Environmental toxicology

Environment, Ecology

Introduction to Environmental Toxicology A lecture by Dr Rick Leah (Long version of Notes prepared by Dr R T Leah, Biological Sciences, University of Liverpool but including material summarized and adapted from various locations on the www*) Aims The impact of toxic chemicals on wildlife and humans has been of great concern for the last fifty years. Unfortunately this is a very large, complex subject area which can only be covered superficially within the time available.

However, this lecture is intended to give an introduction to fundamental aspects of how some pollutants interact with living organisms to cause deleterious effects. The complexity will be explained and simplified where possible. You should understand at least a little about the biology of key organisms and how pollutants cause damage at a physiological level. You should be aware of how pollutants can induce change in organisms which can be used as a ‘ biomarker’ of the presence and action of the pollutants (although this will form the subject of a later lecture in this course).

Thus as the main outcome of this lecture you should have an appreciation of the wide range of contemporary issues that are caused by toxic chemicals in theenvironmentand what regulatory authorities are doing to monitor and control them. You should understand the main hazards that toxic chemicals pose and how risk to humans and wildlife is controlled. You should be aware of the main groups of pollutants of contemporary concern.

The material covered will be useful for the consideration of two case studies on the impact of toxic chemicals in the Great Lakes of North America and the Baltic Sea in later lectures. [pic] Environmental Toxicology or Ecotoxicology? [pic] Introduction It was after World War II that increasing concern about the

impact of toxic chemicals on the environment led Toxicology to expand from the study of toxic impacts of chemicals on man to that of toxic impacts on the environment. This subject became known as Environmental Toxicology.

Ecotoxicology is a relatively new discipline and was first defined by Rene Truhaut in 1969. It attempts to combine two very different subjects: ecology (" the scientific study of interactions that determine the distribution and abundance of organisms" Krebs 1985) and toxicology (" the study of injurious effects of substances on living organisms", usually man). In toxicology the organisms sets the limit of the investigation whereas Ecotoxicology aspires to assess the impact of chemicals not only on individuals but also on populations and whole ecosystems.

During the early years, the major tools of Environmental Toxicology were: detection of toxic residues in the environment or in individual organisms and testing for the toxicity of chemicals on animals other than man. It was however, a very big jump in understanding from an experimental animal to a complex, multivariate environment and the subject of ECOTOXICOLOGY developed from the need to measure and predict the impact of pollutants on populations, communities and whole ecosystems rather than on individuals.

There is an on-going debate as to the exact scope and definition of ecotoxicology. The simplest definition found to date is that ecotoxicology is " the study of the harmful effects of chemicals upon ecosystems" (Walker et al, 1996). A more complete definition of Ecotoxicology comes from Forbes & Forbes 1994 “ the field of study which integrates the ecological and toxicological effects of chemical pollutants on populations, communities and ecosystems with the fate (Transport, transformation and breakdown) of such

pollutants in the environment". nd several books have been written recently which discuss this in some depth, these include: Cairns, J Jr & Niederlehner B R (1994) Ecological Toxicity Testing. CRC Press Inc: Boca Raton Forbes, V E & Forbes T L (1994) Ecotoxicology in Theory and Practice. Chapman & Hall Ecotoxicology Series 2: London. Walker C H, Hopkin S P, Sibly R M & Peakall, D B (1996) Principles of Ecotoxicology. Taylor & Francis: London There are three main objectives in ecotoxicology (Forbes & Forbes 1994): • obtaining data for risk assessment and environmental management. meeting the legal requirements for the development and release of new chemicals into the environment. • developing empirical or theoretical principles to improve knowledge of the behaviour and effects of chemicals in living systems. (More information about the highlighted terms used below can be found in the Definitions section.) In order to achieve these objectives, the main areas of study are: The distribution of POLLUTANTS in the environment, their entry, movement, storage and transformation within the environment.

The effects of pollutants on living organisms. At an individual level, TOXICANTS may disrupt the biochemical, molecular and physiological structure and function which will in turn have consequences for the structure and function of communities and ecosystems. At the population level it may be possible to detect changes in the numbers of individuals, in gene frequency (as in resistance of insects to insecticides) or changes in ecosystem function (e. g. soil nitrification) which are attributable topollution.

