIA FOR ORGANOPHOSPHATE INSECTICIDES

Protection of the aquatic environment - see Table 9

Protection of human health (drinking water quality) - see Table 11.

TABLE 11. DRINKING WATER QUALITY CRITERIA FOR ORGANOPHOSPHATE INSECTICIDES DERIVED FROM NH & MRC (or FAO) ADI DATA AND ASSUMING A 70 kg INDIVIDUAL WITH A 2 LITRE/DAY CONSUMPTION OF WATER AND THAT 20% OF THE ADI IS DERIVED FROM WATER.

COMPOUND	DERIVED CRITERION (ug/L)
Acephate	70
Azinphos-methyl	10
Bromophos-ethyl	30
Carbophenothion	1
Chlorfenvinphos	30
Chlorpyrifos	2
Demeton	30
Diazinon	10
Dichlorvos	30
Dimethoate	100
Disulfoton	30
Ethion	7
Ethoprophos	0.007
Fenchlorphos	70
Fenitrothion	30
Fensulfothion	20
Malathion	100
Methidation	70
Mevinphos	7
Monocrotophos	3
Omethoate	0.4
Parathion	30
Parathion-methyl	7
Pirimiphos-ethyl	1
Pirimiphos-methyl	70
Profenofos	5
Pyrazophos	7
Sulprophos	2
Temephos	300
Thiometon	30
Trichlorphon	30

3.4 CARBAMATE INSECTICIDES

General

A number of carbamate insecticides are in worldwide use. One of these, aldicarb, is reported to be the most toxic widely used pesticide (NAS, 1977j) with oral LD50 levels for animals of <1 mg/kg.

Residues in the aquatic environment

Carbamate insecticide residues are rarely detected in the aquatic environment. They, like organophosphate insecticides are relatively labile under aqueous conditions, especially under alkaline conditions, e.g. carbofuran (Siddaramappa & Seiber, 1979) and carbaryl (Wolfe et al., 1978). They have little tendency to accumulate in organisms, although low levels of residues can be found in exposed organisms (Korn, 1973). The carbamate insecticides, like the organophosphate insecticides can promote algal growth at low levels, e.g. carbofuran (Kar & Singh, 1978) and propoxur (Wurtsbaugh & Apperson, 1978).

Carbamate insecticides act on the cholinesterase enzymes and are thus determined with organophosphate insecticides in procedures which measure "cholinesterase inhibitors". Carbaryl has been identified in the River Rhine using mass spectrometry as part of the cholinesterase inhibiting compounds present in that river (Greve et al., 1972). Other more recent occurrences include carbaryl and carbofuran in water from treated rice fields (Brown et al., 1979), and propoxur in water in Canada following spraying (Gummer, 1979). The paucity of recent data on the occurrences of these compounds in the aquatic environment reflects their facile degradation.

Aquatic toxicology and criterion for the protection of the aquatic environment

The US EPA, the main originators of water quality criteria, derived no criteria for the protection of aquatic life for carbamate insecticides in its 1976 criteria document, nor are any criteria proposed (US EPA, 1979a). The only carbamate insecticides for which toxicity data for a number of organisms have been compiled are carbaryl and mexacarbate (US EPA, 1972). The organism most sensitive to carbaryl was the insect Pteronarcella badia with a 96 hour LC50 of 1.7 ug/L. On the basis of this and an application factor of 0.1 since carbaryl does not accumulate, a criterion of 0.1 ug/L is derived for the protection of the aquatic environment. Mexacarbate has an acute toxicity of 10 ug/L (96 hour LC50) for the most sensitive species, the insect Pteronarcys californica. Thus a criterion of 1 ug/L is derived for this compound using the same rationale. From the limited data for propoxur and aminocarb, it appears that these compounds would have acute toxicities for the most sensitive species similar to those for mexacarbate and carbaryl respectively. Thus criteria of 1 and 0.1 ug/L respectively are derived for these two compounds. The derived criteria for the carbamate insecticides are summarised in Table 12. They are based on acute toxicity data and therefore relate to acute exposures. Cholinesterase levels recover more quickly after exposure to carbamate insecticides in comparison with exposure to organophosphate insecticides (Pesticides Section, Australian Department of Primary Industry, 1980b).

TABLE 12. DERIVED CRITERIA FOR CARBAMATE INSECTICIDES FOR THE PROTECTION OF THE AQUATIC ENVIRONMENT

COMPOUND	DERIVED CRITERION (ug/L)
Aminocarb	0.1
Carbaryl	0.1[a]
Mexacarbate	1[b]
Propoxur	1

(a) A no-effect level of 0.21 mg/L has been reported for fathead minnows (Carlson, 1971). A criterion of 0.1 ug/L will adequately protect this species.

(b) Mexacarbate toxicity is markedly pH dependent (Mauck et al., 1977).

Consequently frequent exposure to carbamate insecticides should pose less of a hazard than organophosphate insecticides. The derived criteria therefore offer some degree of protection for chronic exposures but a lower application factor may be necessary for complete protection. For example, in a lifecycle test involving fathead minnows and carbaryl, an application factor of between 0.023 and 0.075 was experimentally determined suggesting an application factor of 0.05 to be a suitable compromise for chronic situations.

Although no criterion for carbofuran has been derived, an MATC of >15 <23 ug/L has been reported for sheepshead minnows, Cyprinodon variegatus (Parrish et al., 1977) which gives some idea of the level necessary to protect fish.

Criteria for the protection of human health

Some work has been directed at establishing drinking water quality criteria for carbamate insecticides. The NAS has reviewed the relevant data for aldicarb, carbaryl and methomyl and derived criteria for the protection of human health of 7 and 574 ug/L for aldicarb and carbaryl respectively (NAS, 1977j). The data for methomyl were considered inadequate for criterion derivation. The Canadian authorities have recommended a maximum acceptable concentration of 70 ug/L for carbaryl in drinking water (Health & Welfare, Canada, 1978). Table 13 shows the criteria for carbamate insecticides, derived from ADI data and assuming a 70 kg individual consumes 2 litres/day of water with 20% of the ADI derived from water. The ADI are of NH & MRC or FAO origin (NH & MRC, Pesticides and Agricultural Chemicals Subcommittee, undated). These derived criteria are the criteria of choice as they are derived in a manner consistent with that used for other non-carcinogenic compounds in this report.

It has been reported recently that carbamate insecticides react with nitrite under acidic conditions to produce carcinogenic nitroso derivatives (Lijinsky & Schmahl, 1978). This represents a source of carcinogenic risk if this were to occur in the environment or in the human body after ingestion (most likely in the stomach) and the proposed criteria would need to be re-evaluated, taking this factor into account.

TABLE 13. DRINKING WATER QUALITY CRITERIA FOR CARBAMATE INSECTICIDES DERIVED FROM NH & MRC (OR FAO) ADI DATA AND ASSUMING A 70 kg INDIVIDUAL WITH A 2 LITRE/DAY CONSUMPTION OF WATER AND THAT 20% OF THE ADI IS DERIVED FROM WATER

COMPOUND	DERVIED CRITERION (ug/L)
Carbaryl	70
Carbofuran	30
Methomyl	70
Pirimicarb	100
Promecarb	100
Propoxur	1000

SUMMARY OF PROPOSED CRITERIA FOR CARBAMATE INSECTICIDES

Protection of the aquatic environment - see Table 12.

Protection of human health (drinking water quality) - see Table 13.

3.5 MISCELLANEOUS INSECTICIDES

General

Apart from the three common groups already discussed, a number of other insecticides of varying chemical structures and modes of action are registered for use in Australia. Perhaps the most important are the pyrethroids, a mixture of compounds derived from natural sources, viz, flowers from certain species of plants. A number of analogues have been synthesised and their toxicity to aquatic organisms recently evaluated (Zitko et al., 1977, 1979; Jolly et al., 1978; Coats & O'Donnell-Jeffery, 1979). The situation with pyrethroids is complicated by the fact that they are formulated with the synergist, piperonyl butoxide. This compound which itself has low toxicity (Haley, 1978) is effective in enhancing the toxicity of the pyrethroids by deactivating the enzymes responsible for degrading these insecticides.

Aquatic toxicology and criteria for the protection of the aquatic environment

There appears to be no reports of these miscellaneous insecticides as water pollutants. They, therefore, have assumed very low priority as far as criteria derivation are concerned. Some data have been compiled, e.g. for allethrin, pyrethrum and rotenone (US EPA, 1972). Sufficient organisms have been tested for criteria to be established with some degree of confidence. Based on acute toxicities to the most sensitive species and an application factor of 0.1, criteria of 0.2, 0.1 and 10 ug/L are derived for allethrin, pyrethrum and rotenone respectively.

Some toxicity data for a new class of insecticide, the insect growth regulators, have been reported (McKague & Pridmore, 1978). These compounds appear to be relatively non-toxic to fish compared with other insecticides and should become very useful where pollution of the aquatic environment as a result of insect control is unavoidable.

Criteria for the protection of human health

Few attempts have as yet been made to derive criteria for the protection of human health for these compounds. The Australian Department of Health (1980) has included a few miscellaneous insecticides in their list of recommended maximum residue limits. These were based on ADI data and assumptions of a 60 kg individual with a 2 litre/day consumption of water and 20% of the intake in water (NH & MRC, Pesticides and Agricultural Chemicals Subcommittee, undated). Based on a more generally accepted figure of 70 kg for the average weight of an individual, criteria of 70, 20, 40, 300 and 1 000 ug/L are derived for bioresmethrin, chlordimeform, fenvalerate, permethrin and propargite respectively. Piperonyl butoxide can also be included with a criterion of 200 ug/L.

SUMMARY OF PROPOSED CRITERIA FOR MISCELLANEOUS INSECTICIDES

Protection of the aquatic environment

INSECTICIDE	CRITERION (ug/L)
allethrin	0.2
pyrethrum	0.1
rotenone	10

Protection of human health (drinking water quality)

INSECTICIDE	CRITERION (ug/L)
bioresmethrin	70
chlordimeform	20
fenvalerate	40
permethrin	300
propargite	1000
piperonyl butoxide	200

3.6 HERBICIDES

General

Herbicides constitute a large number of compounds of various chemical classes used in agriculture. They are widely used and a number of compounds, the chlorophenoxy herbicides 2,4-D and 2,4,5-T in particular, are used in large quantities.

A survey of pesticide usage in the Adelaide Hills showed 2,4,5-T to be most widely used herbicide in terms of numbers of properties using this chemical and acreage to which it was applied (Henderson, 1972). Australian Bureau of Statistics data show that the annual value of 2,4-D and 2,4,5-T herbicides produced during the period 1963-76 was generally greater than the total annual value of organochlorine and organophosphate insecticides produced during that time (Pesticides Section, Australian Department of Primary Industry, undated). The data for 1976-77 also show this trend with the value of 2,4-D and 2,4,5-T herbicides produced being greater than 10 million dollars (Australian Bureau of Statistics, 1978). It has been reported that 2 000 tonnes of 2,4-D and 50 to 300 tonnes of 2,4,5-T are used in Australia each year (Clark et al., 1979).

Residues in the aquatic environment

The persistence of herbicides varies widely. Some such as 2,4-D degrade rapidly while others such as diuron and picloram are more persistent. Residues have been detected in the aquatic environment due to widespread land applications for weed control and application for control of aquatic weeds and weeds on the banks of irrigation and drainage channels (Frank, 1972). In these latter instances herbicides are applied to the water or in the immediate vicinity so that the presence of residues is not unexpected.

A number of occurrences of herbicide residues in the aquatic environment as a result of land applications has been reported. Atrazine residues were determined in waters from corn growing areas in Canada (Muir et al., 1978; Frank & Sirons, 1979), and Iowa, USA (Richard et al., 1975), the herbicide being used for weed control in corn crops. Triazine herbicides, mainly atrazine, were determined in European rivers (Hormann et al., 1979).

A number of herbicides were identified in a survey of well waters in Canada. Pollution was thought to have arisen from a number of sources including accidental spillage, spray drift and run-off. The herbicides which were particularly persistent were amitrole, dinoseb and picloram (Frank et al., 1979b). Picloram residues were also determined in water samples in West Virginia, USA. Picloram was persistent and reached levels in the water considered to be detrimental to susceptible crops irrigated with the water (Dennis et al., 1977). Picloram and 2,4,5-T were determined in run-off water in Texas. Levels depended on the time between application and rain, but no damage to neighbouring crops from run-off was observed (Bovey et al., 1974).

Residues of the herbicides CNP (Suzuki et al., 1977a) and benthiocarb (Suzuki et al., 1977b) were determined in rivers and agricultural drainages in Japan, where the sources were treated rice fields. The sources of diuron determined in water and sediments, and ametryne in some sediments, from Hawaii, were sugar cane and pineapple plantations (Green et al., 1977). Chlorthal-dimethyl was identified in water samples from California (Picker et al., 1979). This compound has been detected during routine monitoring for organochlorine insecticides in waters in South Australia (Engineering and Water Supply Department, unpublished results).

The chlorophenoxy acid herbicides 2,4-D and 2,4,5-T, and dichlorprop were determined in surveys of surface waters in Canada. Part of the load of 2,4-D residues was contributed by municipal and industrial effluents (Gummer, 1979). 2,4-D was monitored in run-off following application in a corn growing area. 2,4-D was rapidly degraded in soil and concentrations in run-off were low (White et al., 1976).

2,4,5-T residues were determined in streams in Victoria as a result of aerial spraying of pine plantations for control of woody 'weeds' such as Acacia and Eucalyptus sp. (McKimm & Hopmans, 1978; Rawlinson, 1979). 2,4,5-T residues were also determined in streams leading into the Warren Reservoir in South Australia and arose as a result of spraying blackberries adjacent to these streams. However residues were not detected in the reservoir outlet indicating extensive dilution or degradation in the reservoir itself (Clark et al., 1979). The highest level determined in the streams (133 ug/L) was considerably higher than the maximum permissible level for this compound (20 ug/L), recommended by WHO (1971).

A number of herbicides used on aquatic, or irrigation and drainage channel bank weeds (so called aquatic herbicides) have been monitored in the aquatic environment as field studies of the fate and effects of these compounds. The herbicides include acrolein (Bowmer & Higgins, 1976), amitrole (Demint et al., 1970), 2,4-D (applied as amine salts), (Bartley & Hattrup, 1970; Frank et al., 1970; Schultz & Harman, 1974; Brooker, 1976a; Averitt & Gangstad, 1976; Boyle, 1980), diuron (Bowmer & Adeney, 1978), 2,2-DPA (dalapon) (Frank et al., 1979; Brooker, 1976a), endothal (Serns, 1977; Holmberg & Edwards, 1977), glyphosate (Comes et al., 1976), paraquat (Brooker & Edwards, 1973), simazine (Mauck et al., 1980) and trichloroacetic acid (Frank et al., 1970). Trifluralin residues have been monitored in an experimental ecosystem (Yockim et al., 1980) and the persistence of herbicides used in rice culture has also been investigated (Deuel et al., 1977, 1978).

In relation to aquatic herbicides, some are sufficiently persistent and resistant to degradation that their residues can also be detected in exposed organisms. Dichlobenil residues were found to persist for some time in exposed fish (Newbold, 1975). Diuron residues were sufficiently persistent to accumulate in exposed organisms, a factor which has suggested that chronic exposures may produce adverse effects due to accumulated residues (Johnson & Julin, 1974).

Residues of 2,4-D (Schultz & Harman, 1974), diquat (Dodson & Mayfield, 1979), MCPA (Hattula et al., 1978), and simazine (Dodson & Mayfield, 1979; Mauck et al., 1980) have also been determined in fish exposed to these herbicides but the levels were insufficiently different from those of the surrounding water indicating little or no tendency to concentrate. For those which tend to concentrate, e.g. diuron, the bioaccumulation factors are very low compared with those for some of the organochlorine insecticides and therefore herbicides do not represent such a great hazard. Residues in aquatic organisms as a result of land applications of herbicides are considered to be negligible as associated contamination of nearby waters is generally low.

Aquatic toxicology and criterion for the protection of the aquatic environment

The generally low mammalian toxicity of herbicides (their toxicity is directed towards plants), their ready degradation with little tendency to accumulate in organisms, has resulted in their being considered of minor importance as water pollutants. Toxicity data are not readily available and no attention has as yet been paid to deriving water quality criteria for these compounds to protect the aquatic environment. However their frequent occurrences in the aquatic environment and the fact that a number are applied directly to water for the control of aquatic weeds suggest that more attention be paid to criteria for these compounds.

Some acute toxicity data for herbicides and aquatic organisms has been compiled (US EPA, 1972) and their effects on the aquatic environment discussed (Mullison, 1970). Later data are shown in Table 14. Only a few species of aquatic organisms have been tested in most cases and the data are considered inadequate for criteria derivation since it is impossible to ascertain whether those tested include the more sensitive species. The diversity of chemical classes and the wide toxicity ranges make it impossible to generalise about the identity of the more sensitive species, c.f. organophosphate insecticides. These factors also make it impossible to make generalisations about the toxicities of herbicides not included in these compilations. As with pesticides, herbicide resistant organisms are being reported (Fabacher & Chambers, 1974).

In relation to chronic toxicity of herbicides, information available for trifluralin indicates an MATC much less than the LC50 value (Parrish et al., 1978) which suggests that the accumulated residues have an adverse effect. Therefore for the herbicides which have some tendency to accumulate, criteria derived from acute toxicity data cannot be used for chronic exposure situations. Although criteria cannot be derived the toxicity data are presented so that some estimate of short term effects to particular types of organisms can be made (Table 14).

TABLE 14. TOXICITY DATA FOR HERBICIDES IN ADDITION TO THAT COMPILED BY THE US EPA (1972)

HERBICIDE	ORGANISM	ACUTE TOXICITY mg/L	hours	NO-EFFECT LEVEL	REFERENCE
Ametryne	*Gambusia affinis*	>10	48		Darwazeh & Mulla, 1974
Amitrole	*Cyclops vernalis*	22.1	96		Robertson & Bunting, 1976
	Gambusia affinis	2100	96		Johnson, 1978
	Adelotus brevis[a]	3000	96		Johnson, 1976
Analap	*Ictalurus punctatus*	>10	48		McCorkle *et al*., 1977
Asulam	*Ctenopharyngodon idella*	>10000	96		Tooby *et al*., 1980
Atrazine	*Daphnia pulex*			<1 mg/L	Schober & Lampert, 1977
	Gambusia affinis	>10	48		Darwazeh & Mulla, 1974
Bensulide	*Ictalurus punctatus*	0.379	96		McCorkle *et al*., 1977
Chloramben	*Ictalurus punctatus*	>10	48		" "
Cyantryn	*Daphnia longispina*	15.4	96		Cited in Scorgie & Cooke, 1979
	Daphnia pulex			<0.2 mg/L	Scorgie & Cooke, 1979
	Ctenopharyngodon idella	58	96		Tooby *et al*., 1980
	Lymnaea peregra			<0.2 mg/L	Scorgie & Cooke, 1979
	Rana temporaria	30	96		Cited in Scorgie & Cooke, 1979
2,4-D[c] (free acid)	*Anabaenopsis raciborskii*			<10 ug/L[b]	Das & Singh, 1977
	Cyclops vernalis	8.82	96		Robertson & Bunting, 1976
	Daphnia magna	135	48		Benijts-Claus & Persoone, 1975
	Cancer magister			<1000 ug/L (MATC)	Caldwell *et al* 1979
	Ictalurus punctatus	>10	48		McCorkle *et al*., 1978
	Morone saxatilis	70.1	96	0.1 mg/L[d]	Rehwoldt *et al*., 1977
	Fundulus diaphanus	26.7	96	0.1 mg/L[d]	"
	Pumpkinseed	94.6	96	0.1 mg/L[d]	"
	Roccus americanus	40.0	96	0.1 mg/L[d]	"
	Anguilla rostrata	300.6	96	0.1 mg/L[d]	"
	Cyprinus carpio	96.5	96	0.1 mg/L[d]	"
	Lebistes reticulatus	70.7	96	0.1 mg/L[e]	"
	Brachydanio rerio	160	96		Benijts-Claus & Persoone, 1975
2,4-D[c] (Alkanolamine salt)	*Ictalurus punctatus*	>10	48		McCorkle *et al*., 1978
	Cyclops vernalis	142.0	96		Robertson & Bunting, 1976
	" "	54.8 (expressed as acid equiv.)	96		"
2,4-D[c] (DMA salt)	Ictalurus punctatus	>10	48		McCorkle *et al*., 1978
	Gambusia affinis	405	96		Johnson, 1978
2,4-D amine[c] (amine not specified)	Ctenopharyngodon idella	1313	96		Tooby *et al*., 1980
	Adelotus brevis[a]	200	96		Johnson, 1976
	Limnodynastes peroni[a]	100	96		"
	Bufo marinus (tadpoles)	288	96		"
2,2-DPA	*Ictalurus punctatus*	>10	48		McCorkle *et al*., 1978
	Gambusia affinis	19100	96		Johnson, 1978
	Ctenopharyngodon idella	>30000	96		Tooby *et al*., 1980
	Adelotus brevis[a]	4200	96		Johnson, 1976
	Limnodynastes peroni[a]	2000	96		"
Dicamba	*Gambusia affinis*	465	96		Johnson, 1978
	Adelotus brevis[a]	185	96		Johnson, 1976
	Limnodynastes peroni[a]	106	96		"
DEF	*Cancer magister*			>0.95,< 6.9 ug/L (MATC)	Caldwell *et al*., 1979
Dichlobenil	*Ctenypharyngodon idella*	9.4	96		Tooby et al., 1980
Dinoseb (DNBP)	*Ictalurus punctatus*	0.118	96		McCorkle *et al*., 1978
Diquat	*Daphnia magna* (adults)	3	48		Benijts-Claus & Persoone, 1975
	Daphnia magna (new born)	0.6	48		" "
	Daphnia pulex (adults	0.4	48		" "
	Daphnia pulex (new born)	0.3	48		" "
	Ctenypharyngodon idella	1718	96		Tooby *et al*., 1980
	Brachydanio rerio	23.5	96		Benijts-Claus & Persoone, 1975
Diuron	*Gammarus lacustris*	0.38[f]	48		Cited in Johnson & Julin, 1974
	Daphnids			< 0.05 mg/L[g]	" "
	Carp			< 0.01 mg/L[h]	" "
	Striped bass	0.5[i]	96		" "
	Ictalurus punctatus	>10	48		McCorkle et al., 1978
	Ctenypharyngodon idella	31	96		Tooby *et al*., 1980
EPTC	*Ictalurus punctatus*	>10	48		McCorkle *et al*., 1978
Fenoprop	*Gambusia affinis*	0.35	96		Johnson, 1978
	Adelotus brevis[a]	54	96		Johnson, 1976
	Limnodynastes peroni[a]	22	96		"
	Bufo marinus (tadpoles)	34	96		"
Fluometuron	*Ictalurus punctatus*	>10	48		McCorkle *et al*., 1978
Glyphosate[j]	*Chironomus plumosus*	55	96		Folmar *et al*., 1979
	Salmo gairdneri	140	96		"
	Pimephales promelas	97	96		"
	Ictalurus punctatus	130	96		"
	Lepomis macrochirus	140	96		"
	Ctenopharyngodon idella	15	96		Tooby *et al*., 1980
MCPA-sodium salt	*Salmo trutta*	147	?		Hattula *et al*., 1978b
Metribuzin	*Ictalurus punctatus*	>10	48		McCorkle *et al*., 1978