It may be possible to use BIOMARKERS to establish that a natural population has been exposed to pollution and these can provide a valuable guide to whether or not a natural population is at risk or in need of further

investigation. For the purposes of the Regulation and Registration of chemicals the toxicity of individual chemicals is principally investigated via TOXICITY TESTING, the main tool of which is the Standard Toxicity Test (STT) which usually tests the DOSE or CONCENTRATION of a particular chemical that is toxic to under controlled, laboratory conditions.

Toxicity tests are mainly carried out using individual animals although there has been a move towards the use of more complex systems known as MESOCOSMS. In some situations, particularly in the case of pesticides, it may be possible to carry out FIELD TRIALS to assess toxicity. Toxicity data are used to make assessments of the HAZARD and the RISK posed by a particular chemical. [pic] Significant Issues with Chemicals that have driven the development of Ecotoxicology [pic] 1. DDT – around the world 2. Cadmium in Japan 3. Mercury in Japan 4. PCBs in Japan and Taiwan 5.

Dioxins – various 6. The contamination of pristine environments (eg Arctic) by atmospheric transport of organohalogens Most workers in the field of ecotoxicology refer to the publication of Rachel Carson’s Silent Spring (1962) as a landmark in the public’s awareness of potential damage to human and environmentalhealthfrom man-made toxic substances. According to Rodricks (1992), Carson’s book “ almost single-handedly created modern society’s fears about synthetic chemicals in the environment and, among other things, fostered renewed interest in thescienceof toxicology”.

Certainly the consolidation ofacademicand related pursuits into the study of toxic substances in the environment dates from about the same time as the publication of Silent Spring. Prior to the 1960s, there were no coordinated programmes in research, ineducationor in regulation that systematically

addressed toxic substances in the environment. Considerable progress has been made in all these areas during the past four decades. Fate of chemicals in the environment and within organisms As ecotoxicologists we are concerned with the movement and fate of toxic chemicals at both the organism level and that of the whole ecosystem.

The relevant issues are: • the source, • transport, • modification and • final fate of the pollutants. At the organism level we need to be concerned with • Uptake • Excretion • Sites of action, metabolism or storage Toxicity testing and the regulation and release of toxic chemicals As ecotoxicology largely arose from toxicology and the need to regulate the introduction of potentially toxic chemicals into the environment, toxicity testing remains central to the subject today. Most toxicity testing for pollutants is still based on tests on individual organisms in artificial test situations (see list of examples in next section).

These tests are cheap, reliable and easy to perform but there is much debate about the relevance of many standard toxicity tests to 'real life'. Initially in the early days of environmental toxicology the concept of the 'most sensitive species' was used to relate the results of toxicity tests to the 'real world'. Certain species in a particular community were assessed as being 'most sensitive' to pollutants. The logic was that if a pollutant was non-toxic to the 'most sensitive' species then it would be safe for the rest of the community.

Essentially, this logic remains today - the results of tests on single species, in artificial situations are extrapolated to predict the effects of pollutants on whole communities or ecosystems. It is assumed that if you have enough information about the effects of a pollutant on the parts of an ecosystem,

then you can assemble the effects on the whole. There is however, some question about the usefulness of extrapolating from simple, highly artificial, single-species toxicity tests to complex, multi-variate ecosystems.

Forbes & Forbes (1994) argue that " understanding and predicting the consequences of pollutant-induced effects on ecosystems requires that the effects be examined at the level of interest" i. e. the population, community or ecosystem. This debate has been the source of much division in ecotoxicology, between the Applied, often Industrial, Ecotoxicologists concerned with the practicalities of chemical registration and testing and the Pure or Academic Ecotoxicologists who regard many toxicity testing regimes as inappropriate or at worst useless.

Unfortunately, never the twain shall meet and the level ofcommunicationbetween the two camps has not been great. A fictional exchange makes the point well (from Forbes & Forbes 1994): " Academic Ecotoxicologist: Single species acute toxicity tests are too simplistic and have no connection with what is really going on out in nature. These standard tests are not only irrelevant and a waste of time, they may in fact do more harm than good if they lead us to believe that we can use them to adequately protect the environment when in fact we cannot.