TABLE 14. (Cont'd)

HERBICIDE	ORGANISM	ACUTE TOXICITY		NO-EFFECT LEVEL	REFERENCE
		mg/L	hours		
Molinate	Palaemonetes kadiakensis	15.90	96		Chaiyarach et al., 1975
	Procambarus simulans	21.80	96		"
	Rangia cuneata	197	96		"
	Gambusia affinis	16.40	96		"
	Ictalurus punctatus	33.24	96		Brown et al., 1979
Monuron	Ictalurus punctatus	>10	48		McCorkle et al., 1978
Nitralin	Ictalurus punctatus	>10	48		McCorkle et al., 1978
Paraquat	Asellus meridianus	1.3	Incipient		Brooker & Edwards, 1974
	Cloeon dipterum	29.00	"		" "
	Sigara sp.	5.00	"		" "
	Daphnia hyalina	2.50	"		" "
	Psectrocladius sp.	>100	"		" "
	Daphnia magna (adults)	3.7	48		Benijts-Claus & Persoone, 1975
	Daphnia magna (new born)	2.8	48		" "
	Daphnia pulex (adults)	2.4	48		" "
	Daphnia pulex (new born)	1.3	48		" "
	Gambusia affinis	604	96		Johnson, 1978
	Brachydanio rerio	40	96		Benijts-Claus & Persoone, 1975
	Ctenopharyngodon idella	66	96		Tooby et al., 1980
	Adelotus brevis[a]	262	96		Johnson, 1976
	Limnodynastes peroni[a]	100	96		"
Picloram	Gambusia affinis	120	96		Johnson, 1978
	Adelotus brevis[a]	95	96		Johnson, 1976
	Limnodynastes peroni[a]	105	96		"
Prometon	Gambusia affinis	>10	48		Darwazeh & Mulla, 1974
Prometryne	Gambusia affinis	>10	48		" "
Propanil	Palaemonetes kadiakensis	19.00	96		Chaiyarach et al., 1975
	Procambarus simulans	28.30	96		"
	Cancer magister			≥80,< 1700 ug/L (MATC)	Caldwell et al., 1979
	Gambusia affinis	9.46	96		Chaiyarach et al., 1975
	Rangia cuneata	132			"
	Ictalurus punctatus	3.796	96		McCorkle et al., 1978
	Ictalurus punctatus	7.94	96		Brown et al., 1979
Propazine	Gambusia affinis	>10	48		Darwazeh & Mulla, 1974
Simazine	Gambusia affinis	>10	48		" "
2,4,5-T (free acid)	Morone saxatilis	14.6	96	0.1 mg/L[c]	Rehwoldt et al., 1977
	Fundulus diaphanus	17.4	96	0.1 mg/L[c]	"
	Pumpkinseed	20.0	96	0.1 mg/L[c]	"
	Roccus americanus	16.4	96	0.1 mg/L[c]	"
	Anguilla rostrata	43.7	96	0.1 mg/L[c]	
	Cyprinus carpio	41.1	96	0.1 mg/L[c]	"
	Lebistes reticulatus	28.1	96	0.1 mg/L[e]	"
	Ictalurus punctatus	10	48		McCorkle, 1978
2,4,5-T amine (amine not specified)	Gambusia affinis	1600	96		Johnson, 1978
	Adelotus brevis[a]	200	96		Johnson, 1976
	Limnodynastes peroni[a]	169	96		"
	Bufo marinus	340	96		"
Terbutryne	Ctenopharyngodon idella	5.8	96		Tooby et al., 1980
Trifluralin	Cancer magister			≥26,< 220 ug/L (MATC)	Caldwell et al., 1979
	Cyprinodon variegatus	0.19	96	>1.3,< 4.8 ug/L (MATC)	Parrish et al., 1978
	Ictalurus punctatus	0.417	96		McCorkle et al., 1978

(a) Australian organisms (frog tadpoles).

(b) 10 ug/L stimulated growth of this alga.

(c) The toxicity of 2,4-D depends on the nature of the ester or salt (Meehan et al., 1974; Caldwell et al., 1979). The acid, although formed readily by hydrolysis, is the least toxic (Meehan et al., 1974).

(d) 10 month exposure.

(e) 10 month exposure. Breeding occurred with no adverse effects.

(f) Most sensitive invertebrate.

(g) 0.05 mg/L decreased reproduction efficiency.

(h) 0.1 mg/L had pathological effects on carp.

(i) Most sensitive fish.

(j) The surfactant used in the commercial formulation was substantially more toxic than the glyphosate and accounted almost totally for the toxicity of formulations.

The concentrations of herbicides used for aquatic weed control are generally considered to be harmless to aquatic organisms (Brooker & Edwards, 1975). However more recent data on sublethal effects shows this to be far from true in a number of cases. Aquatic herbicides therefore warrant more attention in relation to water quality criteria for the protection of the aquatic environment. Also with the application of aquatic herbicides an important part of the aquatic environment is destroyed (the aquatic plants). It is realised that the total environment cannot be preserved, but important non-target organisms should be protected.

The effects of aquatic herbicides on the aquatic environment have been reviewed (Newbold, 1975; Brooker & Edwards, 1975; Pesticides Section, Australian Department of Primary Industry, 1979). The use of diuron as an aquatic herbicide has also been specifically reviewed (Johnson & Julin, 1974). The major aquatic herbicides appear to be aromatic solvents, acrolein, asulam, chlorthiamid, 2,4-D, 2,4-DPA (dalapon), dichlobenil, diquat, endothal, maleic hydrazide, paraquat, 2,4,5-T, the triazines (e.g. simazine and terbutryne) and the ureas (e.g. diuron) (Newbold, 1975; Brooker & Edwards, 1975; Pesticides Section, Australian Department of Primary Industry, 1979).

The generalisation that aquatic herbicides have no effect on non-target organisms appears to be based on gross observations in the short term, e.g. no fish deaths. On closer examination the data are inconclusive and appear to depend on the herbicide and the severity of the effect monitored.

Endothal (Holmberg & Edwards, 1976; Serns, 1977), 2,4-D (dimethylamine salt) (Brooker, 1976a; Boyle, 1980), and simazine (Mauck et al., 1980) were reported to have no adverse effects on fish at the recommended dose rate. Diquat and dichlobenil at the recommended application rates were reported to have no effect on amphibians (Cooke, 1977). Simazine (Mauck et al., 1980), 2,4-D and 2,2-DPA (Brooker, 1976b) had no marked adverse effects in field experiments at recommended application rates. However a mixture of 2,4-D and 2,4,5-T, a common herbicidal formulation, was toxic to invertebrates at levels as low as 1 mg/L (Seuge et al., 1978), which is of the order of recommended applications rates.

Diuron had no effect on fish at recommended application rates as evidenced by mortality. Pathological changes were observed at these levels however, indicating the probability of long-term effects. The recommended rates are also near LC50 levels for invertebrates (Johnson & Julin, 1974). Kersting (1975a,b) has reported sublethal effects at recommended application rates for diuron which would impair the continued survival of Daphnia populations.

Cyanatryn had effects on Daphnia and tadpoles at the recommended application rates (Scorgie & Cooke, 1979). Similarly atrazine had sublethal effects (impairment of reproduction) on Daphnia at levels as low as 1-2 mg/L, (Jordan et al., 1977). Paraquat was lethal to the isopod Asellus meridianus at levels less than recommended dose rates (Brooker & Edwards, 1974). The failure of these results to be repeated with paraquat in field trials probably indicates rapid absorption of the herbicide, thereby reducing the period of exposure to toxic doses. Fish appear to be unaffected by field applications of paraquat at 1 mg/L (Brooker & Edwards, 1974) although this level would have some effect on Daphnia (Benijts-Claus & Persoone, 1975). Diquat applied at recommended rates would also be toxic to Daphnia (Benijts-Claus & Persoone, 1975). Acrolein is toxic to fish at the recommended application rates (Pesticides Section, Australian Department of Primary Industry, 1979).

Other secondary effects of aquatic weed control must also be considered in predicting the effects of aquatic herbicides, e.g. elimination of habitats and elimination of habitats or substrates for food organisms (Newbold, 1975; Brooker & Edwards, 1975; Pesticides Section, Australian Department of Primary Industry, 1979). The most serious secondary effect in the short term would be deoxygenation associated with the decay of dead plant material (Brooker, 1974; Newbold, 1975; Brooker & Edwards, 1975).

Criteria for aquatic herbicides in order to protect non-target organisms cannot be yet determined with confidence. Whether the recommended application doses will harm non-target organisms will depend on a number of factors. Each case must be considered on its own merits. In irrigation channels aquatic life may be considered adventitious and therefore expendable (Pesticides Section, Australian Department of Primary Industry, 1979), but this may not always be so.

Criteria for the protection of human health

The generally low mammalian toxicity of herbicides has resulted in little attention being paid to drinking water quality criteria for these compounds. Although herbicides generally have low mammalian toxicity, some such as DNOC (also used as an insecticide), paraquat and pentachlorophenol (used also for other purposes and discussed elsewhere) have been responsible for human death (Barnes, 1976). The high toxicity of some compounds is sufficient to warrant derivation of criteria.

The US EPA (1976l) has derived drinking water quality criteria for 2,4-D and fenoprop (2,4,5-TP, silvex) of 100 and 10 ug/L respectively. The NAS (1977k) has reviewed the data for a number of herbicides and derived criteria for the protection of human health (Table 15). Canadian authorities have set maximum acceptable concentrations of 100 and 10 ug/L for 2,4-D and fenoprop respectively (Health & Welfare, Canada, 1978), presumably in line with the US EPA criteria. The Australian Department of Health (1980) has recommended maximum residue limits for a number of herbicides in water based on ADI data (NH & MRC, Pesticides & Agricultural Chemicals Subcommittee, undated). Using the same ADI data but assuming 70 kg as the weight of an average individual consuming 2 litre/day of water with 20% of the intake from the water, the criteria in Table 16 are derived. These are proposed as the more widely accepted value of 70 kg as the average weight of an individual is used in the derivation.

TABLE 15. DRINKING WATER QUALITY CRITERIA FOR HERBICIDES DERIVED BY NAS (1977k)

HERBICIDE	DERIVED CRITERION (ug/L)
Acrolein	- *
Alachlor	700
Atrazine	150
Benfluralin (benefin)	- *
Bromacil	86
Butachlor	70
Chloramben	1750
Cyanazine	- *
2,4-D	90
Dicamba	9
Fenoprop (2,4,5-TP)	5.25
MCPA	9
Nitralin	- *
Paraquat	60
Propachlor	700
Propanil	140
Propazine	320
Simazine	1505
2,4,5-T	700
Trifluralin	700

* No criterion derived - insufficient data.

A number of herbicides have been tested for their mutagenicity. MCPA and its metabolites were negative in the Ames test (Rasanen et al., 1977). A trifluralin containing herbicide was found to be mutagenic to mouse chromosomes (Nehez et al., 1979). A DNOC containing herbicide was also mutagenic in some systems (Nehez et al., 1978a,b). Several other herbicides are also mutagenic (Fishbein, 1972; Waters et al., 1980). The herbicide oryzalin has recently been blamed for birth defects in children (Dickson, 1979). Thus further toxicological testing of some herbicides needs to be performed and criteria re-evaluated if more conclusive data regarding mutagenicity are obtained.

2,4-D and 2,4,5-T

The chlorophenoxy herbicides, especially 2,4-D and 2,4,5-T are sufficiently important to warrant a separate discussion and rationale concerning the derivation of criteria. The level of the impurity 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD or dioxin) in 2,4,5-T and fenoprop must be carefully considered as it modifies the toxicities of these herbicides. The controversy over 2,4,5-T and TCDD has been well documented and mainly concerns exposures to Agent Orange, a 50:50 mixture of the n-butyl esters of 2,4-D and 2,4,5-T used during the Vietnam war. It also includes to a lesser extent exposures to 2,4,5-T during agricultural and forestry operations. Concern regarding the possible adverse effects of 2,4,5-T has resulted in it being banned from a number of uses in the USA (Cookson, 1979).

TABLE 16. DRINKING WATER QUALITY CRITERIA FOR HERBICIDES DERIVED FROM NH & MRC (OR FAO) ADI DATA AND ASSUMING A 70 kg INDIVIDUAL WITH A 2 LITRE/DAY CONSUMPTION OF WATER AND THAT 20% OF THE ADI IS DERIVED FROM WATER

HERBICIDE	DERIVED CRITERION (ug/L)
Alachlor	10
Asulam	300
Barban	300
Bentazone	500
Bromacil	700
Bromoxynil octanoate	300
Butachlor	300
Chloroxuron	300
2,4-D	2000[a]
2,4,5-T	- [b]
Dicamba	300
Dichlobenil	20
Dichlofop-methyl	3
Difenzoquat	200
Dinitramine	700
Diquat	50
Endothal	700
EPTC	70
Fenoprop	20
Flamprop-methyl	1
Fluometuron	100
Fosamine ammonium	3000
Glyphosate	500
Hexazinone	700
Metolachlor	50
Metribuzin	40
Nitralin	1000
Oryzalin	70
Paraquat	10
Pendimethalin	1000
Perfluidone	20
Phenisopham	1
Picloram	10000
Thiobencarb	50
Tiocarbazil	500
Trifluralin	500

(a) Substantially greater than that recommended by NH & MRC and US EPA.

(b) No ADI given. NH & MRC maximum residue limit of 20 ug/L is the detection limit of the analytical method for 2,4,5-T.

TCDD is formed in the manufacture of 2,4,5-T (and fenoprop) by the secondary reaction of condensation of two molecules of 2,4,5-trichlorophenol. This may also occur during the preparation of 2,4,5-trichlorophenol (IARC, 1977c; Jenkins, 1978). Since 2,4-D is not prepared from 2,4,5-trichlorophenol but from 2,4-dichlorophenol TCDD is not a contaminant of 2,4-D.

2,4-D is rapidly degraded in the aquatic environment (Schultz & Harman, 1974; Watson, 1977) and thus residues are not expected to be a major problem. Nevertheless drinking water quality criteria for 2,4-D must be derived since it is used in such large quantities (estimated at 2 000 tonnes per year in Australia (Clark et al., 1979)) and residues have been occasionally determined in the aquatic environment.

2,4-D is a weak mutagen or gives negative results in a number of short-term tests for mutagenicity. This is true for the other chlorophenoxy acid herbicides other than 2,4,5-T (Seiler, 1978). The carcinogenicity data are inconclusive for 2,4-D (IARC, 1977b; Seiler, 1978). It may be a very weak carcinogen but more testing is needed. Based on the maximum no-effect level in animal experiments and a safety factor of 1 000, a criterion for the protection of human health of 90 ug/L has been derived for 2,4-D (NAS, 1977l). This criterion was derived assuming a 70 kg individual with a 2 litre/day consumption of water and 20% of the intake from the water. A safety factor of 5 000 is recommended for possible carcinogens as discussed earlier. However 2,4-D is only very weakly carcinogenic (if at all) and the safety factor of 1 000 appears to be a reasonable compromise. The criterion of 90 ug/L is much lower than the value (2 000 ug/L) calculated from NH & MRC ADI data. The value of 90 ug/L is however, more appropriate as it is derived in manner consistent with that used for other compounds in this document.

The data for 2,4,5-T and TCDD can be divided into two types - laboratory and epidemiolgical. In both it is sometimes difficult to differentiate between effects due to 2,4,5-T and those due to TCDD. In earlier experiments the levels of TCDD in the 2,4,5-T were unknown, but could conceivably have been in the mg/kg range. TCDD is an extremely powerful teratogen; it is also carcinogenic and extremely toxic in acute exposures, death aften occurring some time after exposure (IARC, 1977a, US EPA, 1978i). TCDD could have been responsible for some effects attributed to 2,4,5-T. Similarly for human exposures, e.g. Agent Orange in Vietnam, some effects could have been due to the relatively high TCDD levels at that time (up to 30 mg/kg prior to 1965 (IARC, 1977c)).

The properties of TCDD have been well documented (IARC, 1977a; Ramel, 1978; Reggiani, 1978; US EPA, 1978i). TCDD is considered teratogenic, mutagenic and carcinogenic. A number of cases of workers and people in the vicinity of chemical plants being exposed to TCDD as a result of accidents have been reported. Effects such as chloracne were readily apparent but carcinogenic or teratogenic effects could not be demonstrated (IARC, 1977a, Ramel, 1978, Reggiani; 1978; US EPA, 1978i). TCDD is not considered a sufficiently important drinking water contaminant to warrant derivation of a criterion for this water use at this time. The US EPA has made risk estimates for exposure to TCDD in drinking water. These risks also included estimated exposures from other sources, viz, eating fish and shellfish which had been raised in the water (US EPA, 1978i).

As already discussed it is difficult to differentiate between the effects of 2,4,5-T and TCDD since these compounds are usually associated with each other. Although prior to 1965 levels of up to 30 mg/kg of TCDD were present in 2,4,5-T, the current levels are less than 0.05 mg/kg (IARC, 1977c). UK (Anonymous, 1979) and Australian (Lee, 1979) authorities allow the use of 2,4,5-T if TCDD levels are less than 0.1 mg/kg. It is considered that the best estimate of a criterion for 2,4,5-T necessary to protect human health relates to 2,4,5-T containing less than 0.1 mg/kg TCDD and is therefore derived from toxicity data for 2,4,5-T with less than this amount of TCDD.

The teratogenicity, mutagenicity and carcinogenicity of 2,4,5-T have been reviewed (IARC, 1977c; NAS, 1977m; Ramel, 1978; Grant, 1979). Although the data are contradictory, 2,4,5-T with TCDD levels less than 0.1 mg/kg appears to be weakly mutagenic and teratogenic in laboratory experiments. The carcinogenicity of 2,4,5-T is still unclear. Thus 2,4,5-T even in its present relatively pure state, has important toxic properties which do not appear attributable to TCDD contamination. It therefore also represents a potential threat to man, the major effect expected being teratogenesis (from the laboratory data).

A number of epidemiological studies of birth defects in areas subject to heavy 2,4,5-T usage have been made (NZ Dept. of Public Health, 1977; Consultative Council, Victoria, 1978; Field & Kerr, 1979; Nelson et al., 1979; US EPA, 1979d), and produced inconclusive results as to the effects of low level 2,4,5-T exposures on man. Involvement of 2,4,5-T in medical problems of soldiers who served in Vietnam has also been suggested. Numerous effects, including birth defects (which would indicate mutagenic activity) have been attributed to 2,4,5-T. However the evidence for 2,4,5-T as a cause is far from convincing.

The NAS has derived a criterion of 700 ug/L for 2,4,5-T in drinking water after assessment of the available data (NAS, 1977m). The maximum no-effect level was 10 mg/kg/day in laboratory experiments, which with a safety factor of 100, produced an ADI of 0.1 mg/kg/day. A more recent report has shown 10 mg/kg/day to have minimal effects in rats whereas 3 mg/kg/day had no effect (Kociba et al., 1979). A no-effect level of 3 mg/kg/day is thus warranted in the derivation process. Since 2,4,5-T may be carcinogenic, even with very low TCDD levels, a more conservative safety factor of 5 000 is also recommended until more information is available. A more conservative safety factor of 5 000 is also suggested by the serious implications of birth defects, although the data are still inconclusive at this stage. Using this safety factor, a no-effect level of 3 mg/kg/day and the usual assumptions (a 70 kg individual consuming 2 litres of water per day with 20% of the exposure from the water), a criterion for the protection of human health of 4.2 ug/L is derived. A criterion of 4 ug/L for 2,4,5-T is therefore proposed for drinking water.

Criteria for agricultural uses

The presence of herbicide residues in water has frequently been reported, and reports of crop damage as a result of these residues have also been made, e.g. damage to tomatoes in England due to herbicide contaminated irrigation water has been reported. 2,3,6-TBA was the herbicide responsible and reached the irrigation water as a result of discharge of factory effluent (Hills, 1973; Williams et al., 1977). Although damage to crops by aquatic herbicides used for weed control in irrigation channels has not been reported, these herbicides represent a potential hazard due to locations in which they are used. Thus it is important that criteria in relation to the agricultural uses of water, and in particular irrigation water and aquatic herbicides be considered.

Although criteria to protect crops are of some importance, the derivation of a criterion or safe level for each herbicide to protect all crops does not appear warranted. An attempt was made to derive safe levels for herbicides for individuals crops. However this was unsuccessful as some herbicides at low levels increase crop yields. As a result reference is made to original publications for toxicity data.