Industrial Ecotoxicologist: These tests may be oversimplified, but they are also cost-efficient, easy to perform, the procedures have been worked out, and the fact is they are required by government. We have absolutely no incentive to do more than is required by law, and, frankly, you have given us little hard evidence that current test procedures do fail to protect the

environment adequately. Government Ecotoxicologist: Do you have any idea of the number of new chemicals that we have to assess each year?

We can't tell industry to stop producing new chemicals and we can't wait until we understand the whole system before we try to protect it. If you think current procedures fail, then come up with some better tests - which must of course be simple, cheap and fast. Academic Ecotoxicologist: (Pause) ... Well, it's very complex, and of course I'll need much more data before I can give you an answer. But those single-species acute tests are oversimplified and have no connection with what is really going on out in the field ... Government Ecotoxicologist: We need tests! Give us tests! "

The way forward for Ecotoxicology must be to integrate its two halves much more fully. Toxicity testing, using single species, do provide useful information and will almost certainly remain central to the regulation and registration of toxic chemicals but much can be done to expand the scope of toxicity testing, to add tests that apply to higher levels of organisation and so increase their relevance to the communities and ecosystems that are being protected. Testing methodologies An extensive range of ecotoxicological and biodegradation tests are required for the chemical, agrochemical and pharmaceutical industries.

The tests often used include: • Bacterial toxicity tests • Algal Growth tests with a variety of species • Acute toxicity tests with Lemna minor • Acute and Reproduction tests in Daphnia magna • Acute toxicity tests with the marine copepod Acartia tonsa • Oyster embryo larval toxicity test • Acute toxicity test with the marine invertebrate Mysidopsis bahia • Earthworm toxicity tests • Toxicity Tests with sediment dwelling organisms such as Chironomus

or Lumbriculus • Acute toxicity tests with freshwater and marine fish • Bioaccumulation in fish • Fish growth tests Early Life Cycle tests with fish Algal tests Several freshwater species are routinely tested. The most commonly used are Scenedesmus subspicatus and Pseudokirchneriella subcapitata. Other species used include Navicula Pelliculosa. Skeletonema costatum is the marine species preferred by most regulatory bodies. Electronic particle counters and size distribution analysers are used to monitor the growth of algae in the studies. Lemna is a useful substitute for higher plants. Invertebrate Tests Acute and reproduction studies are routinely conducted with Daphnia magna.

Acute tests with other species are also available including the marine copepod Acartia tonsa, the freshwater sediment dwelling species Chironomus riparius or Lumbirculus variegatus and the amphipod Gammarus pulex. Fish Acute tests are conducted under static, semi-static or flow-through conditions. The choice of test regime is dependent upon the chemical properties. Tests using species commonly encountered wild in the UK are rare as most tests are conducted using species adapted for life in the laboratory including: The species used include: • Rainbow trout • Common carp Golden orfe • Bluegill sunfish • Fathead minnow • Japanese killifish • Zebra fish Studies can also be conducted using marine species such as Turbot and Sheepshead minnow. Definitions used in Ecotoxicology Some of the terms used in ecotoxicology, such as LD50, have simple, widely accepted definitions and hence can be defined here with some confidence. Others however vary quite widely in their interpretation from one text to another. I

have tried to indicate these below and can only suggest that the reader refer carefully to the introduction of the text they are using.

Where there is likely to be some contradiction I have listed the reference for the definitions given. [pic] ECOTOXICOLOGY • is concerned with the toxic effects of chemical and physical agents on living organisms, especially on populations and communities within defined ecosystems: it includes the transfer pathways of those agents and their interactions with the environment. Butler, 1978. • investigates the effects of substances on organisms. The hazard to animal and plant populations can be determined by using survey data (retrospective) or by performing specific tests (prospective).

Rudolph & Boje, 1986. • the science that seeks to predict the impacts of chemicals on ecosystems. Levin et al 1989. • the study of harmful effects of chemicals upon ecosystems. Walker et al 1996. [pic] POLLUTANT or CONTAMINANT, XENOBIOTIC or ENVIRONMENTAL CHEMICAL? Variations of use of these terms are commonplace. “ Environmental chemical” may be used to describe simply any chemical that occurs in the environment (Walker et al 1996) or substances which enter the environment as a result of human activity or occur in higher concentrations than they would in nature (Rombke & Moltmann 1995).

The terms contaminant and pollutant can be described separately but are often used as synonyms. Both words are used to describe chemicals that are found at levels judged to be above those that would normally be expected. “ Pollutants” carries the connotation of the potential to cause harm, whereas contaminants are not by definition harmful. This is however, not an easy

distinction to make. Whether or not a contaminant is a pollutant may depend on its level in the environment and the organism or system being considered, thus one particular substance may be a contaminant relative to one species but pollutant relative to another.

Finally, in practice it is often difficult to demonstrate that harm is not being caused so that in effect pollutant and contaminant become synonymous. (Walker et al 1996). Xenobiotic is used to describe compounds that are 'foreign' to a particular organism, that is they do not play a part in their normal biochemistry. A chemical that is normal to one organism may be foreign to another and so xenobiotics may be naturally occurring as well as man-made compounds (Walker et al 1996). The term Xenobiotic is sometimes also used in a more general sense to describe " foreign substances" in the environment (Rombke & Moltmann 1995). [pic]

HARM or DAMAGE? Biological systems are resilient to harm caused by adverse factors in the environment since they are able to adapt to some insults. There is a fundamental difference in viewpoint between these two words, one defines harm as an effect regardless of any biological compensation that the population might make, the other defines damage as occurring only if there is an effect subsequent to any compensation. harm: biochemical or physical changes which adversely affect individual organisms' birth, growth or mortality rates. Such changes would necessarily produce population declines were it not that other processes may compensate. Walker et al 1996). damage: " the interaction between a substance and a biological system. The substance's potential to cause damage is weighed against the protective potential inherent in the biological system (e. g.

excretion or metabolic reactions, adaptation or regeneration)" (Rombke & Moltmann 1995). [pic] ENDPOINTS, DOSE and CONCENTRATION There are many different ways in which toxicity can be measured but they are usually assessed relative to a particular outcome or END POINT. Initially, most Toxicity Tests measured the number of organisms killed by a particular DOSE or CONCENTRATION of the chemical being tested.

With terrestrial animals the DOSE of chemical (taken orally, applied to the skin or injected) administered is usually recorded. DOSE is usually used where the dietary dose of a test chemical can be accurately determined. For aquatic organisms or where the test chemical is dosed into the surrounding medium, the tests usually measure the CONCENTRATION of chemical in the surrounding water/medium. The following measures, known as a group as EDs or ECs (Effective Doses or Effective Concentrations) are frequently used to describe data from toxicity tests: LD50

Median lethal dose, that is the dose that kills 50% of the population LC50 Median lethal concentration. ED50/EC50 Median effect dose/concentration, that is the dose that produced a defined effect to 50% of the population. NOED/NOEC No Observed Effect Dose (or Concentration) NOEL No Observed Effect Level. Sometimes this more general term is used to describe either of the above. It can be defined as the highest level (that is dose or concentration) of the test chemical that does not cause a statistically significant difference from the control. LOED/LOE Lowest Observed Effect Dose (or Concentration)

There has been a move away from the use of lethal end points in toxicity testing towards the measurement of EFFECTS rather than death. Examples

of EFFECTS which can be used include changes in: reproduction (eg. number of eggs laid or young hatched); growth (e. g. biomass or body length) and biochemical or physiological effects (e. g. enzyme synthesis or respiration). [pic] HAZARD AND RISK Toxicity data is used to make assessments of the HAZARD and the RISK posed by a particular chemical. Where: HAZARD is the potential to cause harm RISK is the probability that harm will be caused.

Defining HAZARD involves answering two questions, 'how much damage are we prepared to tolerate' and 'how much proof is enough'. The first is a question for society, alleviating/avoiding/repairing damage involves costs, how much are we prepared to pay? The second is largely a scientific problem of providing sufficient evidence that damage is due to pollution. HAZARD is not necessarily directly related to toxicity, it is a product of exposure and toxicity, a compound with moderate toxicity but very high exposure may cause more damage that a very toxic chemical with very low exposure.