A number of reports have been published recently detailing the effects of aquatic herbicides on crops (see Table 17), and grasses (Hiltibran & Turgeon, 1977). Additional data have been reported by Hart (1974b) and Bill & Graham (1970). Toxicity data obtained from laboratory experiments aimed at determining the toxicity of herbicides to plants has, however not been considered in the preparation of this report.

In relation to another aspect of the agricultural use of water, the effect of herbicides on livestock, little information is available. The effects of the chlorophenoxy herbicides on sheep and cattle have been reported (Clark et al., 1975). Although anorexia was observed, this occurred at levels much greater than those likely to be encountered on pastures irrigated with water containing herbicide residues. Residues in exposed animals were relatively low and rapidly decreased on termination of exposure. Thus chlorphenoxy herbicide residues in water as a result of normal agricultural practice would not be expected to pose a threat to the health of farm animals. Negligible residues of such herbicides would also be expected in the meat of exposed animals.

TABLE 17. CROPS ON WHICH THE EFFECTS OF HERBICIDES IN IRRIGATION WATER HAVE BEEN INVESTIGATED

HERBICIDE*	CROPS	REFERENCE
Chlorfenac	Dwarf corn, field beans, grain sorghum, potatoes, soybeans, sugarbeets	Bruns et al., 1976
"	Alfalfa, corn, cotton, soybeans, sugarbeets	Bruns et al., 1972
2,4-D	Alfalfa, cucumbers, field beans grain sorghum, potatoes, sweet peppers	Bruns et al., 1974
"	Corn, soybeans, sugarbeets	Bruns et al., 1973
Dicamba	Cotton	Bruns et al., 1972
Dichlobenil	Alfalfa, corn, soybeans sugarbeets	Bruns et al., 1972
Endothal	Corn, cotton, soybeans, sugarbeets	Bruns et al., 1972
"	Sugarbeets	Bruns et al., 1972
Fenoprop (Silvex)	Corn, soybeans, sugarbeets	Bruns et al., 1973
Glyphosate	Alfalfa, field beans, grain sorghum, squash, sugarbeets, tomatoes	Bruns & Kelley, 1975; Comes & Kelley, 1979
Picloram	Cotton	Bruns et al., 1972

* Most herbicides were applied as their alkali metal or amine salts, or esters where appropriate.

SUMMARY OF PROPOSED CRITERIA FOR HERBICIDES

Protection of the aquatic environment - insufficient data. Toxicity data are presented in Table 14.

Protection of human health (drinking water quality) -

HERBICIDE	CRITERION (ug/L)
2,4-D	90
2,4,5-T	4

For other herbicides see Table 16, or if not listed, those derived by NAS (Table 15) would suffice.

Criteria for agricultural uses - insufficient data. Crops on which effects of herbicides in irrigation water have been investigated are presented in Table 17.

3.7 FUNGICIDES

General

Fungicides represent another group of pesticides covering a wide range of chemical types. These are not used in very large quantities and are generally readily degradable in water, e.g. thiram (Odeyemi, 1979). They are not applied to the aquatic environment and therefore do not represent water pollutants of any great importance. There are two exceptions to this generalisation however, hexachlorobenzene (HCB) and pentachloronitrobenzene (PCNB). HCB is particularly persistent, accumulates in exposed organisms and has been frequently determined in the aquatic environment as a result. Although PCNB would also be expected to be reasonably stable, residues do not appear to have been detected in the aquatic environement.

HCB is used as a fungicide chiefly on seed wheat. Human poisonings with fatalities have been reported due to consumption of wheat treated with HCB. Chronic sub lethal exposures produced the disorder porphyria cutanea tarda (Courtney, 1979). Its use as a seed dressing has been banned in Australia since 1972, but occasional high residue levels are still determined and are causing concern (Pesticides Section, Australian Department of Primary Industry, 1975).

HCB is also produced as a waste in the manufacture of other chemicals. In some instances the disposal of these wastes has been established as the source of HCB in the environment. HCB is a contaminant of the herbicide chlorthal-dimethyl and the fungicide PCNB. Levels of HCB in these pesticides were in the order of 10%, but have now been reduced to less than 5% (Courtney, 1979).

Further details on HCB and its occurrences in the environment can be found in the reviews by Courtney (1979), Fishbein (1979a,c), (in particular industrial sources of HCB) and NAS (1977o).

Residues in the environment

HCB is the only fungicide determined in the aquatic environment with any frequency. Recent determinations outside Australia are shown in Table 18.

TABLE 18. RECENT DETERMINATIONS OF THE FUNGICIDE, HCB, IN THE AQUATIC ENVIRONMENT

LOCATION	DETERMINED IN	REFERENCE
CANADA (Gulf St. Lawrence)	seals	Rosewell et al., 1979
(Lake Ontario)	fish (salmonids)	Niimi, 1979
(Lake Ontario)	fish, herring gull eggs	Norstrom et al., 1978
CZECHOSLOVAKIA (R. Danube)	fish, water	Sackmauerova et al, 1977
JAPAN	sewage, effluents	Morita, 1977
NETHERLANDS	mussels	Quirijns et al., 1979
	porpoise	Duinker & Hillebrand, 1977
NORWAY	fish	Ofstad et al., 1978
USA (Mississippi River)	fish, organisms	Laska et al., 1976
MEDITERRANEAN SEA	fish, organisms	Amico et al., 1979

HCB has frequently been detected in the Australian environment. Occurrences include fish (Neuhaus et al., 1973), water rats from the Murrumbidgee irrigation area of NSW (Olsen & Settle, 1979), various animals from Tasmania (Bloom et al., 1979), birds from the Northern Territory (Best, 1973), and food samples (eggs and animal fat) (Brady & Siyali, 1972). HCB residues have also been determined in the human population of Australia. Residues were determined in fat (Brady & Siyali, 1972; Siyali & Simson, 1973), blood (Siyali, 1972; Siyali & Simpson, 1973; Siyali et al., 1974), and milk (Newton & Greene, 1972; Siyali, 1973, Miller & Fox, 1973; Stacey & Thomas, 1975).

Aquatic toxicology and criteria for the protection of the aquatic environment

Acute toxicity data for some fungicides have been compiled by the US EPA (1972). Little subsequent data have been published. Canton (1976) determined the acute toxicities of benomyl, thiophanate-methyl and carbendazim (BCM) for various aquatic organisms (Table 19). The toxicity data for fungicides are generally inadequate for the derivation of criteria for the protection of the aquatic environment. They can however be used to estimate the sensitivity of various organisms to some fungicides in short term exposures. In one long term study of the effects of carbendazim on Daphnia, an ecological limit of 16 ug/L was proposed (Canton, 1976).

TABLE 19. ACUTE TOXICITIES OF BENOMYL, THIOPHANATE-METHYL AND CARBENDAZIM (BCM) FOR AQUATIC ORGANISMS (Taken from Canton, 1976).

FUNGICIDE	ORGANISM	48 HOUR LC50 (mg/L)
Benomyl	*Chlorella pyrenoidosa*	1.4*
	Daphnia magna	0.64
	Lebistes reticulatus	3.4
	Salmo gairdneri	0.48
Thiophanate-methyl	*Chlorella pyrenoidosa*	8.5*
	Daphnia magna	16
	Lebistes reticulatus	130
	Salmo gairdneri	7.8
Carbendazim	*Chlorella pyrenoidosa*	0.34*
	Daphnia magna	0.46
	Lebistes reticulatus	-
	Salmo gairdneri	1.8

* EC50 levels - 50% inhibition of growth.

Bioaccumulation and the production of fish suitable for human consumption

The maximum residue limit for HCB in meat fat has been set at 1 mg/kg in Australia (NH & MRC, 1978). Extending this to fish, and using the bioaccumulation factor of 44 000 cited by Fishbein (1979a,c), a criterion of 0.02 ug/L is derived which will provide for the production of fish suitable for human consumption.

Criteria for the protection of human health

Some attention has been paid to drinking water quality criteria for fungicides. The NAS has reviewed the relevant data and derived criteria for a number of fungicides (NAS, 1977n). These are shown in Table 20.

TABLE 20. DRINKING WATER QUALITY CRITERIA FOR FUNGICIDES DERIVED BY NAS (1977n).

FUNGICIDE	DRINKING WATER QUALITY CRITERION (mg/L)
Maneb	0.035
Zineb	0.035
Ziram	0.088
Thiram	0.035
Captan	0.35
Folpet	1.1
HCB	0.007
PCNB	-

Since the decomposition product of the ethylene-bis-dithiocarbamate fungicides (zineb, maneb and nabam) is the carcinogenic ethylenethiourea, a risk estimate for exposure to this latter compound was also made.

HCB has recently been found to be carcinogenic. It also contains dioxins (Fishbein, 1979a,c; Courtney, 1979). It is therefore recommended that a safety factor of 5 000 rather than 1 000 used by NAS (1977o) be employed in deriving a criterion until sufficient data are available to calculate risk estimates. A criterion of 1 ug/L rather than 7 ug/L is thus proposed in order to protect human health.

For PCNB which is carcinogenic, a lifetime risk estimate of 9.1×10^{-8} per ug/L per litre of water consumed daily was made by the NAS (1977p). Assuming a 2 litre per day consumption and an acceptable risk of 1×10^{-6}, a criterion of 3 ug/L is derived using the upper 95% estimate of the risk (1.4×10^{-7} per ug/L per litre of water consumed daily).

The Australian Department of Health (1980) has recommended maximum residue limits for some fungicides in water based on ADI data (NH & MRC, Pesticides & Agricultural Chemicals Subcommittee, undated). Using the same ADI data but assuming 70 kg as the weight of an average individual consuming 2 litre/day of water with 20% of the intake from the water, the criteria in Table 21 are derived. These are more appropriate as the more widely accepted value of 70 kg as the average weight of an individual is used in the derivation.

TABLE 21 DRINKING WATER QUALITY CRITERIA FOR HERBICIDES DERIVED FROM NH & MRC (or FAO) ADI DATA AND ASSUMING A 70 kg INDIVIDUAL WITH A 2 LITRE/DAY CONSUMPTION OF WATER AND THAT 20% of the ADI IS DERIVED FROM THE WATER

FUNGICIDE	DERIVED CRITERION (ug/L)
benomyl	100
carbendazim	400
mancozeb	400
maneb	30
nabam	30
quintozene	40
thiophanate	400
thiram	30
zineb	30

The strong mutagenic activity of some fungicides in short-term tests, e.g. captan and folpet (Waters et al., 1980) suggests that further testing is still needed on higher animals before criteria for these compounds can be derived with more confidence.

SUMMARY OF PROPOSED CRITERIA FOR FUNGICIDES

Protection of the aquatic environment - insufficient data. Some toxicity data are presented in Table 19.

Production of fish suitable for human consumption - 0.02 ug/L for HCB.

Protection of human health (drinking water quality)-

FUNGICIDE	CRITERION (ug/L)
Ziram	80
Captan	300
Folpet	1 000
HCB	1
PCNB	3

For others see Table 21

3.8 FUMIGANTS AND OTHER PESTICIDES

General

These chemicals, which include molluscicides, nematicides and vermicides, can be considered together as there are few compounds and limited data available.

1,2-dichloroethane (ethylene dichloride), a fumigant, has been detected in water. It is manufactured in large quantities but the major uses are other than as a fumigant (Fishbein, 1979b,d). Similarly *p*-dichlorobenzene is used as a fumigant, has other major uses, and has been found in the aquatic environment (Fishbein, 1979a,c).

Aquatic toxicology and criteria for the protection of the aquatic environment

For 1,2-dichloroethane US EPA (1978j, 1979a) has proposed a draft criterion of 3.9 mg/L as a 24 hour average and 8.8 mg/L not to be exceeded at any time for the protection of freshwater aquatic life. The corresponding values for saltwater aquatic life were 0.88 and 2 mg/L respectively.

Acute toxicity data of 550 mg/L for the bluegill, *Lepomis macrochirus* (96 hour LC50), 218 mg/L for *Daphnia magna* (40 hour LC50) (freshwater organisms) and 113 mg/L for the marine shrimp *Mysidopsis bahia* (96 hour LC50) were reported. The most sensitive organism tested was *Mysidopsis bahia* and a criterion of 5.5 mg/L can thus be calculated from its LC50 level, using an application factor of 0.05. A value of 0.05 is appropriate because 1,2-dichloroethane shows little tendency to accumulate (a bioaccumulation factor of 2 has been reported (US EPA, 1978j)) and up to the present time the toxicity has not been established for many organisms. A criterion of 5 mg/L is therefore proposed for the protection of the aquatic environment. It is calculated by a procedure which is more simple than that used by the US EPA (1978j, 1979a) but which is as equally justifiable.

Criteria for the protection of human health

p-dichlorobenzene is a suspected carcinogen although the evidence is far from conclusive (Fishbein, 1979a,c). On this basis a safety factor of 5 000 is recommended in deriving a criterion, until better data are available. Thus a criterion of 20 ug/L is recommended rather than the 94 ug/L derived by NAS (1977q) which was based on a safety factor of 1 000. NAS reviewed the relevant data for 1,2-dichloroethane and considered it inadequate for criterion derivation. However US EPA later determined risk estimates for this compound which is carcinogenic (US EPA, 1978j, 1979a). These estimates also included the added risk of consuming fish and shellfish from waters containing 1,2-dichloroethane. The consumption of the water alone was calculated to be 96% of the exposure. Thus for an acceptable risk of 1×10^{-6} a criterion of 0.7 ug/L can be derived for consumption of water alone.

The fumigants 1,2-dibromoethane (IARC, 1977d) and 1,2-dibromo-3-chloropropane (DBCP) (IARC, 1977e), are also carcinogenic and mutagenic. DBCP is mutagenic in the dominant lethal test with rats (Teramoto et al., 1980), and has caused sperm count depressions in factory workers involved with its use (Glass et al., 1979). Due to this latter fact its use has been banned in the USA. Insufficient data are available for criteria derivation but the toxic properties show the need to minimise exposures to these compounds.

SUMMARY OF PROPOSED CRITERIA FOR FUMIGANTS

Protection of the aquatic environment -
1,2-dichloroethane - 5 mg/L

Protection of human health (drinking water quality) -

COMPOUND	CRITERION (ug/L)
p-dichlorobenzene	20
1,2-dichloroethane	0.7

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4. DETAILED CRITERIA FOR PHENOLS

General

Phenols comprise a large group of compounds with molecular structures which include aromatic rings to which are attached hydroxyl groups. They are used in large quantities as raw materials in the manufacture of plastics, dyes, drugs and other chemicals, e.g. 2,4,5-T and hexachlorophene are manufactured from 2,4,5-trichlorophenol. The chlorophenols, tetrachlorophenols and pentachlorophenol are used as wood preservatives. Phenolic compounds are produced during coking and coal distillation and in the pulp and paper industries. Effluents from the above industrial processes represent obvious sources of phenolic contamination of the aquatic environment.

The uses of certain phenols result in the direct introduction of these compounds into the environment. For example pentachlorophenol is used as a herbicide, molluscicide and fungicide and 3-trifluoromethyl-4-nitrophenol is used as a lampricide in North America. The phenol DNOC (2-methyl-4, 6-dinitrophenol) is used both as a herbicide and insecticide. The herbicides bromoxynil, ioxynil and dinoseb are also phenols. Some pesticides are derivatives of phenols and their degradation can result in the production of phenols, e.g. 2,4,5-T (Crosby & Wong, 1973).

Phenols in the aquatic environment can also result from natural sources e.g. the degradation of the complex humic and fulvic substances. Some red algae produce bromophenols. Water treatment procedures such as disinfection can modify organic compounds, often forming other compounds with less desirable characteristics. With waters containing phenols, chlorination can produce chlorophenols (Burttschell et al., 1959) and, in the presence of bromide ion, it has recently been shown that brominated phenols can be produced (Sweetman & Simmons, 1980). The halogenated phenols are undesirable in drinking waters as they have very low taste and odour thresholds.

The many possible origins of phenols as water pollutants have been extensively reviewed by Buikema et al. (1979).

Residues in the aquatic environment

Phenols are potentially major water pollutants and documented occurrences in the aquatic environment are relatively common. More recent occurrences shown in Table 22.

The persistence of phenols in the aquatic environment is variable. Phenol is rapidly degraded in the presence of bacteria which have adapted to utilising phenol as a substrate (Visser et al., 1977; Borighem & Vereechen, 1978). It does not accumulate to any significant extent in exposed organisms and is rapidly eliminated on termination of exposure (Swift, 1979). On the other hand, pentachlorophenol has been shown to persist for extended periods in contaminated sediments (Pierce et al., 1977). It concentrates in exposed organisms, although not to the same degree as organochlorine insecticides, with concentration factors of up to 1000 having been reported (Kobayashi & Akitake, 1975). It is not readily eliminated on termination of exposure, cf. phenol (Holmberg et al., 1972; Pruitt et al., 1977). In the eel, pentachlorophenol has been shown to be quite persistent (Holmberg et al., 1972). Similarly, 2,4,6-trichlorophenol, tri- and tetrachloroguaiacol were shown to concentrate in exposed fish, with elimination requiring several weeks on termination of exposure (Lander et al., 1977).

TABLE 22 RECENT DETERMINATIONS OF PHENOLS IN THE AQUATIC ENVIRONMENT.

PHENOLS IDENTIFIED	LOCATION	COMMENT	REFERENCE
Phenols	Victoria, Australia	Determined in bores adjacent to a quarry used as a disposal pit for phenolic wastes. Maximum level 608 mg/L.	Riha, 1977
Phenol	Wisconsin, USA	Wells contaminated with levels up to 1130 mg/L after a spill. Illness caused by drinking contaminated water.	Baker et al., 1978
Phenol, cresols and xylenols	California, USA	Petroleum refinery effluents.	Baird et al., 1977
Phenols and phenol-carboxylic acids	Tokyo, Japan	River water polluted by sewage and industrial effluents.	Matsumoto et al., 1977
Phenolic acids	Japan	Sediments. Most derived from lignin from plants.	Matsumoto & Hanya, 1980
Catechol, t-butylphenol	-	Contaminated drainage water.	Armentrout et al., 1979
Chlorophenols	Sweden	Spent bleach liquor from a pulp mill.	Lindstrom & Nordin, 1976
Chlorophenols	The Netherlands	Surface (river) water. Pentachlorophenol found in all samples, maximum concentration 11 ug/L.	Wegman & Hofstee, 1979
Chlorophenols	Weser Estuary, German Bight, Germany	Water samples. Maximum level (pentachlorophenol) 409 ng/L.	Weber & Ernst, 1978
Chlorophenols	Weser Estuary, Germany	Sediments and suspended matter.	Eder & Weber, 1980.
Chlorophenols	Weser Estuary, Germany	Organisms.	Ernst & Weber, 1978b
Chlorophenols	-	Wastewater and waste brines.	Armentrout et al., 1979
Pentachlorophenol, hexachlorophene	Oregon, USA	Sewage, effluent, river water and treated drinking water. Some removal during sewage and water treatment.	Buhler et al., 1973
Pentachlorophenol	Weser Estuary, German Bight, Germany	Water samples. Maximum level 496 ng/L.	Ernst & Weber, 1978a
Pentachlorophenol	-	Wastewater from a wood treating plant.	Ingram et al., 1979
Pentachlorophenol	Mississippi, USA	Fish, water, sediments following contamination of a creek by wood treating wastes.	Pierce et al., 1977
Pentachlorophenol	Pennsylvania, USA	Contamination of a creek downstream of a wood preserving company.	Fountaine et al., 1976

Aquatic toxicology and criteria for the protection of the aquatic environment

Some attention has been paid to the derivation of water quality criteria for phenols in relation to protection of the aquatic environment. Criteria were derived by the European Inland Fisheries Advisory Commission (EIFAC) in order to protect freshwater fish (EIFAC, 1973). To protect salmonid fish it was recommended that levels of phenol, cresols or xylenols should not exceed 1.0 mg/L, either singly or collectively. If 2,5-xylenol were the main constituent the concentration should not exceed 0.5 mg/L. For coarse fish a criterion of 2.0 mg/L with a reduction of 50% if temperatures were below 5°C was recommended. In order to protect commercial fisheries levels of xylenols less than 0.5 mg/L were recommended. This was considered sufficient to prevent tainting. The criteria for the protection of salmonid and coarse fish were based on the observations of survival of these fish in polluted streams in the wild. However since a growth reduction of 20% in salmonids exposed to 1.0 mg/L was also reported, it appears that the 1.0 mg/L criterion for this fish is too high.

The US EPA has derived tentative criteria in order to protect freshwater and saltwater life for a number of phenols (US EPA, 1978a-g; 1979a,b) (Table 23). Those for 2-chloro-, 4-chloro-, 2,4-dichloro- and 2,4,6-trichlorophenol were based on the highest levels estimated not to cause taining of fish flesh. This was consistent with the US EPA definition of criterion to protect aquatic life which states that the criterion should 'protect both the presence and uses of aquatic organisms' (US EPA, 1979a).

TABLE 23 US EPA DRAFT CRITERIA FOR PHENOLS, FOR PROTECTION OF FRESHWATER AND SALTWATER AQUATIC LIFE

	FRESHWATER AQUATIC LIFE		SALTWATER AQUATIC LIFE		REFERENCES
PHENOL	24 hour average (ug/L)	Not to exceed (ug/L)	24 hour average (ug/L)	Not to exceed (ug/L)	
Phenol	600	3400	-	-	US EPA, 1978a, 1979b
2-chlorophenol	60*	180	-	-	US EPA, 1978b, 1979a
4-chlorophenol	45*	180	-	-	US EPA, 1978c, 1979b
2,4-dichlorophenol	0.4*	110	-	-	US EPA, 1978c, 1979a
2,4,6-trichlorophenol	52*	150	-	-	US EPA, 1978c, 1979b
Pentachlorophenol	6.2	14	3.7	8.5	US EPA, 1978d, 1979a
2,4-dimethylphenol	38	86	-	-	US EPA, 1978f, 1979a
2-nitrophenol	2700	6200	-	-	US EPA, 1978g, 1979b
4-nitrophenol	240	550	53	120	US EPA, 1978g, 1979b
2,4-dinitrophenol	79	180	37	84	US EPA, 1978g, 1979b
2,4-dinitro-6-methylphenol	57	130	-	-	US EPA, 1978g, 1979b
2,4,6-trinitrophenol	1500	3400	150	340	US EPA, 1978g, 1979b

* based on tainting of fish flesh.

Russian authorities have set limits for resorcinol and o-cresol in order to protect aquatic life (Buikema et al., 1979). Other Russian workers have established maximum permissible concentrations of a number of phenols for a number of aquatic organisms (Alekseyev & Antipin, 1976; Bobrov & Sudakova, 1977).

The toxicity data for phenols have been reviewed in the US EPA criteria documents (US EPA, 1978a-g), by EIFAC (1973) and by Buikema et al. (1979). From these some generalisations about the toxicity of phenols can be made. Fish appear to be among the most sensitive organisms although molluscs are equally as sensitive to pentachlorophenol (US EPA, 1978d); pentachlorophenol is used as a mollusicicide. Phenols, cresols and xylenols have similar toxicity to fish and their effects are additive. The toxicity may arise as a result of direct toxic action or through low oxygen levels brought about by the BOD of the phenol. The toxicity is also increased by low dissolved oxygen levels and increases with salinity. Salmonids are more sensitive than coarse fish and in many cases they are the most sensitive organism. Juveniles are more sensitive than adults (EIFAC, 1973). Rainbow trout are less sensitive to phenol at higher temperatures (Brown et al., 1967; Cairns et al., 1978) but the reverse is true for golden shiners and goldfish (Cairns et al., 1978).

The toxicity of phenols to aquatic organisms generally increases as the degree of chlorine substitution in the phenol increases, e.g. pentachlorophenol is much more toxic than phenol. The increase in toxicity also parallels the degree of bioaccumulation (Kobayashi et al., 1979). Hence the tendency to bioaccumulate increases as degree of chlorine substitution increases. However, a comparison of the LC50 data for 2,3,6-trichloro- and pentachlorophenol for the crayfish *Astacus fluviatilis* shows that there are exceptions to these generalisations (Kaila & Saarikoski, 1977).

Criteria for a number of phenols have been derived in this report from the toxicity data for the most sensitive organism using an application factor of 0.1, only where the data were considered adequate (Table 24). The factor 0.1 was chosen from a consideration of the relatively low accumulation potential for phenols, and the value of the experimentally determined application factor for pentachlorophenol derived for fish from a lifecycle test involving reproduction (Parrish et al., 1978). The resultant criteria were also compared with sublethal effect data in order to test their suitability. These are the proposed criteria for the protection of the aquatic environment.

Bioaccumulation and the production of fish suitable for human consumption

Phenols can taint the flesh of fish and render them unpalatable. The chlorophenols are mainly responsible as they have very low taste and odour thresholds. Since the tainting of fish by some phenols can occur at levels much less than the criteria necessary to protect the aquatic environment, separate criteria may be required for the production of fish suitable for human consumption.

TABLE 24. ACUTE TOXICITY DATA FOR SENSITIVE ORGANISMS AND DERIVED CRITERIA FOR THE PROTECTION OF THE AQUATIC ENVIRONMENT FOR PHENOLS (Criteria derived using an application factor of 0.1).

PHENOL	ORGANISM	ACUTE TOXICITY (hrs)	(ug/L)	REFERENCE	DERIVED CRITERION (ug/L)
Phenol	*Baetis* sp.	48	1500	cited in Buikema *et al.*, 1979	100^b
	Daphnia magna	96	5000a	cited in US EPA, 1978a	
	Daphnia magna	50	7000	cited in US EPA, 1978a	
	Salmo gairdneri	96	5020	McLeay, 1976	
o-cresol	*Daphnia magna*	48	15800	cited in Buikema *et al.*, 1979	1000
	Pimephales promelas	96	12550	cited in Buikema *et al.*, 1979	
m-cresol	*Daphnia magna*	24	8900	cited in Buikema *et al.*, 1979	1000
p-cresol	*Daphnia magna*	48	12000	cited in Buikema *et al.*, 1979	1000
	Pimephales promelas	?	19000	cited in Buikema *et al.*, 1979	
2-chlorophenol	*Daphnia magna*	48	2580	cited in US EPA, 1978a	200
	Lepomis macrochirus	96	6590	cited in US EPA, 1978b	
4-chlorophenol	*Daphnia magna*	48	4060	cited in US EPA, 1978c	400
	Lepomis macrochirus	96	3830	cited in US EPA, 1978c	
2,4-dichlorophenol	*Daphnia magna*	24	2600	cited in Buikema *et al.*, 1979	
2,4,6-trichlorophenol	*Daphnia magna*	48	6040	cited in US EPA, 1978c	30
	Lepomis macrochirus	96	320	cited in US EPA, 1978c	
Pentachlorophenol	*Crassostrea virginica*	192	34^c	cited in US EPA, 1978d	1^d
	Palaemon elegans	96	84	Van Dijk *et al.*, 1977	
	Salmo gairdneri	96	48	Davis & Hoos, 1975	
	Lagodon rhomboides	96	38	cited in US EPA, 1978d	
	Oncorhynchus kisutch	96	37	Davis & Hoos, 1975	
	Oncorhynchus tschawytscha	96	78	Iwama & Greer, 1979	

(a) EC50 immobilisation

(b) 3074 ug/L phenol was toxic to *Daphnia* in a long term study involving reproduction (US EPA, 1978a). A criterion of 100 ug/L will protect this organism.

(c) EC50 for reduced shell deposition in oysters.

(d) Based on the toxicity to the most sensitive organism and an application factor of 0.1, a criterion of 3 ug/L can be derived. However sublethal effects at less than this level have been reported, e.g. EC 50 values of 1.74 ug/L for growth rate and 1.80 ug/L for conversion efficiency were determined for the sockeye salmon, *Oncorhynchus nerka* (Webb & Brett, 1973). Thus reduction of the criterion to 1 ug/L is recommended and should afford some protection against these effects. The MATC for sheepshead minnows, *Cyprinodon variegatus* was determined as $>47<88$ ug/L (Parrish *et al.*, 1978). The criterion will protect this species. 7 ug/L has been shown to have effects on the species composition of estuarine communities (Tagatz *et al.*, 1977). A criterion of 1 ug/L would afford protection against these effects.

The effects of chlorophenols on the taste of fish have been reported (US EPA 1978b,c,e). The highest concentrations at which it is estimated taste will not be affected are summarised in Table 25. These can be taken as criteria for the production of fish suitable for human consumption. They have been used in a number of the US EPA draft criteria as a basis for the protection of aquatic life, but, as discussed earlier, are not considered ideal for this purpose.

It should be noted that the values appearing in Table 25 are inconsistent with these appearing in a summary table of threshold tainting levels (US EPA, 1978c, 1979b). The paper from which the data in Table 25 originated appears to have been omitted in preparing the summary table.

TABLE 25 HIGHEST ESTIMATED CONCENTRATIONS OF CHLOROPHENOLS WHICH WILL NOT IMPAIR THE TASTE OF FISH (data cited by US EPA, 1978b,c,e).

PHENOL	HIGHEST ESTIMATED NO TASTE IMPAIRMENT LEVEL (ug/L)
2-chlorophenol	60
4-chlorophenol	45
2,3-dichlorophenol	84
2,4-dichlorophenol	0.4
2,5-dichlorophenol	23
2,6-dichlorophenol	35
2,4,6-trichlorophenol	52*

* This level is higher than the criterion for the protection of the aquatic environment.

Criteria for the protection of human health

With respect to drinking water quality, some standards for phenols in general are already in existence. WHO has set standards of 1 ug/L as the highest desirable level and 2 ug/L as the maximum permissible level for phenolic compounds (WHO, 1971). The Indian standard (Kshirsagar, 1979), and the criteria and objectives recommended by the Australian Department of Health (1980) are identical and have presumably been taken directly from the WHO standards. The South African standard for phenolic compounds of 1 ug/L (Wells, 1978) is presumably taken from the WHO "highest desirable level" for this class of compounds. The maximum acceptable concentration for phenols recommended by Canadian authorities is 2 ug/L based on aesthetic considerations of taste and odour (Health and Welfare, Canada, 1978). The EEC has proposed maximum admissible concentrations of 0.5 ug/L for phenols in its water quality standards (Commins, 1980).

Standards for phenols in general as distinct from standards for individual compounds suffer from two disadvantages. Firstly some phenols have much higher taste and odour thresholds than others. Consequently levels of greater than 1 or 2 ug/L of these compounds could be present without the aesthetic quality of the water being impaired. Thus standards of 1 or 2 ug/L are too restrictive for these compounds. Secondly, the general analytical procedures for the determination of phenols on which the enforcement of standards must be based, have several limitations. Those based on the formation of coloured 4-aminoantipyrine derivatives are insensitive to some para substituted phenols (APHA, 1975). Therefore, unless it is known which phenols are present, the levels could be severely underestimated. If the odorous phenols are under-estimated the apparent level could be less than the criterion but the quality would be unacceptable. Individual phenols are not determined and because of the uncertainties inherent in the analytical procedure, the accuracy is indeterminable. Criteria and standards for individual phenols are clearly warranted. These should be based not only on aesthetic consideration but consideration should also be given to the effects on human health.

Some drinking water criteria for individual phenols have been published. The US EPA has derived a criterion of 1 ug/L for phenol in order to ensure the quality of drinking water and to protect against fish flesh tainting. This was based on the odour threshold levels for chlorophenols (US EPA 1976). Thus the basis was aesthetic rather than a consideration of health effects. The NAS attempted to derive drinking water quality criteria for 2,4-dichloro-, 2,4-dimethyl-, o-methoxy- and pentachlorophenol, and hexachlorophene (NAS, 1977). The data were considered adequate for criteria derivation only for pentachlorophenol and hexachlorophene. Based on a consideration of health effects, criteria of 21 ug/L and 8 ug/L respectively were derived from the calculated ADI using the usual assumptions. However pentachlorophenol contains chlorinated dibenzofurans and chlorinated dibenzodioxins (Johnson et al., 1973; Pfeiffer et al., 1978), and the presence of these toxic impurities may also need to be monitored and considered in assessing the suitability of the water for consumption. The formation of chlorinated dioxins from pentachlorophenol from preserved timber (Gebefugi et al., 1979), also creates additional concern regarding this compound, especially since the dioxins in pentachlorophenol have now been reported to be highly toxic (Dickson, 1980).

Russian authorities have set maximum permissible concentrations for a number of phenols in drinking water based on toxicological or organoleptic considerations (Stofen, 1973). Little comment can be made regarding these limits as the procedure for their derivation has not been given.

The US EPA has recently reviewed the data for a number of phenols in their establishment of draft water quality criteria for these compounds (US EPA 1978a-g). For some compounds the drinking water quality criteria were based on the odour thresholds where these were lower than criteria calculated from toxicity data. In some cases sufficient toxicity data were not available and the odour threshold concentrations had to suffice.

The criteria for phenol and some chlorophenols based on toxicological data are shown in Table 26. These criteria are calculated on the basis of consumption of water, and fish and shellfish grown in that water. Also included are criteria calculated from the US EPA ADI levels and based on exposure through water alone. These latter criteria, except those for tetra- and pentachlorophenol, are proposed in this report, since it is considered that the total exposure assumptions of the US EPA are not completely justifiable. For pentachlorophenol the criterion derived by NAS is preferred and differs by a factor of 10 from the US EPA value. The NAS employed a more conservative safety factor in their calculations (1 000 compared with 100 used by US EPA) and this is considered warranted due to the possibility of toxic impurities in pentachlorophenol. Because of the similarities between tetra- and pentachlorophenol (US EPA, 1978c, 1979b), the criterion for pentachlorophenol derived by NAS (21 ug/L) should also apply to tetrachlorophenol.

TABLE 26. US EPA DRAFT CRITERIA FOR THE PROTECTION OF HUMAN HEALTH FOR PHENOLS, AND CRITERIA DERIVED FROM THE SAME ADI BUT ASSUMING EXPOSURE THROUGH INGESTION OF WATER ALONE (US EPA, 1978a,c,d,e, 1979a,b)

PHENOL	NO-OBSERVED-EFFECT LEVEL (mg/kg/day)	SAFETY FACTOR	ADI (mg/kg)	US EPA CRITERION (ug/L)	CRITERION DERIVED ON BASIS OF EXPOSURE THROUGH INGESTION OF WATER ALONE[a] (ug/L)
Phenol	50	500	0.1	3 400	700
2,4-dichlorophenol	100	1 000	0.1	371[b]	700
2,4,5-trichlorophenol	100	1 000	0.1	1 600	700
Tetrachlorophenol	3	100	0.03	263	210
Pentachlorophenol	3	100	0.03	680	210[c]

(a) Calculated on the basis of a 70 kg individual consuming 2L of water per day with 20% of the ADI in the water.

(b) Calculated by an unusual procedure. Using the more typical procedure, a criterion of 2600 ug/L is derived.

(c) A factor of 10 higher than NAS criterion.

The lowest odour thresholds for a number of chlorophenols which formed the basis of some US EPA draft criteria (US EPA 1978b,c,e, 1979a,b) are shown in Table 27. The odour threshold data have also been reviewed by Buikema et al. (1979).

Taste and odour thresholds for 126 phenolic compounds have recently been reported (Dietz & Traud, 1978) and these are shown in Table 28. The taste thresholds were found to be much lower than the odour thresholds for most compounds. The taste thresholds were somewhat less than, or similar to, the thresholds forming the basis of the US EPA criteria. The taste threshold data shown in Table 28 are proposed as criteria based on organoleptic properties for these compounds. These should only be used as drinking water quality criteria if lower than criteria which could be derived from toxicological data, e.g., phenol.

TABLE 27. LOWEST REPORTED ODOUR THRESHOLDS FOR A NUMBER OF CHLOROPHENOLS (US EPA 1978b,c,e, 1979b)

PHENOL	ODOUR THRESHOLD (ug/L)
2-chlorophenol	0.33
3-chlorophenol	200
4-chlorophenol	33
2,4-dichlorophenol	0.65
2,5-dichlorophenol	3.3
2,6-dichlorophenol	3
2,4,5-trichlorophenol	11
2,4,6-trichlorophenol	100
Tetrachlorophenol	915

TABLE 28 TASTE THRESHOLD CONCENTRATIONS FOR PHENOLS (Data of Dietz & Traud, 1978)

PHENOL	TASTE THRESHOLD CONCENTRATION (ug/L)
Phenol	300
o-cresol	3
m-cresol	2
p-cresol	2
2,3-xylenol	30
2,4-xylenol	500
2,5-xylenol	500
3,4-xylenol	50
2,4,6-trimethylphenol	10
o-ethylphenol	30
m-ethylphenol	10
p-ethylphenol	10
o-*tert*-butylphenol	30
m-*tert*-butylphenol	30
p-*tert*-butylphenol	30
o-chlorophenol	0.1
m-chlorophenol	0.1
p-chlorophenol	0.1
2,3-dichlorophenol	0.04
2,4-dichlorophenol	0.3
2,5-dichlorophenol	0.5
2,6-dichlorophenol	0.2
3,4-dichlorophenol	0.3
2,3,6-trichlorophenol	0.5
2,4,5-trichlorophenol	1
2,4,6-trichlorophenol	2
2,3,4,6-tetrachlorophenol	1
Pentachlorophenol	30
3-methyl-4-chlorophenol	50
o-nitrophenol	1
Resorcinol	2000
Guaiacol	50
p-hydroxybenzoic acid	1600
-napthol	500
Indole	500
Skatole	50

In some cases the criterion based on toxicity data is lower tnan that based on organoleptic properties, e.g. pentachlorophenol and should alwasy be used. Where the toxicity data are inadequate for criteria derivation and care must be taken in assessing water quality on the basis of organoleptic properties.

A summary of proposed criteria based on toxicological data, and a comparison with those obtained from taste threshold data where such data were available, are shown in Table 29.

TABLE 29 PROPOSED DRINKING WATER QUALITY CRITERIA FOR PHENOLS/ BASED ON TOXICOLOGICAL DATA AND A COMPARISON WITH THOSE OBTAINED FROM TASTE THRESHOLD DATA

	DERIVED CRITERION (ug/L)	
PHENOL	BASED ON TOXICOLOGICAL DATA	BASED ON ORGANOLEPTIC DATA
Phenol	700	300
2,4-dichlorophenol	700	0.3
2,4,5-trichlorophenol	700	1
2,3,4,6-tetrachlorophenol	20	1
Pentachlorophenol	20	30*
Hexachlorophene	8	-

*Should not be used as a drinking water quality criterion as it is higher than the value derived from toxicological data.

SUMMARY OF PROPOSED CRITERIA FOR PHENOLS

Protection of the aquatic environment - see Table 24.

Production of fish suitable for human consumption - see Table 25

Protection of human health (drinking water quality) -

Based on toxicological data - see Table 29
Based on organoleptic properties - see Table 28

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5. DETAILED CRITERIA FOR POLYCHLORINATED BIPHENYLS AND RELATED COMPOUNDS

General

Polychlorinated biphenyls (PCBs) are complex mixtures of compounds manufactured by the reaction of biphenyl with chlorine. There are 209 possible individual compounds which can be produced, ranging from the simplest, chlorobiphenyl (of which there are 3 isomers), up to the most highly chlorinated, decachlorobiphenyl (Mieure et al, 1976). Chlorinated dibenzofurans (Vos et al., 1970; Bowes, 1976), and chlorinated napthalenes (Vos et al., 1970) are also present in these mixtures.

PCB mixtures have two distinctive properties, high dielectric constants and inertness which makes them eminently suitable for a number of applications. The two main uses of PCBs are in capacitors and transformers (Durfee, 1976). A number of other, minor uses have been developed such as plasticisers, diffusion pump oils, fuel flow improvers, etc. Usage in Australia is now restricted to closed system applications and is decreasing, with almost all being utilised in capacitors and transformers. No PCBs are manufactured in Australia (Richardson & Waid, 1979). Further discussion on the uses of PCBs can be found in a number of reviews (Fishbein, 1972; Panel on Hazardous Trace Substances, 1972; Hutzinger et al., 1974, Peakall, 1975; Fishbein, 1979; Richardson & Waid, 1979).

The PCBs manufactured by different companies are marketed under different tradenames which include Aroclor (Monsanto, USA), Clophen (Bayer, Germany), Phenoclor and Pyralene (Prodelec, France), Fenchlor (Caffaro, Italy) and Kanechlor (Kanegafuchi, Japan). In 1975 the Aroclors were the sole PCBs manufactured in the USA and represented nearly 99% of the usage in that country (Durfee, 1976). It is the group of PCBs for which most data have been generated. PCBs imported into Australia have come from a number of manufacturers. In the past few years the major PCBs imported would appear to be the Aroclors, Phenoclors and Pyralenes, judging by the manufacturers represented in Australia (Richardson & Waid, 1979). Since Monsanto stopped sales in Australia in 1977 (Richardson & Waid, 1979) the Phenoclors and Pyralenes would appear to be the only PCBs still used in Australia.

PCB mixtures are marketed on the basis of the degree of chlorination (chlorine content) which is controlled by the chlorine contact time during manufacture (Durfee, 1976). The chlorine content is differentiated by a number following the name. The Aroclor mixtures have a 4 digit number following the name. The first 2 digits designate the type of molecule (12 = biphenyls; 25 = 75% biphenyls, 25% terphenyls; 44 = 60% biphenyls, 40% terphenyls; 54 = terphenyls). The second 2 digits indicate the percentage chlorine content. Thus Aroclor 1242 is a chlorinated biphenyl containing 42% chlorine. The exception to this nomenclature is Aroclor 1016 which contains 41% chlorine.

The Aroclors of higher chlorine content naturally have a greater percentage of the more highly chlorinated compounds. Aroclor 1221 is comprised of mainly monochlorobiphenyls while Aroclor 1254 is comprised of mainly pentachlorobiphenyls (Mieure et al., 1976). Although Aroclor 1254 consists mainly of pentachlorobiphenyls, there are still 46 possible isomers of this compound. In total, Aroclor 1254 is comprised of 85 different compounds (Mieure et al., 1976).

Although PCBs have had a number of industrial uses since 1929, it was recognised only in 1966 that they were a significant environmental pollutant (Anonymous, 1966). Since that time a wealth of information on the fate in, and effects on, the environment, and toxicology of PCBs has been published. These topics have been covered in a number of comprehensive reviews (Fishbein, 1972; Panel on Hazardous Trace Substances, 1972; Kimbrough, 1974; Huntzinger et al, 1974; Peakall, 1975; Addison, 1976; Fishbein, 1979; Kalmaz & Kalmaz, 1979; Richardson & Waid, 1979).

Once discharged into the environment, PCBs persist and are widely transported. Being inert and highly fat soluble, they accumulate in the lipids of exposed organisms resulting in bioconcentration along the food chain. Their behaviour is identical to the persistent organochlorine insecticides and they are often found in association with these compounds. Their ubiquitous presence in sediments and organisms throughout the environment has led to their use being restricted and attempts made to find replacements for them.

In the USA, PCBs are no longer manufactured (US EPA, 1978). Similarly, their manufacture and use has been banned in Japan since 1972 (Yagi & Sudo, 1980). However it has been estimated that a substantial quantity of PCBs manufactured prior to 1971 are still in service and represent a significant potential pollution source (US EPA, 1978).

Other sources of PCBs are also possible. DDT has been shown to produce PCBs on chlorination (Plimmer & Kligebiel, 1973). Similarly chlorinated benzenes have been shown to give rise to PCBs under photochemical conditions (Uyeta et al., 1976). Chlorination of aqueous samples containing biphenyl can also produce PCBs (Gaffney, 1977; Snider & Alley, 1979), but the reaction is slow. These sources would appear to be very minor at the present time compared with the large quantities of manufactured PCBs in use.