RISK is usually defined using the predicted environmental concentration (PEC) and the predicted environmental no effect concentration (PNEC). Information on the movement and behaviour of pollutants in the environment are used to calculate the PEC whereas data from Toxicity Testing must be extrapolated to calculate the PNEC. The making of these calculations is not a precise art, apart from doubts about the extrapolation of Toxicity data from the lab to the field it can be very difficult to estimate the degree of exposure, particularly for mobile taxa such as birds and mammals. [pic]

BIOMARKERS A Biomarker can be defined as a " biological response to a chemical or chemicals that gives a measure of exposure and sometimes,

also, of toxic effect" (Walker et al 1996), they can be divided into biomarkers of exposure and of toxic effect. Examples of biomarkers range from the inhibition of AChE (acetylcholinesterase) in the nervous system of animals to the thinning of eggshells in birds. Biomarkers can help to bridge the gap between the laboratory and the field by giving direct evidence of whether or not a particular animal, plant or ecosystem is being affected by pollution.

They will often provide more reliable evidence of exposure than measurements of the pollutants themselves in the environment, the latter are often short-lived and difficult to detect, whereas their effects (detectable via biomarkers) may be much longer-term. [pic] A QUESTION OF SCALE AND ACCURACY The difficulty in extrapolating from simple, highly artificial, single-species toxicity tests to complex, multi-variate ecosystems has led to attempts to develop more complex systems which can be used in toxicity tests.

Such systems are usually termed microcosms, mesocosms or macrocosms, that is small, medium or large multispecies systems. It must be possible to control conditions in these systems to such an extent that they can provide meaningful, reproducible (that is, the system could be accurately copied elsewhere), replicable (that is, two replicates of the same experiment would produce the same results) data in toxicity tests. Simply because they are more complex systems it is seldom possible to produce tests that are as precise and controlled as those carried out in single species STTs.

However, despite their limitations these larger-scale tests can provide important insights into the effect of pollutants on whole systems rather than on single species. [pic] MIXTURES OF CHEMICALS, ADDITION OR

MULTIPLICATION? In natural systems, organisms are often (usually) exposed to more than one pollutant at the same time. However, regulatory authorities usually assume - unless there is evidence to the contrary - that the toxicity of combinations of chemicals is roughly additive.

Fortunately in many cases this is quite correct but in some cases, toxicity is more than additive in that is there is POTENTIATION of toxicity. One particular type of potentiation called SYNERGISM occurs where the effect of two or more chemicals combine to have greater impact than expected from their individual concentrations. [pic] Ecotoxicology - Pesticide Definitions [pic] What is a pesticide? A literal definition of a pesticide would be " a killer of pests". In practice pesticides are no longer aimed exclusively at killing the pests they are used to control and the term has acquired a rather wider meaning uch as " the chemical tools used to manage all kinds of pests" or in the US it's more official definition is " any substance used for controlling, preventing destroying, repelling or mitigating any pest" (all definitions from Ware 1991). Hence pesticides include not only those chemicals which kill the pest they are used against but also those such as insect chemosterilants or plant and insect growth regulators which control pest populations without necessarily, physically, killing the pests they come into contact with.

Pesticides have been divided into many different classes. Firstly, according to the target organism that they control, so insect-icides kill (or control) insects, rodent-icides control rodents etc. The -icide suffix has been widely used in the past, as shown in Table 1, but relatively few of these terms are in common use today. Secondly, pesticides can be classified according to their

mode of action, that is the way in which they act on the pest population, e. g. attractants, repellents, chemosterilants etc.

Finally, the definition of a pesticide has been widened once again to: " pesticides are used by man as intentional additions to his environment in order to improve environmental quality for himself, his animals or his plants" (Ware 1991). This definition allows the inclusion of 2 new classes of treatment. Firstly those such as plant growth regulators, which are not only used as herbicides to control weeds, but also to control directly the growth of the crop and hence improve its success.