A difficulty encountered in the derivation of water quality criteria for PCBs is the effect of impurities in PCBs on their toxicities. Polychlorinated dibenzofurans (PCDFs) which have been shown to be present in PCB mixtures have very high toxicities (Vos et al., 1970, Moore et al., 1979), and may account for some of the toxicity attributed to PCBs. The incident in Japan, where a number of people were poisoned as a result of ingestion of cooking oil contaminated with PCBs is a good example (Kuratsune et al., 1976;

Nagayama et al., 1976; Umeda et al., 1978). The high levels of PCDFs in the oil may have been responsible for the resultant toxicity (Kuratsune et al., 1976; Nagayama et al., 1976). It is possible that PCDFs are formed from PCBs, either during the normal use of the PCB mixture as a heat exchanger, or by reaction with the cooking oil on heating (Kuratsune et al., 1976). PCDFs can also be formed photochemically from PCBs under conditions which are similar to those which occur in the environment (Crosby & Moilanen, 1973). Thus PCB residues in the environment may pose a hazard through the formation of PCDFs.

Residues in the aquatic environment

The high stability of PCBs has resulted in residues being frequently determined in the aquatic environment. A number of recent reports of determinations relate to samples from the Great Lakes area of North America (Table 30). Analysis of museum specimens of fish from the area revealed that PCBs first appeared in Lake Michigan in 1949 (Neidermyer & Hickey, 1976). In Lake Ontario the levels in water and fish exceeded US EPA criteria (Waller & Lee, 1979). Similarly in most of the lakes, fish samples have exceeded the tolerances set by the US (5 ug/g) and Canadian (2 ug/g) authorities (Veith et al., 1977; Frank et al., 1978a,b; Delfino, 1979). These data, along with the organochlorine insecticide residue data for this area, indicate a severe organic pollution problem in the Great Lakes.

PCB residues have frequently been determined in the US aquatic environment (Table 31) and widespread contamination is indicated. In one comprehensive survey of fish from US waters, 93% contained PCB residues. 53% contained levels greater than the 5 ug/g tolerance level set by the US Food & Drug Administration (Veith et al., 1979).

PCB residues have frequently been determined in the aquatic environments of other countries and in the oceans of the world. Recent determinations are shown in Table 32.

Several studies have provided evidence for atmospheric deposition as an important source of PCBs in many areas (Harvey & Steinhauer, 1976; Kihlstrom & Berglund, 1978; Eisenreich et al., 1979). It would appear that high levels in surface films result mainly from atmospheric deposition (Ofstad et al., 1979). The occurrence of PCB residues in rainwater (Wells & Johnstone, 1978; Strachan & Huneault, 1979) tends to confirm this distribution pathway.

Data for PCB residues in the Australian aquatic environment are scant. Residues in captive water rats most probably originated from the fish on which they wer fed as these were shown to contain significant residues (Anonymous, 1978; Woollard & Settle, 1978). PCBs were also found in fish and other animals from the Brisbane River estuary with the highest level (15.7 mg kg^{-1}) being found in pelicans (Shaw & Connell, 1980). However no PCBs were found in a survey of organochlorine residues in organisms from the Great Barrier Reef (Olafson, 1978).A few other occurrences were cited in the review by Richardson & Waid (1979).

TABLE 30. RECENT DETERMINATIONS OF PCBs IN GREAT LAKES, NORTH AMERICA

LAKE	DETERMINED IN	REFERENCE
Erie	Fish	Frank et al., 1978a; Moccia et al., 1978
	Sediments	Frank et al., 1977, 1978a
Huron	Fish	Frank et al., 1978b; Moccia et al., 1978; Zabik et al., 1978
	Suspended matter	Glooschenko et al., 1979
	Sediments	Glooschenko et al., 1976; Frank et al., 1979
Michigan	Fish	Veith, 1975; Glooschenko et al., 1976, Willford et al., 1976; Moccia et al., 1978 Zabik et al., 1978
	Suspended matter	Glooschenko et al., 1978
	Sediments	Glooschenko et al., 1978
Ontario	Water	Waller & Lee, 1979
	Fish	Moccia et al., 1978; Norstrom et al., 1978; Waller & Lee, 1979
	Herring gull eggs	Norstrom et al., 1978
St. Clair	Fish	Frank et al., 1978a
	Sediments	Frank et al., 1977, 1978a
Superior	Water	Veith et al., 1977
	Plankton	Veith et al., 1977
	Fish	Glooschenko et al., 1976, Kaiser, 1977, Veith et al., 1977; Passino & Kramer, 1980
	Sediments	Veith et al., 1977; Eisenreich et al., 1979
Various	Rainwater	Strachan & Huneault, 1979

TABLE 31. RECENT DETERMINATIONS OF PCBs IN US AQUATIC ENVIRONMENTS

LOCATION	DETERMINED IN	REFERENCE
California	Wastewaters	Young et al., 1976
	Mussels, crabs, fish	McDermott et al., 1976
	Fish	McDermott-Ehrlich et al., 1978
	Sediments	Young et al., 1977
Florida	Oysters	Wilson & Forester, 1978
Idaho	Fish, sediments	Perry, 1979; Kent&Johnson, 1979
Illinois	Wastewater from a PCB manufacturing plant	Stratton & Sosebee, 1976
Minnesota, Wisconsin	Heron chicks	Ohlendorf et al., 1979
New England	Fish	Butler & Schutzmann, 1979
New York	Fish	Wszoleck et al., 1979
	Sediments, sewage sludge	West & Hatcher, 1980
	Molluscs, sediments	Stainken & Rollwagen, 1979
	Water, sediments, organisms	Nadeau & Davis, 1976
Oklahoma	Fish, sediments	Hunter et al., 1980
Oregon, Washington	Marine organisms	Claeys et al., 1975
South Carolina	Raw and drinking water	Billings et al., 1978
Virginia	Water, fish, sediments	Martell et al., 1975
Washington	Water, suspended solids	Hafferty et al., 1977
	Fish, mussels, sediments	Mowrer et al., 1977
	Water, suspended matter, zooplankton, sediments	Pavlou & Dexter, 1979a,b
Wisconsin	Mayflies	Mauck & Olson, 1977
Various	Estuarine fish	Butler & Schutzmann, 1978
	Fish	Veith et al., 1979
	Mussels	Goldberg et al., 1978
	Water, sediments	Dennis, 1976
	Water, fauna, sediments, flora	Crump-Wiesner et al., 1974
	Sewage, effluent, sludge (municipal treatment works)	Bergh & Peoples, 1977

TABLE 32. RECENT DETERMINATIONS OF PCBs IN COUNTRIES OTHER THAN THE USA AND AUSTRALIA

LOCATION	DETERMINED IN	REFERENCE
CANADA	Fish	Zitko, 1971; Graham, 1976
	Marine organisms	Albright et al., 1975; Sims et al., 1977
	Fish eggs	Zitko & Saunders, 1979
	Fish eating birds	Vermeer & Peakall, 1977
	Seals	Addison & Brodie, 1977; Rosewell et al., 1979
	Sewage, sludge	Lawrence & Tosine, 1977
	Sewage, sludge, plants grown on sludge treated land	Shannon et al., 1976
FINLAND	Aquatic plants	Sarkka et al., 1978a
	Fish	Hattula et al., 1978
	Plankton	Sarkka et al., 1978b
FRANCE	Fish	Keck & Raffenot, 1979
GERMANY	Fish, invertebrates	Goerke, 1979
IRAN	Sediments	Sodergren et al., 1978
JAPAN	Marine organisms	Nakamura & Kashimoto, 1978
KENYA	Aquatic organisms	Greichus et al., 1978b
MEXICO	Oysters	Rosales et al., 1979
NETHERLANDS	Fish, shellfish	Hagel & Tuinstra, 1978
	Mussels	Quirijns et al., 1979
	Porpoise foetus	Duinker & Hillebrand, 1979
NORWAY	Fish	Ofstad et al., 1978
PUERTO RICO and VIRGIN ISLANDS	Biota	Reimold, 1975
RHODESIA	Aquatic organisms, birds, sediments	Greichus et al., 1978a
SOUTH AFRICA	Aquatic organisms, birds, sediments	Greichus et al., 1977
SWEDEN	Fish	Olson et al., 1978
UNITED KINGDOM	Rainwater	Wells & Johnstone, 1978
	Coastal waters	Dawson & Riley, 1977
	Marine organisms	Riley & Wahby, 1977
	Fish, invertebrates	Wharfe & Van Den Broek, 1976
	Sediments	Cooke, 1979
Adriatic Sea	Fish, mussels	Picer et al., 1978
Atlantic Ocean	Water	Harvey & Steinhauer, 1976
	Organisms	Harvey et al., 1974
Baltic Sea	Organisms	Kihlstrom & Berglund, 1978
Ligurian Sea	Organisms	Contardi et al., 1979
Mediterranean Sea	Water	Elder & Villeneuve, 1977
	Organisms	Fowler & Elder, 1978
		Amico et al., 1979
Pacific Ocean (north-west)	Water, zooplankton	Clayton et al., 1977

PCBs are virtually insoluble in water but are associated strongly with particulate matter (Paris et al., 1978; Steen et al., 1978; Hiraizumi et al., 1979; Wildish et al., 1980). They tend to be mainly found in sediments or concentrated at the surface film (Ofstad et al., 1979) rather than in the water. In determining water concentrations accurately it is important then that the surface film be precluded when sampling. Similarly the amount of particulate matter sampled could also influence the apparent water concentration. Standardised sampling and analysis procedures are therefore required if data are to be properly evaluated.

Even if the usage of PCBs were to cease immediately it would probably take many years for residues to be eliminated from the aquatic environment. Sediments act as sinks for PCBs and have been shown to act as sources of PCBs which continue to contaminate fish long after the original source has been removed (Young et al., 1977). From an evaluation of the data for Lake Michigan using mathematical models it was predicted that the sediments would act as major source of PCBs for several years and that legislation would not alleviate PCB pollution of this lake in the short term (Neely, 1977).

Aquatic toxicology and criterion for the protection of the aquatic environment

The US EPA has derived a water quality criterion of 0.001 ug/L for PCBs in order to protect aquatic life, and to ensure that edible fish from these environments meet the tolerance of 5 ug/g (US EPA, 1976). More recently, a draft criterion of 0.0015 ug/L as a 24 hour average and 6.2 ug/L not to be exceeded at any time, has been derived by the US EPA in order to protect freshwater aquatic life. The corresponding values for saltwater aquatic life were 0.024 and 0.20 ug/L respectively (US EPA 1978, 1979).

Assessing toxicity and establishing criteria for PCBs is complicated by the complexity of the mixtures. Criteria should relate to each PCB mixture, i.e. Aroclors 1242 and 1254 for example should have individual criteria. Ideally each component of the mixture should have its own criterion, but the complexity of PCB mixtures make this latter approach completely impractical.

In relation to the toxicities of the various PCB mixtures and individual components, it is difficult to generalise due to the contradictory nature of the data. Mayer et al. (1977) found the more highly chlorinated Aroclor mixtures to be less toxic to fish, and tests with the individual homologues to parallel this observation. Crustacea were more susceptible than fish. DeFoe et al. (1978) found Aroclor 1260 to be more toxic to fathead minnows than Aroclor 1248 however. Nebeker and coworkers (Nebeker, 1976; Nebeker & Puglisi, 1974; Nebeker et al. 1974) tested a number of organisms with various Aroclor mixtures. Some results showed the more highly chlorinated mixtures to be more toxic (Nebeker et al. 1974; Nebeker, 1976) while others showed no trends (Nebeker & Puglisi, 1974). For algae the biphenyls with a low degree of chlorination show a higher toxicity (Zullei & Benecke, 1978). As there is generally not a great difference in the toxicities of the various PCB mixtures to a particular organism, the only conclusion that can be made is that these mixtures have roughly similar toxicities.

In assessing the toxicity data it is difficult to assess those data which are clearly above the solubilities of PCBs, i.e. those generated using emulsions or solubilisers to extend the aqueous solubilities (Zitko, 1970). The aqueous solubilities are also not well defined. Aroclor 1254 has been reported to have solubilities as high as 2-3 mg/L in freshwater (Zitko, 1970), while it has been suggested that the true solubility is less than 0.1 ug/L (Dexter & Pavlou, 1978). For seawater, solubilities of 0.04 ug/L (Dexter & Pavlou, 1978), up to 56 ug/L (Haque et al., 1974), and 1-1.5 mg/L (Zitko, 1970) have been reported. Different isomers have different solubilities with the highly chlorinated isomers being less soluble (Schoor, 1975; Sundstrom et al., 1975; Tulp & Hutzinger, 1978).

Preferential adsorption or degradation of some components also contribute to the complexity and uncertainty of criteria derivation. In relation to adsorption Schoor (1975) reported the less highly chlorinated components to be preferentially adsorbed on surfaces and therefore reduced in concentration in the aqueous phase. This is the opposite to that reported by Zitko (1970) and the reasons for this discrepancy are not clear, but the more highly chlorinated components would be expected to be more strongly adsorbed.

In relation to degradation of PCBs, the less chlorinated compounds have been shown to degrade more rapidly (Metcalf et al., 1976; Furukawa et al., 1978; Yagi & Sudo, 1980). PCB mixtures in the environment become enriched in the more highly chlorinated components. Thus exposures to PCBs in the environment does not necessarily involve exposures to a mixture of the original composition. With the more highly chlorinated PCBs (e.g. Aroclors 1254 and 1260) it appears that the major components are not at all significantly degraded and these residues accurately match standards (Ballschmiter et al., 1978). The major components in these mixtures are chlorinated biphenyls with 5 or more chlorine atoms and since pentachlorobiphenyl has been shown to be about as stable as DDE, which is very persistent (Metcalf et al., 1976), this is not surprising. Experiments have also shown tri- and pentachlorobiphenyls to be extremely resistant to degradation by activated sludge (Herbst et al., 1977). Photochemical decomposition has also been shown to be slow but is predicted to be an important means of PCB degradation, especially in shallow waters (Bunce & Kumar, 1978).

The availability of PCBs adsorbed to particulate matter or concentrated in surface films in exposures of aquatic organisms is also not clear. Bottom feeding fish were shown to have higher levels of PCBs than other fish (Hunter et al., 1980), suggesting that sediments contributed to exposures. The continuing high levels of PCBs in fish off the California coast were thought to arise from contaminated sediments since other sources of contamination had been eliminated (Young et al., 1977). In laboratory experiments worms were shown to take up PCBs from contaminated sediments thereby making them available to organisms further up the food chain (Courtney & Langston, 1978; Elder et al., 1979). The significance of accumulated residues to exposed organisms is not yet clear. Sensitivity to other stresses including other toxic chemicals has been reported (Bills et al., 1977). Levels as high as 6.48 ug/g (as Aroclor 1254) in salmon eggs did not affect hatchability or survival of fry (Zitko & Saunders, 1979). Aroclor 1254 fed to ducks at 25 mg/kg did not affect reproductive success (Custer & Heinz, 1980). Any adverse effects due to accumulated residues no doubt depend on the levels, and generalisations cannot be made.

The above factors must be taken into account when assessing the significance of PCB exposure on the aquatic environment and in applying derived criteria. It is not feasible to derive criteria for the different PCB mixtures, let alone the individual components. Criteria in this document for the protection of the aquatic environment are derived from the toxicity data for the most sensitive organism and for the most toxic PCB mixture in the usual way. This results in a derived criterion which should be more than adequate to protect against the less toxic mixtures.

Most of the toxicity data necessary for the derivation of criteria have been reviewed by the US EPA (1978). A number of acute and chronic toxicity studies have been performed and a significant feature of PCBs is their delayed toxicity. Therefore acute toxicity data derived from short testing periods of a few days can seriously underestimate the toxicity (Hansen, 1976).

A number of organisms have been reported to have similar sensitivities to PCBs. The toxicity data for the more sensitive organisms are shown in Table 33. Invertebrates appear to have slightly greater sensitivities to PCBs than fish. Based on the acute toxicity to the most sensitive species (3 week LC50 of 0.45 ug/L for Aroclor 1254 and the midge Tanytarsus dissimilis (Nebeker & Puglisi, 1974)), and an application factor of 0.01 since PCBs accumulate strongly, a criterion of 0.004 ug/L is derived for the protection of the aquatic environment.

A number of chronic and sublethal studies have been performed which can be used for comparison with the derived criterion in order to test its suitability. Fathead minnows did not spawn in 4.6 ug/L Aroclor 1254. At 1.8 ug/L spawning occurred but was less than at lower concentrations tested which were similar to the control. 2.2 ug/L Aroclor 1248 greatly reduced growth. The growth and survival of flagfish was also reduced at 2.2 ug/L Aroclor 1248 (Nebeker et al., 1974). Growth of second generation fathead minnows was reduced in 0.4 ug/L Aroclor 1248 in a lifecycle study (DeFoe et al., 1978). Mauck et al. (1978) determined the no-effect level for Aroclor 1254 on backbone composition of brook trout as 0.43 ug/L. 50% impairment of reproduction in Daphnia in 1.1 ug/L Aroclor 1254 was reported by Nebeker and Puglisi (1974). Schimmel et al. (1974) determined the no-effect level of Aroclor 1254 for

TABLE 33. TOXICITY DATA FOR THE MORE SENSITIVE ORGANISMS AND PCBs

	LC50			
ORGANISMS	TIME	ug/L	PCB	REFERENCE
FISH:				
Pimephales promelas (newly hatched)	96 hr	7.7	Aroclor 1254	Nebeker *et al.*, 1974
Pimephales promelas (newly hatched)	96 hr	15	Aroclor 1242	Nebeker *et al.*, 1974
Leiostomus xanthurus	12 day	5.0	Aroclor 1254	Hansen *et al.*, 1971
Lagodon rhomboides	18 day	5.0	Aroclor 1254	Hansen *et al.*, 1971
Salmo gairdneri	25 day	3.4	Aroclor 1248	Mayer *et al.*, 1977
Pimephales promelas	30 day	4.7	Aroclor 1248	DeFoe *et al.*, 1978
Pimephales promelas	30 day	3.3	Aroclor 1260	DeFoe *et al.*, 1978
CRUSTACEA:				
Penaeus aztecus	96 hr	10.5	Aroclor 1016	Hansen *et al.*, 1974
Palaemonetes pugio	96 hr	12.5	Aroclor 1016	Hansen et al, 1974
Daphnia magna	2 wk	24	Aroclor 1254	Maki & Johnson, 1975
Penaeus duorarum	15 day	0.94	Aroclor 1254	Nimmo *et al.*, 1971
Daphnia magna	3 wk	1.3	Aroclor 1254	Nebeker & Puglisi, 1974
Tanytarsus dissimilis (larvae)	3 wk	0.65	Aroclor 1254	Nebeker & Puglisi, 1974
Tanytarsus dissimilis (pupae)	3 wk	0.45	Aroclor 1254	Nebeker & Puglisi, 1974
MOLLUSCS:				
Crassostrea virginica	96 hr	10.2	Aroclor 1016	Hansen *et al.*, 1974

the sheepshead minnow as 0.1 ug/L in a partial lifecycle test. Aroclor 1254 also decreased hatchability, time to hatching and alevin survival in coho salmon at 4.4 ug/L (Halter & Johnson, 1974). A few studies have reported the unusual phenomenon of increased growth in fish exposed to PCBs in their diet (Gruger et al., 1976; Bengtsson, 1980) but it is not clear how these particular data relate to the criterion derivation process.

A number of studies of the effects of PCBs on phytoplankton have been reported. 10 ug/L Aroclor 1254 reduced phytoplankton photosynthesis (Harding, 1976). Levels of Aroclor 1254 as low as 1 ug/L reduced phytoplankton biomass and size. It was suggested that this could give rise to a changed species composition in higher trophic levels (O'Connors et al., 1978).

A criterion of 0.004 ug/L therefore should be sufficient to protect against adverse effects, both in phytoplankton and other organisms for which chronic and sublethal studies have been performed. Since PCBs are strongly adsorbed to particulate matter consideration should also be given to levels in sediments when applying criteria.

Bioaccumulation and the production of fish suitable for human consumption

US authorities have set a tolerance of 5 mg/kg for PCBs in fish for human consumption with a proposal to reduce it to 2 mg/kg. (Veith et al., 1979). No maximum residue limits appear to have been set in Australia.

Rhesus monkeys developed a number of significant symptoms when fed Aroclor 1248 in the diet at levels as low as 2.5 mg/kg (Allen & Norback, 1976). Although fish are only a small part of the human diet, the development of toxic effects at such low levels in primates warrants the adoption of the lower limit of 2 mg/kg.

In determining a water quality criterion consistent with a 2 mg/kg limit, bioaccumulation factors must be considered. Bioaccumulation factors are very high for PCBs and elimination by fish is very slow on termination of exposure (DeFoe et al., 1978; Bengtsson, 1980). Bioaccumulation factors of 270 000 have been reported for fish for exposures to the PCBs Aroclor 1242 and 1260 in the laboratory (Nebeker et al., 1974; DeFoe et al., 1978 respectively). From levels of PCBs in fish and water from Lake Superior, bioaccumulation factors of up to 1.2×10^6 were estimated (Veith et al., 1977). Using a maximum residue limit of 2 mg/kg and a bioaccumulation factor of 10^6, a criterion for the production of fish suitable for human consumption of 0.002 ug/L is derived. The accumulation of PCBs may not be so straightforward however. Different components in the mixtures are accumulated to different extents because of their different properties. The less highly chlorinated PCBs have been shown to be metabolised more readily than the more highly chlorinated components (Sanborn et al., 1976; Bailey & Bunyan, 1972). The more highly chlorinated are also preferentially accumulated (Mayer et al., 1977) and retained for longer periods of time on termination of exposure (Calambokidis et al., 1979). Thus exposed organisms would be expected to contain higher proportions of the more highly chlorinated compounds. This has been shown to be the case with fish from New York (Wszoleck et al., 1979), but most reports make no mention of the relative abundance of the various components. It has been suggested that the persistence of the more highly chlorinated PCBs probably accounts for the widespread recording of Aroclor 1254 as the predominant PCB residue, even though less highly chlorinated PCBs are used in greater quantities (Bailey & Bunyan, 1972). Recently, however, PCB residues matching the less highly chlorinated mixtures (Aroclors 1016 and 1042) have been reported (Stainken & Rollwagen, 1979; Veith et al., 1979).

The greater uptake of the less highly halogenated PCBs and PBBs reported by some workers (Zitko & Hutzinger, 1976; Zitko, 1977) may reflect the short time of the experiments. The less halogenated compounds were probably taken up at a faster rate and bioaccumulation coefficients measured before equilibrium was reached. Langston (1976) reported a selective accumulation of pentachlorobiphenyls by molluscs. Thus residue levels of the various components of PCB mixtures in exposed organisms appear to be a complex relationship between rates of uptake, bioaccumulation coefficients (which are also affected by temperature (Edgren et al., 1979)) and rates of metabolism and elimination. A bioaccumulation factor of 10^6 used in the criterion derivation is much more conservative than those determined in laboratory experiments and should therefore allow for these uncertainties.

Criterion for the protection of human health

Although PCBs are ubiquitous in the aquatic environment it is not yet known what effects low level intakes are having on humans. That the human population is exposed is evidenced by the residues in human fat samples which have been frequently reported from overseas. However, a survey conducted in Australia found no PCBs in human milk (Siyali, 1973). This is surprising since all samples of human fat examined in a New Zealand study contained residues (Solly & Shanks, 1974). Levels averaging 400 ug/kg were found in workers involved with the handling of Aroclor 1254 in an electrical industry in NSW (Ouw et al., 1976).

In the Yusho incident already described high levels of PCBs were found in poisoned individuals but PCDFs may have, in fact, been responsible for the toxicity. Elevated cancer rates have also been suggested (Kuratsune et al., 1976) which is in turn suggestive that either PCBs or PCDFs are carcinogenic.

A large number of studies of the toxicity of PCBs to animals have been performed and these have been reviewed (Kimbrough, 1974; US EPA, 1978). A number of responses to PCB exposure were reported, including carcinogenic, cocarcinogenic and inhibitory responses. PCBs were considered carcinogenic by the IARC (1978). PCBs were mutagenic in short-term mutagenicity tests with one exception which could not, however, be repeated by other workers (US EPA, 1978).

The carcinogenicity data for Aroclor 1260 obtained by Kimbrough et al (1975) for rats was used by the NAS to derive risk estimates for exposure to PCBs in drinking water (NAS, 1977). The US EPA draft drinking water quality criterion was derived from the same data, but consideration was given to the consumption of fish and shellfish grown in this water. The fish and shellfish were shown to contribute over 99% of the exposure (US EPA, 1978, 1979).

Based on the upper 95% confidence limits of the NAS risk estimate (3.1×10^{-6}) and assuming an acceptable risk of 1×10^{-6} and a consumption of 2L water per day, a criterion of 0.1 ug/L for PCBs in water is derived in order to protect human health. This is based on exposure to PCBs in drinking water alone. Although the evidence demonstrates that other exposures are very important, e.g. fish, there are insufficient data available to reliably include this factor for the Australian situation. The criterion based on exposure through drinking water alone is therefore proposed and this approach is consistent with that used for other compounds in this document.

SUMMARY OF PROPOSED CRITERIA FOR PCBs

Protection of the aquatic environment - 0.004 ug/L

Production of fish suitable for human consumption - 0.002 ug/L

Protection of human health (drinking water quality) - 0.1 ug/L

Other related compounds

Other highly chlorinated compounds are produced for a number of uses. These include polychlorinated terphenyls (PCTs), polybrominated biphenyls (PBBs) and polychlorinated napthalenes (PCNs) (Fishbein, 1979). These compounds are beginning to be detected in the aquatic environment since, like all highly halogenated compounds they tend to be very stable. PCNs have been detected in sediments (Crump-Wiesner et al., 1974) and PCTs have been detected in the effluent of a manufacturing plant (Stratton & Sosebee, 1976). The levels of these compounds in the Japanese environment were reported to be very low (Doguchi, 1977). PBBs have caused extensive pollution of a localised environment due to a mix up in stock feed additives. A PBB flame retardant was inadvertently substituted for a sweetener in a batch of feed additive (Carter, 1976). A number of persons associated with this incident were also affected (Selikoff, 1979).

A survey of the literature on PCNs indicated that they are a moderate hazard to the environment (Kover, 1975). Because of their increasing usage (PCNs are replacing PCBs (Green & Neff, 1977)) it is prudent to monitor these compounds in the environment and to derive water quality criteria for the protection of the various water uses. Some toxicological and bioaccumulation data are available for PCTs (Fishbein, 1979), PBBs (Zitko & Hutzinger, 1976; Zitko, 1977; Fishbein, 1979) and PCNs (Sundstrom et al., 1975; Green & Neff, 1977; Walsh et al., 1977) but this information is inadequate for criteria derivation at present.

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6. DETAILED CRITERIA FOR POLYNUCLEAR AROMATIC HYDROCARBONS (PAH)

General

Polynuclear or polycyclic aromatic hydrocarbons (PAH) are aromatic compounds with a structure which includes fused benzene rings. They are a large group of compounds which are widespread throughout the environment.

The sources of PAH are many. They are generally formed by incomplete combustion of organic material and are therefore a product of virtually any process which involves heating of organic matter, e.g. the internal combustion engine, incinerators (Eiceman *et al.*, 1979), fires, including the burning of plastics (Liao & Browner, 1978), petroleum refinery operations, coal tar and coke production, and the cooking of food. They also occur naturally in substances such as coal and oil. The formation of PAH by organisms has been indicated by many workers but this source is still questionable (Neff, 1979b).

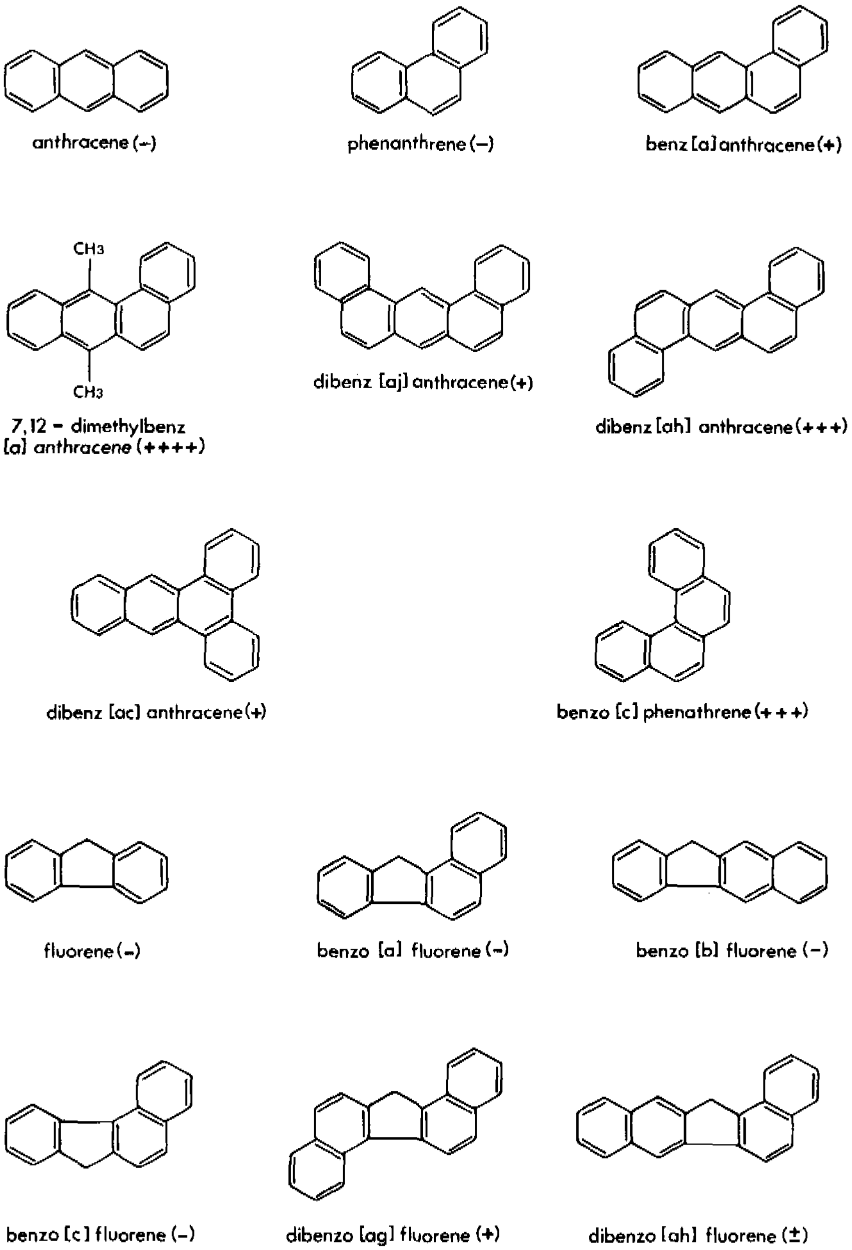
The combustion of fossil fuels would appear to be one of the most important sources of PAH. Youngblood & Blumer (1975) suggested natural fires to be the major source of PAH in the environment. Other important sources in relation to human exposure are cigarette smoking (Lee *et al.*, 1976; Severson *et al.*, 1976), and charcoal grilled and smoked foods (Panalaks, 1976). Badger *et al.* (1960) proposed a mechanism for PAH formation involving free radicals formed during pyrolysis.

Nearly all sources of PAH can contribute these compounds to the aquatic environment. PAH can be discharged in the effluent of petroleum and coal industries. Atmospheric deposition must be important judging by the widespread occurrences of PAH in the atmosphere (Neff, 1979c) and PAH have been detected in rainwater (Piet *et al.*, 1975; Monarca *et al.*, 1979).

Road runoff has been shown to contribute PAH to the aquatic environment, through an accumulation of PAH from automobile exhausts and tyre wear, and leaching from bituminous surfaces (Andelman & Suess, 1970; Andleman & Snodgrass, 1974). PAH levels in soil have been correlated with traffic density (Blumer *et al.*, 1977). PAH in sediments have been correlated with urbanisation which is a reflection of the significance of anthropogenic in relation to natural sources (Hites *et al.*, 1977; Laflamme & Hites, 1978).

Sources of PAH and their fate in, and effects on, the aquatic environments have been discussed in a number of reviews (Andelman & Suess, 1970; Andelman & Snodgrass, 1974; Harisson *et al.*, 1975; Blumer, 1976; Suess, 1976; Neff, 1979a).

Fig. 1 Structures of some PAH and their carcinogenity (carcinogenicity as cited by Neff (1979a))



anthracene (–)

phenanthrene (–)

benz [a] anthracene (+)

7,12 – dimethylbenz
[a] *anthracene* (++++)

dibenz [aj] anthracene (+)

dibenz [ah] anthracene (+++)

dibenz [ac] anthracene (+)

benzo [c] phenathrene (+++)

fluorene (–)

benzo [a] fluorene (–)

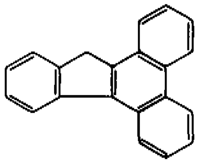
benzo [b] fluorene (–)

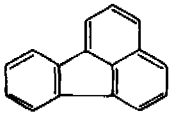
benzo [c] fluorene (–)

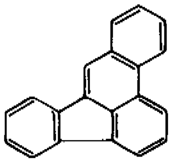
dibenzo [ag] fluorene (+)

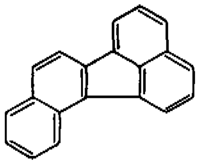
dibenzo [ah] fluorene (±)

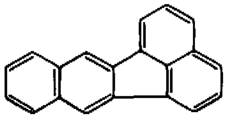
Fig. 1 Continued

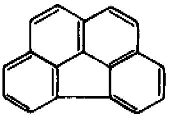

dibenzo [ac] fluorene (±)

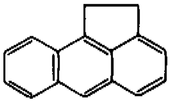

fluoranthene (–)

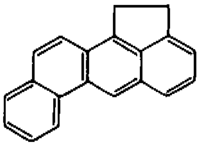

benzo [b] fluoranthene (++)

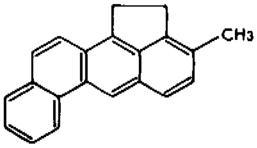

benzo [j] fluoranthene (++)


benzo [k] fluoranthene (–)

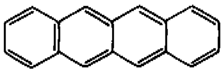

benzo [ghi] fluoranthene (–)

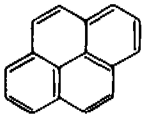

aceanthrylene (–)


benz [j] aceanthrylene
≡ cholanthrene (++)



3-methylcholanthrene (++++)


napthacene (–)


pyrene (–)

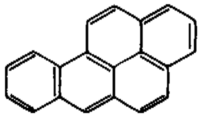
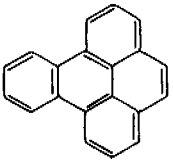
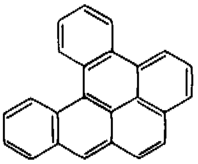

benzo [a] pyrene (+++)

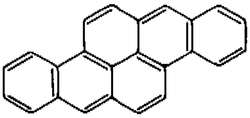
Fig. 1 Continued



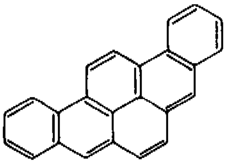
benzo [e] pyrene (−)



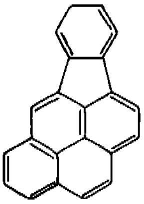
dibenzo [al] pyrene (±)



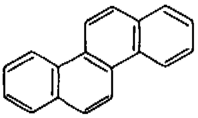
dibenzo [ah] pyrene (+++)



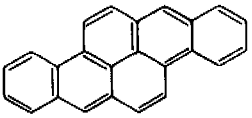
dibenzo [ai] pyrene (+++)



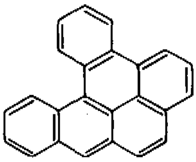
indeno [1,2,3 − cd] pyrene (+)



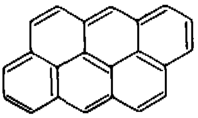
chrysene (±)



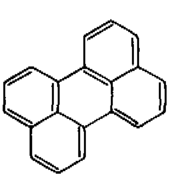
dibenzo [b,def] chrysene (++)



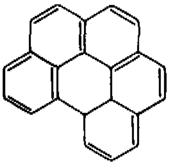
dibenz [def,p] chrysene (+)



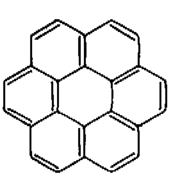
dibenz [def,mno] chrysene (−)
≡anthanthrene



perylene (−)



benzo [ghi] perylene (−)



coronene (−)

−, , non carcinogenic
±, uncertain or weakly carcinogenic
+, carcinogenic
++, +++, ++++, strongly carcinogenic

Since many of the processes which give rise to PAH are extremely common, it is not surprising that these compounds are widespread in the environment. Since PAH are present in naturally occurring substances such as coal and oil and are formed in processes which have existed since the dawn of time, e.g. fire, they may almost be considered naturally occurring compounds in the environment. Thus it would seem that man has always been exposed to PAH.

The concern for PAH in the environment arises from the potent carcinogenicity of some of these compounds (IARC, 1973; Dipple, 1976; Fredenthal & Jones, 1976). Most carcinogenic PAH have 3 to 6 rings. The structures and carcinogenicity of a number of PAH are shown in Figure 1. The correlation between cigarette smoking and cancer (Doll, 1977a, b) is not surprising then, nor is the correlation between petroleum industry activities and cancer (Blot et al. 1977), since both activities contribute to PAH exposures.

Residues in the aquatic environment

The reported occurrences of PAH in the aquatic environment have been documented in reviews by US EPA (1978) and Neff (1979a). The strong tendency for PAH to adsorb onto particulate matter has resulted in most residues being concentrated in sediments. Under anaerobic conditions PAH tend to be stable but microbiological degradation is possible under favourable conditions (Neff, 1979d; Gardner et al., 1979). Photochemical decomposition is also an important process under some conditions (Neff, 1979d). Some of the more recent determinations of PAH in the aquatic environment are documented in Table 34 and indicate the ubiquitous nature of these compounds.

Aquatic toxicology and criteria for the protection of the aquatic environment

Little toxicity data for PAH are available. Most have been summarised by Neff (1979e) and they indicate relatively low toxicity for PAH to aquatic organisms in the short term. Therefore the low levels of PAH found in aquatic environments do not appear to pose an acute toxicity threat. The potent carcinogenicity of some PAH creates concern as to the effects of chronic exposures however. Tumours in aquatic organisms and their correlation with pollution are well documented (Kraybill et al., 1977). In a few cases PAH have been implicated as the cause. Tumours in salamanders in a sewage lagoon were associated with high levels of perylene (Rose & Harshbarger, 1977). Tumours in fish in a polluted river were associated with PAH pollution, but other pollutants such as chlorinated hydrocarbons and heavy metals were present (Brown et al, 1973). At this stage the evidence of PAH residues in the environment being a cause of tumours in aquatic organisms is inconclusive.

TABLE 34 RECENT DETERMINATIONS OF PAH IN THE AQUATIC ENVIRONMENT

LOCATION	DETERMINED IN	REFERENCE
AUSTRALIA	Sediments	Maher _et al_., 1979
BELGIUM	Groundwater	Quaghebeur & De Wulf, 1978
CANADA	Drinking water	Benoit _et al_., 1979
	Mussels	Dunn, 1976
	Fish and shellfish	Dunn & Fee, 1979
	Recycle water from coke oven plant	Das & Thomas, 1978
FRANCE	Mussels	Bories _et al_., 1976
GERMANY	Snow	Schrimpff _et al_., 1979
ITALY	Mussels	Fossato _et al_., 1979
NETHERLANDS	Water	Piet _et al_., 1975
NORWAY	Mussels, sediments	Bjorseth _et al_., 1979
SWITZERLAND	Sediments, suspended matter	Giger & Schaffner, 1978
UNITED KINGDOM	Water, sewage	Acheson _et al_., 1976
	Water (rain, river, drinking)	Monarca _et al_., 1979
	Water, suspended matter	Lewis, 1975
	Sediments	Cooke _et al_., 1979
USA		
Massachusetts	Sediment core	Hites _et al_., 1977
Oregon	Mussels	Mix & Schaffer, 1979
Texas	Shellfish	Guerro _et al_., 1976
New York	Mussels	Heit _et al_., 1980
Off East Coast	Fish	Brown & Pancirov, 1979
	Fish, shellfish	Pancirov & Brown, 1977
Various	Raw, drinking water	Saxena _et al_., 1977 Basu & Saxena, 1979
	Sediments	Heit, 1979
Sub-Antarctic Island	Sediments	Platt & Mackie, 1979
Various (World Wide)	Sediments	Laflamme & Hites, 1978

The available toxicity data were considered inadequate for criteria derivation by the US EPA (1978, 1979). From a consideration of more recent data, however, a criterion can be derived. Rossi & Neff (1978) determined 96 hour LC 50 levels for a number of PAH and the polychaete Neanthes arenaceodentata. The most toxic PAH was 1-methylphenanthrene with a 96 hour LC50 of 0.3 mg/L. Other PAH tested had similar LC50 levels. Ott et al. (1978) determined the toxicity of napthalene and some methyl derivatives to the copepod Eurytemora affinis. The most toxic PAH, trimethylnapthalene had a 24 hour LC50 of 0.316 mg/L. Using the LC50 level of 0.3 mg/L and an application factor of 0.01 since PAH have a tendency to accumulate, a criterion of 3 ug/L for the protection of the aquatic environment is derived. 10 ug/L of a PAH (trimethylnapthalene) had adverse effects on a copepod in a long term test (Ott et al., 1978). Thus a criterion of 3 ug/L should afford some protection. This criterion is calculated from the toxicity of the most toxic PAH to the most sensitive organism. It can be used as a criterion for PAH in general as it will afford protection against less toxic PAH. It should be used with caution however because of the limited toxicity data on which it is based.

The availability of PAH adsorbed to particulate matter and sediments must also be considered in assessing the significance of PAH levels and applying derived criteria for water. The data suggest however, that uptake directly from the sediment itself is insignificant and that PAH leached from the sediment to interstitial or overlying water appears to be involved (Roesijadi et al., 1978).

Bioaccumulation and the production of fish suitable for human consumption

PAH show some tendency to accumulate in exposed organisms. A bioaccumulation factor of 10 000 for benz(a)anthracene and Daphnia has been reported. Bioaccumulation factors depend on the nature of the PAH, a factor of only 100 being reported for naphthalene (Southworth et al., 1978). Accumulated residues are eliminated rapidly on termination of exposure. Mussels contaminated with PAH from creosote treated timber were shown to eliminate benzo(a)pyrene with a half life of 16 days. However the short depuration periods used to remove bacterial contamination from mussels would not be effective in removing PAH contamination (Dunn & Stich, 1976). Elimination depends markedly on the nature of the PAH as well; half lives of 2 and 18 days were determined for naphthalene and benzo(a)pyrene elimination respectively from oysters (Lee et al., 1978). The data for anthracene suggest that elimination from Daphnia is a complex process but nevertheless a high proportion is eliminated rapidly (Herbes & Risi, 1978).

Fish were also reported to accumulate napthalene and 2-methylnapthalene with bioaccumulation factors similar to those reported for mussels. Elimination was also rapid (Melancon & Lech, 1978). Lee et al. (1972) also reported PAH to be taken up rapidly by fish but detoxified and eliminated rapidly. Lu et al. (1977) showed that the fish accumulated PAH from the food chain. Uptake from water varied according to the organism and was related to the presence of microsomal oxidase. Thus PAH have the potential for bioaccumulation in exposed organisms, the degree depending on the nature of the PAH but elimination is rapid on termination of exposure. As no maximum residue limit for PAH in aquatic products for human consumption hase been set, it is not possible to relate the bioaccumulation data to a water concentration in order to meet such a limit.

Criterion for the protection of human health

Some effort has already been expended in developing criteria and standards for PAH in drinking water. WHO has recommended that the concentration of six PAH (fluoranthene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, benzo(ghi)perylene and indeno(1,2,3-cd)pyrene) should not exceed 0.2 ug/L level (WHO, 1971). No basis for this is given, so no comment can be made here. The proposed EEC drinking water standard (maximum admissible concentration) for PAH is 0.2 ug/L for the same six reference PAH (EEC, undated) and presumably is taken directly from the WHO limit. WHO propose to re-examine the data for PAH and re-establish a maximum acceptable level for drinking water (Suess, 1980). The NAS (1977) reviewed the toxicological data for the carcinogenic PAH, benzo(a)pyrene, but considered the data inadequate for deriving a drinking water quality criterion.

The US EPA has reviewed the toxicological data for PAH and derived criteria for benzo(a)pyrene and dibenzanthracene corresponding to various risks. These criteria were based on exposure to PAH both from drinking water and from contaminated fish and shellfish (US EPA, 1978, 1979). The criterion derived for benzo(a)pyrene using the data of Neal and Rigdon (1967), who found gastric tumours in mice fed benzo(a)pyrene, was selected as a criterion for PAH in general, since benzo(a)pyrene was one of the more potent PAH, and a criterion based on this compound should protect against the less carcinogenic PAH. Interactive effects of PAH were however ignored since it was not possible to quantitatively include them.

The US EPA draft criterion of 0.97 ng/L corresponding to a risk of 1×10^{-6} for benzo(a) pyrene and PAH in general attributes 78% of the intake from water and 22% of the intake from fish and shellfish grown in that water. Thus for exposure to PAH in drinking water alone a criterion of 1.24 ng/L corresponding to a risk of 1×10^{-6} can be derived. Since exposures through other sources cannot be completely validated a criterion through exposure in drinking water alone is proposed, i.e. 1 ng/L. This approach is consistent with the approach taken throughout this document.

Two aspects of PAH in drinking water warrant discussion. These are the effects of water treatment processes on PAH levels, and the possibility of drinking water supplies being contaminated by PAH while in the distribution system.

Benoit et al. (1979) reported variable efficiencies of water treatment plants in the removal of PAH. No details of the treatment processes were given but higher levels of PAH were reduced more effectively than lower levels. Some data for the Netherlands also indicated substantial reduction of PAH levels in some cases through various treatment processes, but the results were variable (Piet et al., 1975). Chlorination for removal of PAH has also been studied and found to be efficient (Harrison et al., 1976; Perry & Harrison, 1977). Ozonation was also effective in reducing PAH levels (Andelman & Snodgrass, 1974; Harrison et al., 1975). Basu & Saxena (1978) and Saxena et al. (1977) also reported efficient removal of PAH in water treatment processes. Since a significant proportion of PAH is associated with particulate matter, other simple conventional water treatment processes such as sedimentation, flocculation and filtration can also effectively remove PAH (Andelman & Snodgrass, 1974; Harrison et al., 1975).

PAH are present in bituminous substances (Borneff & Kunte, 1965; Schamp & Van Wassenhove, 1972), and coal tar (Popl et al., 1974) which may be used in distribution systems as coatings for pipes and tanks. Borneff & Kunte (1965) studied this problem and found appreciable PAH to be leached from bitumen into water, but hardening and leaching of coatings reduced the PAH extracted with time. Similarly Alben (1980) found that PAH were leached from coal tar used to coat water storage tanks. The amount leached was substantially reduced from coatings which had been in service for 5 years. This source of contamination of drinking water supplies has as yet received surprisingly little attention.

It is interesting to compare exposures to PAH from drinking water at the criterion level (2 ng per day for a 2 litre/day water consumption) with exposures from other sources. Heavily polluted water has been estimated to contribute only about 0.1% of the amount contributed by food to human exposures (Shabad & Il'nitskii, 1970). The data of Lee et al. (1976) and Severson et al. (1976) indicate considerably more than 2 ng of benzopyrenes is present in the smoke of one cigarette. Smoked foods and charcoal grilled meats can contain up to 15 and 30 ng/kg benzo(a)pyrene respectively (Panalaks, 1976). Thus very little of this food would need to be consumed before an intake of 2 ng was reached. Using the maximum value of 0.65 ug/100 m^3 reported for benzo(a)pyrene in Sydney air by Cleary (1963), an intake of 65 ng per day is derived, assuming 10 m^3 of air is breathed per day. Bridbord et al. (1976) made a more detailed examination of human exposure to PAH. Restaurant employees were calculated to have a daily intake of PAH equivalent to 2 packs of cigarettes. Coke oven and coal tar pitch workers had the greatest exposures.

Levels in unpolluted water would appear to be much greater than the criterion level. Background levels in groundwaters are of the order of 10-15 ng/L (Suess, 1980). A study of Belgian aquifers revealed somewhat lower levels, but these were generally greater than 1 ng/L (Quaghebeur & De Wulf, 1978). Levels in Canadian (Benoit et al., 1979) and US (Basu & Saxena, 1978) drinking waters were also above the criterion level.

On the basis that water meeting the criterion level of 1 ng/L would contribute very little to human PAH exposure and that levels in unpolluted water may exceed this level, it may be unrealistic to use this figure as a basis for a standard. If 1 ng/L is an accurate estimate of a safe level, then there is need for improved water treatment practices and reduction of exposures from other sources.

SUMMARY OF PROPOSED CRITERIA FOR PAH

Protection of the aquatic environment - 3 ug/L

Protection of human health (drinking water quality) - 1 ng/L

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7. DETAILED CRITERIA FOR TRIHALOMETHANES

General

The trihalomethanes (THMs) comprise a number of compounds ranging from trifluoromethane (CHF_3) through to iodoform (CHI_3) and include various mixed halogenated members, e.g. bromodichloromethane ($CHCl_2Br$). The most important THM is chloroform ($CHCl_3$) which has widespread industrial uses as a solvent, and is used in the manufacture of the refrigerant and aerosol propellant chlorodifluoromethane (CHF_2Cl), and in the manufacture of teflon (Fishbein, 1979a,b). Over 1.5 million litres of chloroform were imported into Australia in the 1977-78 financial year (Australian Bureau of Statistics, 1979). The trihalomethanes most frequently detected in water supplies are chloroform, bromodichloromethane, dibromochloromethane ($CHClBr_2$) and bromoform ($CHBr_3$). They are formed during chlorination of drinking water for disinfection purposes.

Although chlorination of drinking water for disinfection was practiced as early as 1908 (NAS, 1977a), it was not until 1974 that it was reported, almost simultaneously, by Rook (1974) and Bellar et al. (1974), that THMs were formed in this process. Since that time there have been several surveys of THMs in drinking water supplies. Of particular interest was the National Organics Reconnaissance Survey (NORS) of the drinking water of 80 US cities carried out by the US EPA. Maximum levels of $CHCl_3$, $CHCl_2Br$, $CHClBr_2$, $CHBr_3$ found were 311, 116, 100 and 92 ug/L respectively (Symons et al., 1975). Chloroform is generally the THM formed in highest concentration although the brominated species may predominate in waters with high bromide levels. Iodinated compounds were also detected in one investigation (Bunn et al., 1975). An intermediate in the haloform reaction, 1,1,1-trichloroacetone, has been identified in chlorinated waters from the USA (Suffet et al., 1976) and this compound has been identified in South Australian water supplies (Engineering and Water Supply Department, South Australia, unpublished data).

Few data have yet been published for THM levels in Australian water supplies although some routine monitoring programs are in operation. The highest THM level recorded in South Australia was 1120 ug/L on Yorke Peninsula. This level is comparable with the highest level reported for water supplies overseas (922 ug/L in Texas, USA) (Glaze & Rawley, 1979). The average THM level for the supplies monitored in South Australia during 1976-79 was 207 ug/L (Lane, 1980).

The formation of THM through chlorination for disinfection occurs by means of the reaction of chlorine with the naturally occurring humic and fulvic acids in water (Trussell & Umphres, 1978). A substantial amount of literature concerning THM formation has been published recently. Although this is important to the problem of THM in drinking water supplies, it is not sufficiently relevant to discuss in detail in this report.

Residues in the aquatic environment

Low levels of THMs and other volatile chlorinated hydrocarbons (VCHs) have frequently been detected in non-chlorinated waters. Levels of THMs tend to be very low in comparison with the levels formed during chlorination and are generally a reflection of contamination from industrial sources, as a number of compounds which are not formed in the chlorination process are often detected. VCHs including chloroform have been recorded in remote areas such as the Atlantic Ocean (Murray & Riley, 1973a, b; Lovelock et al., 1973). Many of the reports dealing with formation of THMs by chlorination also record the low THM levels in the raw water supplies. The occurrence of chloroform in foods and human tissue (McConnell et al., 1975), air and marine organisms (Pearson & McConnell, 1975), and its widespread occurrence in water (McConnell, 1976) suggests that it is as ubiquitous in the environment as DDT and PCBs.

Aquatic toxicology and criteria for the protection of the aquatic environment

Levels of THMs in the aquatic environment are low and do not appear to be of any concern since toxicities to aquatic organisms are low e.g. chloroform has a 96 hour LC50 for the dab, *Limanda limanda* of 28 mg/L (Pearson & McConnell, 1975). An important exception to this generalisation may be the levels generated by disinfection of power plant cooling water. Both ozone (Helz et al., 1978) and chlorine (Helz & Hsu, 1978; Helz et al., 1978) produce bromoform on treatment of seawater. This occurs through the oxidation of bromide to hypobromous acid, which reacts readily with organic matter. Negligible amounts of chloroform are produced (Helz & Hsu, 1978: Helz et al., 1978) which is in contrast to freshwaters where chloroform is generally the predominant compound formed. Thus cooling water discharged by these plants may pose a hazard due to high bromoform levels.

The US EPA has reviewed the toxicity data available for chloroform on aquatic organisms and derived a draft criterion for the protection of freshwater aquatic life of 500 ug/L as a 24 hour average and 1200 ug/L not to be exceeded at any time. The corresponding values for saltwater aquatic life were 620 and 1400 ug/L respectively (US EPA, 1978a, 1979a). Few acute toxicity data were available and only one chronic study had been performed; a lifecycle test with *Daphnia magna* yielded a chronic toxicity value of 2.546 mg/L (US EPA, 1978a). More recent work on the toxicity of chloroform and bromoform to oyster larvae showed survival to be adversely affected at concentrations as low as 50 ug/L (Stewart et al., 1979). This suggests that a lowering of the criterion may be warranted. A criterion of 50 ug/L total THM is therefore proposed in order to protect the aquatic environment.

Criteria for the protection of human health

The prime concern for THMs is in relation to drinking water. Both chloroform (Eschenbrenner & Miller, 1945; National Cancer Institute, 1976) and bromoform (Theiss *et al.*, 1977) have been shown to be carcinogenic in laboratory animals.

The toxicological, including carcinogenicity, data for chloroform have been extensively reviewed in a number of publications (NAS, 1977b; US EPA, 1978a, c). The carcinogenicity data has also been discussed by Tardiff (1977). Although chloroform was not mutagenic in the Ames test, the brominated compounds were (Simmon & Tardiff, 1978). Since bromoform was carcinogenic in one assay in which chloroform was not, it was suggested that bromoform may be a more potent carcinogen (Theiss *et al.*, 1977). No data have yet been reported to substantiate this, but the mutagenic activity of bromoform lends weight to this suggestion. It therefore appears that the brominated THMs should be cause for concern but there is little toxicological information yet available for these compounds.

The US EPA has proposed a maximum contaminant level (MCL) of 100 ug/L for trihalomethanes in drinking waters serving communities with populations greater than 75 000 (US EPA, 1978b). This limit was based not only on a consideration of the toxicological data, but also the feasibility of meeting that limit with existing water treatment technology. More recently this MCL has been implemented and made law for supplies serving communities of greater than 10 000 people (US EPA, 1979b). The level of 100 ug/L imposed in the USA is purely a requirement set by government; it is not a criterion since it is not based solely on toxicological data.

Since THMs are possible human carcinogens, criteria should be based on risk estimation procedures consistent with current practice. A number of risk estimates of cancer induction by exposure to low levels of chloroform have been made using various models (NAS, 1977b; US EPA, 1978a,c). This is the only THM with sufficient data available for these calculations to be made. Risk estimates calculated by Tardiff (1977) were based on an exposure of 10 ug/kg/day and are shown in Table 35. Assuming a 70 kg individual consumes 2L/day of water, this intake corresponds to a chloroform concentration of 350 ug/L. A level giving the same relative exposure to infants would be lower by a factor of approximately five, i.e. 70 ug/L. The National Academy of Sciences estimate of lifetime risk of developing cancer through ingestion of chloroform is $1.5\text{-}17.0\times10^{-7}$ per ug/L per litre of water consumed daily, with upper 95% confidence limits of $3.0\text{-}22.0\times10^{-7}$ (NAS, 1977b). The US EPA Science Advisory Board (SAB) derived a lifetime risk estimate of $0\text{-}100\times10^{-5}$ for developing liver cancer for a 70 kg individual ingesting 4 litres of water per day with a chloroform level of 300 ug/L (US EPA, 1978c). The US EPA Carcinogen Assessment Group (CAG) calculated that at the current levels of chloroform in US waters, between 23 and 207 cancers per year were induced in the human population of that country (US EPA, 1978c).

TABLE 35. RISK ESTIMATES FOR CHLOROFORM INGESTION OF 10 ug/kg/day (2L of water per day containing 350 ug/L chloroform) (Calculated by Tardiff, 1977).

MODEL	BASIS	MAXIMUM RISK x 10^6 Year^{-1}	MAXIMUM RISK x 10^6 Lifetime^{-1}
Probit-log (slope = 1)	rat	0.016-0.040	1.12-2.8
	mouse	0.016-0.683	1.12-47.81
Probit-log (actual slope)	rat	1×10^{-3}	7×10^{-2}
	mouse	1×10^{-3}	7×10^{-2}
Linear (one-hit)	rat	0.420	29.4
	mouse	0.840	58.8
Two-step	rat	0.267	18.69
	mouse	0.283	19.81

The US cancer rates for liver and kidney cancers are 52.5 and 29.2/million/year respectively, or 81.7/million/year total (Tardiff, 1977). Kidney and liver cancers are the most likely to be induced in the human population by chloroform, since these are the organs affected in mice and rats. The CAG figures for chloroform therefore reduce to only 0.144-1.29/million/year, assuming a population of 160 million is exposed to chloroform in drinking water. Thus chloroform would be responsible for only 0.18-1.6% of US kidney and liver cancer deaths per year, based on the CAG model.

The maximum risk (100×10^{-5}) of developing liver tumours calculated by the SAB for chloroform ingestion at 300 ug/L would contribute 40% of the total US liver cancers (US EPA, 1978c). It should be noted that a figure of 260×10^{-5} as the lifetime incidence of liver cancer is used in this calculation. This is equivalent to an incidence of 37 per million per year which is different from the figure of 52.5 per million per year quoted by Tardiff (1977).

The US EPA (1978a, 1979) has recently calculated a draft criterion of 0.21 ug/L corresponding to a lifetime risk of developing cancer of 1×10^{-6} for exposure to chloroform in water. This was based on both consumption of water and consumption of 18.7g/day fish and shellfish grown in that water. 12% of the exposure resulted from consumption of fish and shellfish. Thus a criterion of 0.24 ug/L for a risk of 1×10^{-6} can be calculated for exposure to chloroform in drinking water alone.

Canadian authorities have recommended a maximum acceptable concentration of 350 ug/L for trihalomethanes in water. This was based on the risk estimated by Tardiff for chloroform using the linear (one-hit) model, which, in turn was based on the rat-kidney cancer data. The maximum acceptable concentration of 350 ug/L corresponds to a risk of 1 in 2.5 million per year for a 70 kg person drinking 2 litres of water per day (Health & Welfare, Canada, 1980).

Based on the upper value of the upper 95% confidence limits of the NAS risk estimate (22.0×10^{-7}), a criterion of 0.23 ug/L for chlorform can be derived for a 2L /day consumption of water and a risk of 1×10^{-6}. Since 1×10^{-6} might be considered an acceptable risk, a criterion of 0.2 ug/L is proposed. This is identical to the criterion calculated from the US EPA draft criterion, assuming exposure from drinking water alone. Table 36 shows the criteria for chloroform derived from the various risk estimates. Also included is the criterion based on the minimum-effect dose in the rat, a safety factor of 5 000 and assuming 20% of the daily intake is derived from water. This is an approach used in this document when data indicate carcinogenicity, but are inadequate for risk derivation.

TABLE 36. DRINKING WATER QUALITY CRITERIA FOR CHLOROFORM AS DERIVED FROM REPORTED RISK ESTIMATES

MODEL	CALCULATED FROM	DOSE	ESTIMATED MAXIMUM LIFETIME RISK x 10^6	DERIVED BY	CRITERION (ug/L)
Margin of safety	Minimum-effect dose (kidney carcinoma) in the rat (90mg/kg/day) and a safety factor of 5000: 20% of the intake is in the water	-	-	-	140
Probit-log (Slope = 1)	Rat: kidney cancer	0.01mg/kg[a]	1.12 - 2.8	Tardiff (1977)	120 - 310
	Mouse: liver cancer	"	1.12 - 47.81	"	7.3 - 310
Probit-log (actal slope)	Rat: kidney cancer	"	7×10^{-2}	"	5000
	Mouse: liver cancer	"	7×10^{-2}	"	5000
Linear (one-hit)	Rat: kidney cancer	"	29.4	"	12
	Mouse: liver cancer	"	58.8	"	6.0
Two-step	Rat: kidney cancer	"	18.69	"	18
	Mouse: liver cancer	"	19.81	"	18
Linear	NCI mouse and rat data, and mouse and rat data of Roe	1 ug/L	0.3 - 2.2[b]	NAS (1977)	0.23 - 1.7[c]
Linear	Eschenbrenner & Miller, 1945 data	300 ug/L	0-1000	SAB(US EPA, 1978c)	0.3[d]
?	NCI mouse and rat data	?	10.1 - 90.5	CAG(US EPA, 1978c)	-
Modified one-hit	NCI mouse data	0.24 ug/L	1	US EPA (1978a)	0.24

(a) 350 ug/L for a 70 kg individual consuming 2 litres of water per day

(b) Upper 95% confidence limits

(c) Assuming a 2L/day consumption

(d) 70 kg individual consuming 4L/day water

NAS (1977c) reviewed the data for bromoform and considered it inadequate for criterion derivation. For the other brominated THMs the data are also inadequate for criteria derivation. However since the brominated THMs might be equally as hazardous as chloroform, the criterion for chloroform should apply to THMs in total until adequate data are available for brominated THMs.

A number of epidemiological studies have been performed in an attempt to correlate cancer mortality with THM levels. Kuzma et al. (1977) found a correlation between some cancers and the nature of the water supply in Ohio, viz, rates were higher in areas served by surface waters in comparison with ground waters. As surface waters generally have higher THM levels than ground waters, a relationship between THM content and cancer is suggested. In certain counties in New York, USA a correlation between gastrointestinal and urinary tract cancers, and water chlorination was reported (Alavanja et al., 1978). Cantor et al. (1978) found a correlation between certain cancers and THM levels in US water supplies, and Carlo & Mettlin (1980) reported a correlation between pancreatic cancer in white males and THM levels in Erie County, New York.

The epidemiological studies, mostly unpublished, have been reviewed in several documents (US EPA 1978a; Wilkins et al., 1979). Because of the difficulties in performing such studies, the evidence for THMs in water being a cause of human cancer is not strong, even though some correlation between cancer and water chlorination exists. At the present time the evidence can best be judged as suggestive but inconclusive, both for THMs and organics in general being a cause of cancer through ingestion in water. More studies and more refinements in the treatment of the data are needed before the effects of low levels of THMs in drinking waters can be ascertained.

Bromoform and other brominated compounds are found in an edible seaweed from Hawaii and there is no evidence to suggest that consumption of this seaweed has had detrimental effects on the consumers (Burchfield & Storrs, 1977). Although this is not proof that low levels of THMs are harmless, it is suggestive that the effects in humans may not be as severe as inferred from animal data.

SUMMARY OF PROPOSED CRITERIA FOR THMs

Protection of the aquatic environment - 50 ug/L.

Protection of human health (drinking water quality) - 0.2 ug/L chloroform. This figure should be applied to THMs in general until sufficient data are available for the brominated THMs.

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THE PRODUCTION OF ORGANIC CHEMICALS

The growth in the number of chemicals known to man is remarkable. By November 1977, Chemical Abstracts Service (CAS) computer registry of chemicals contained 4 039 907 distinct entities comprising compounds reported in the literature since 1965, and was growing at the rate of 6 000 per week. The number of compounds in common use was estimated to be 63 000 (Maugh, 1978). Organic chemicals, essentially those containing carbon, comprise the majority of chemicals known to man. Of the chemicals listed in the CAS registry, 96% contained carbon (Maugh 1978) and would be predominantly organic chemicals.

The quantities of chemicals produced are also increasing at a remarkable rate and this is particularly true for organic chemicals. It has been estimated that the world production of organic chemicals increased from 7 million tonnes in 1950 to 63 million tonnes in 1970 (McGinty, 1979). The US production of chemicals, including organic chemicals and some carcinogens has been graphically documented by Davis and Magee (1979). In figure 1 the US production of synthetic organic chemicals is shown.

The wider uses of synthetic materials has resulted in large increases in the production of chemicals used in their manufacture, and Figure 2 shows the US production of polymers (plastics, synthetic fibres and synthetic rubber) for the years 1963-79. Australian production of plastics and synthetic resins is similar although on a smaller scale (Figure 3).

Although not reaching the same high levels of production the rate of increase in manufacture of the far more toxic pesticide chemicals, which are mainly organic chemicals, is equally spectacular. In Figure 4 the increase in the number of pesticides registered for use in the USA is shown and in Figure 5 the US production of pesticide chemicals, and in particular DDT, to 1970. It is estimated that there are some 1 500 different active pesticide ingredients currently available (Maugh, 1978).

Data on pesticide production and usage in Australia are generally not available although the quantities imported of some pesticides, e.g. DDT, BHC, chlordane and lindane, are published by the Australian Bureau of Statistics. Some indication of production and usage can be obtained from the annual values of pesticides produced in Australia which is shown in Figure 6. These data reflect the US trend of large increases in pesticide production where the major increases are in the production of pyrethrin and organophosphate insecticides, and the herbicides 2,4-D and 2,4,5-T. Little change in the level of production of organochlorine insecticides is indicated from the data. The limited data available for organochlorine imports show little change for most pesticides with even a decline in some cases. This parallels the data for US production of DDT and reflects the phasing out of these compounds due to the concern for their persistence in the environment.

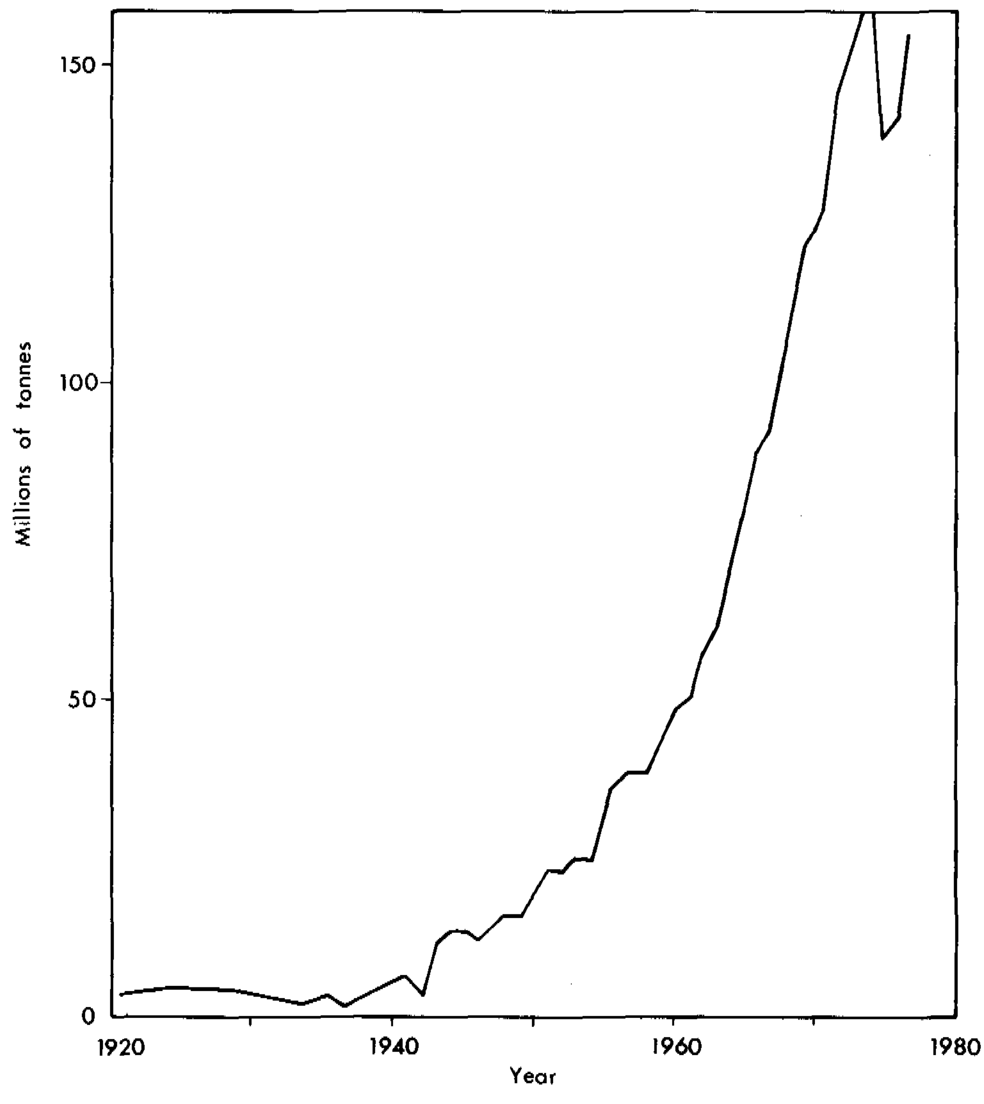

FIG. 1 Total synthetic organic chemicals, annual production (excludes tar, tar crudes, and primary products from petroleum and natural gas)

Taken from Davis & Magee (1979). Copyright 1979 by the American Association for the Advancement of Science

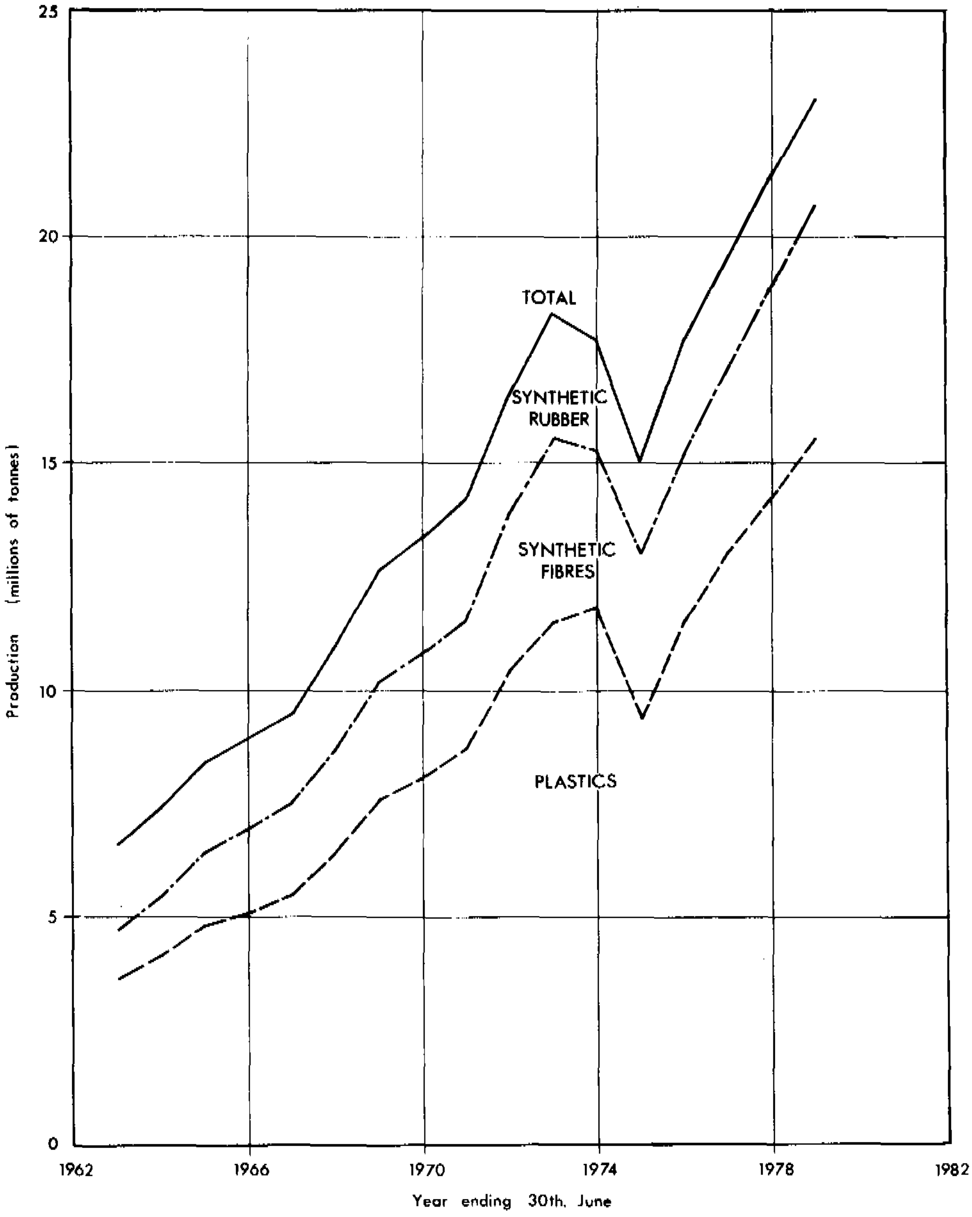

FIG. 2 U.S. Production of plastics, synthetic fibres and synthetic rubber.

Based on data published annually in 'Chemical and Engineering News'

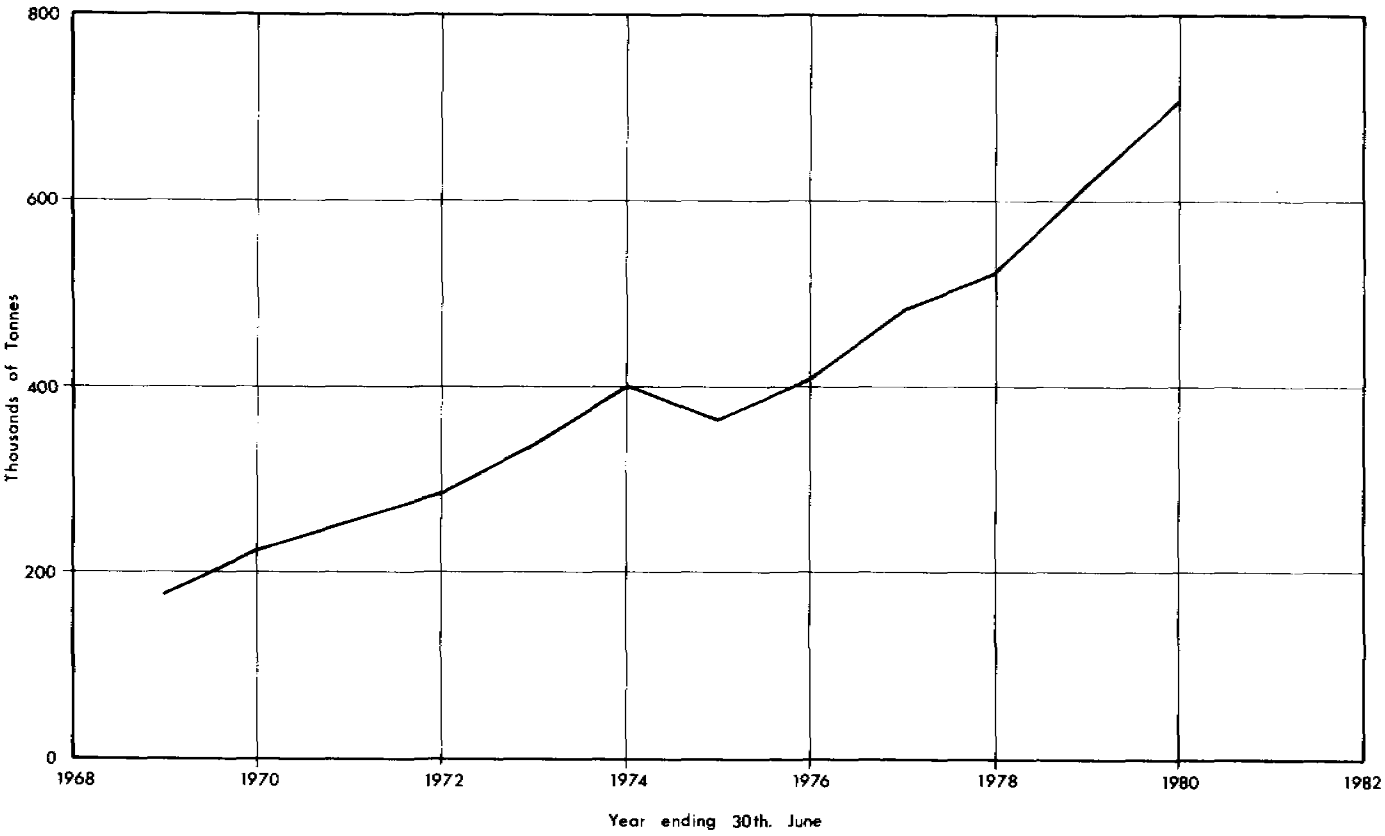

FIG. 3 Australian production of plastics and synthetic resins
(Based on data from Australian Bureau of Statistics)

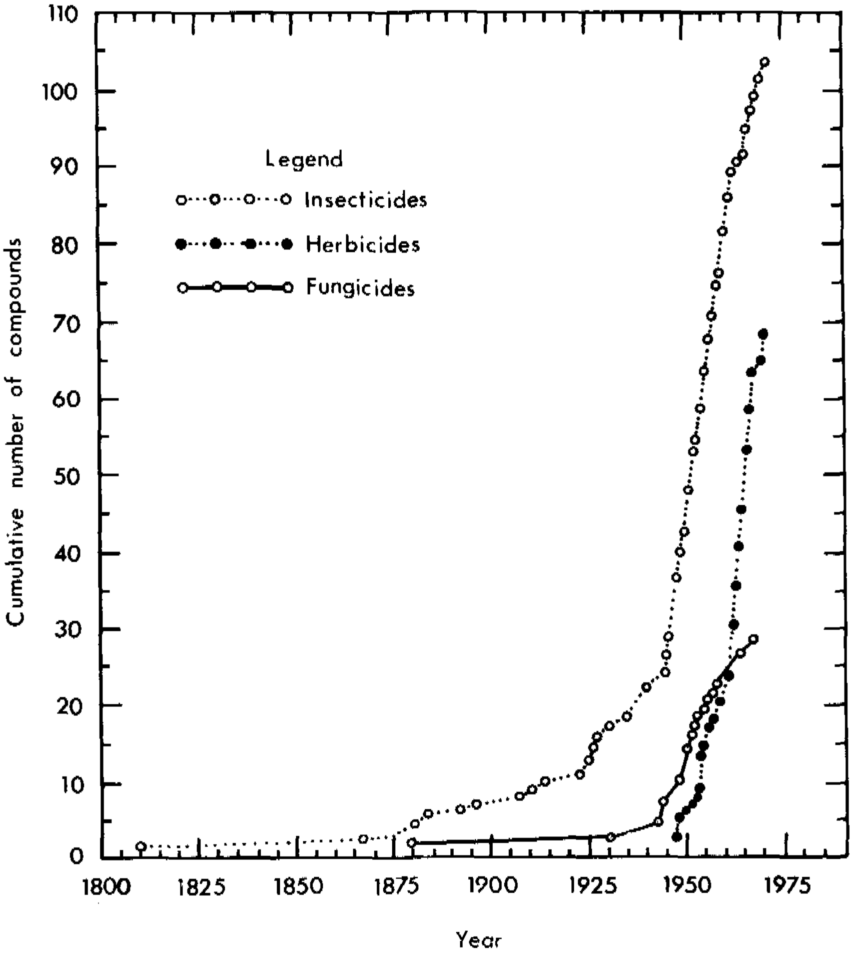

FIG.4 Dates of introduction of some important pesticides

Reprinted from Hayes, W. J., Jnr. (1975), 'Toxicology of Pesticides', Williams & Wilkins Co., Baltimore, with permission of the author.

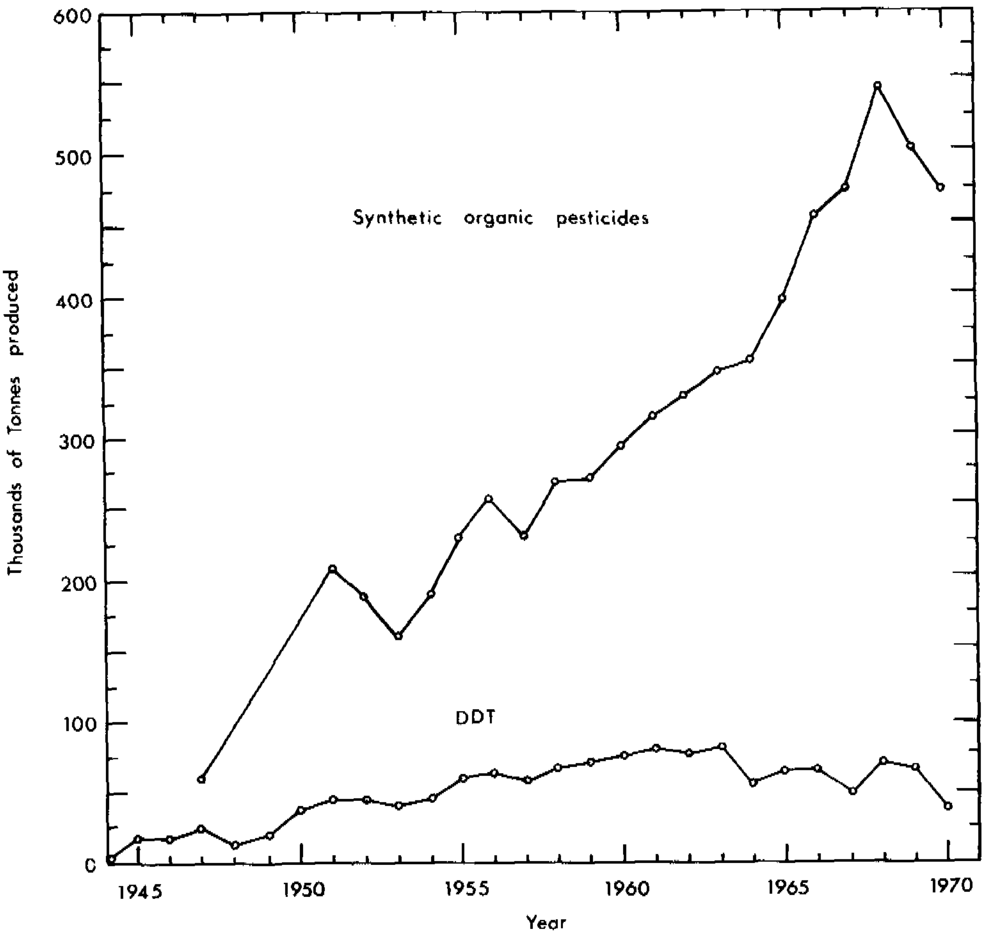

FIG. 5 United States production of DDT and synthetic organic pesticides. Based on data from US Tariff Commission.

Taken from Hayes, W.J., Jnr. (1975) 'Toxicology of Pesticides', Williams & Wilkins Co., Baltimore, with permission of the author.

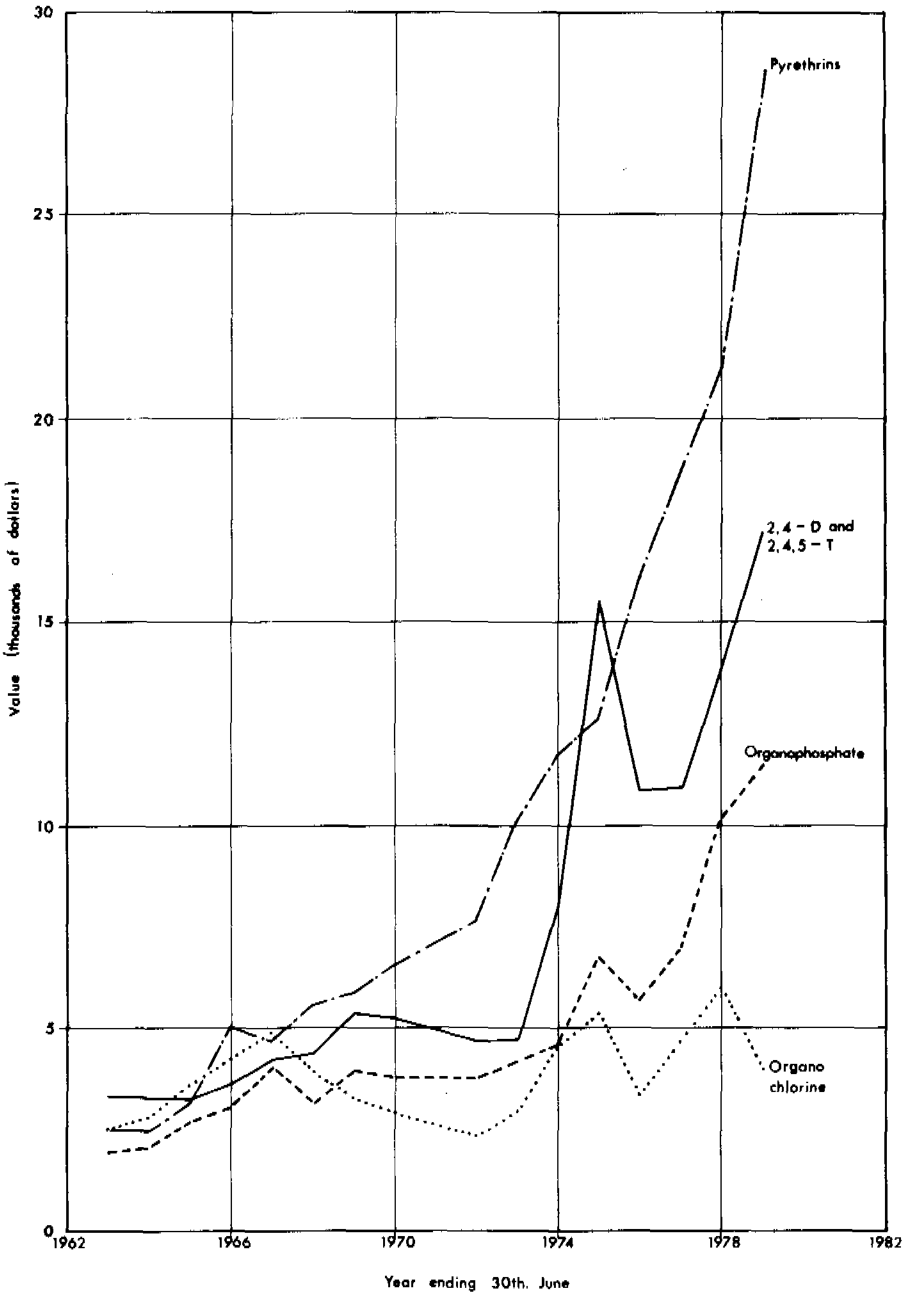

FIG. 6 Value of pesticides produced in Australia.
(Data from Australian Bureau of Statistics)

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