For instance, they are used to reduce the growth of cereals so that they do not become too tall and prone to 'lodging' before harvest. Secondly, microbial pesticides which are not based on a chemical but on bacteria, fungi, nematodes and viruses which attack the pest. [pic] Table 1 Classes of pesticide according to : A. the target organism and B. pesticide mode of action. After Ware (1991). | CLASS | FUNCTION | | A.

By Target Organism | | acaricide | kills/controls mites | | algicide | kills/controls algae | | avicide | kills/controls or repels birds | | bactericide | kills/controls bacteria | | fungicide | kills/controls fungi | | herbicide | kills/controls plants | | insecticide | kills/controls insects | | larvicide | kills/controls larvae (usually mosquitoes) | | miticide | kills/controls mites | | molluscicide | kills/controls snails & slugs.

May include oysters, clams, & mussels | | nematicide | kills/controls nematodes | | ovicide | kills/controls eggs | | pediculicide | kills/controls lice | | piscicide | kills/controls fish | | predicide | kills/controls predators (usually

such as coyotes) | | rodenticide | kills/controls rodents | | silvicide | kills/controls trees & brush | | slimicide | kills/controls slime | termiticide | kills/controls termites | | B. By Mode of Action - by affect on pest | | attractants/pheromones | Attract animals, especially insects usually into traps. Often sexual pheromones. | | chemosterilants | Sterilise insects or vertebrates (birds, rodents). Usually sterilise males. | | defoliants | Remove leaves. | | desiccants | Speed drying of plants. Used not only to kill weeds but also as pre-harvest desiccants to make harvesting | | | easier. | disinfectants | Kill or inactivate harmful micro-organisms | | feeding stimulants | Cause insects to feed more vigorously | | growth regulators | Stimulate or retard plant or insect growth. Natural or artificial hormones used not only to kill weed species | | | but also to protect crops such as cereals from lodging. | | repellents | Repel insects, mites, ticks or pest vertebrates (dogs, rabbits, deer, birds). | | B. By Mode of Action - by timing of application | | curative (fungicides) | applied to the plant after initial infection. | eradicant (fungicides) | applied when disease symptoms have already become visible, often to prevent the spread of disease. | | protectant (fungicides) | applied to the plant surface before infection. | | pre-plant or pre-sowing | applied before crop is sown or planted | |(herbicides) | | | pre-emergence (herbicides) | applied before the crop has germinated | | post-emergence(herbicides) | applied after the crop has germinated | | B.

By Mode of Action - by selectivity | | the degree to which a pesticide discriminates between target and non-target organisms. | | selective | A selective pesticide effects a very narrow range of species other than the target pest or may be. The chemical | | | itself may be selective in that it

does not affect non-target species or it may be used selectively in such a | | | way that non-target species do not come into contact with it. | | non-selective | a non-selective pesticide kills a very wide range of plants, insects, fungi etc. | | B.

By Mode of Action - by site of interaction with pest | | systemic | the pesticide is absorbed by the pest and moves around within the pest system to reach parts of the pest remote | | | from the point of application | | contact | contact pesticides directly affect the parts of the plant, insect, fungus etc to which they are applied. They | | | cause localised damage to the plant or animal tissue on contact. | References Barlow, F (1985) Chemistry and formulation. In: Pesticide Application: Principles and Practice. Ed: P T Haskell. Oxford Science Publications: Oxford. pp 1-34. Dent, D R (1995) Integrated Pest Management.

Chapman & Hall: London, Glasgow, Weinheim, New York, Todyo, Melbourne, Madras. Rombke, J & J M Moltmann (1995) Applied Ecotoxicology. Lewis Publishers: Boca Raton, New York, London, Tokyo. Ware, G W (1991) Fundamentals of Pesticides. A self-instruction guide. Thomson Publications: Fresno USA. [pic] Ecotoxicology - Pesticide Classification - Insecticides [pic] While pesticides can be divided into many classes by target organism, mode of action etc for most purposes chemical pesticides are divided into three major groups according to their target organism, that is: insecticides, herbicides and fungicides. These groups are then subdivided into chemical groups such as organophosphates, organochlorines, carbamates etc.

This simplified classification effectively groups acaricides, nematicides and molluscicides in with insecticides as many chemicals that have acaricidal,

nematocidal or molluscicidal properties are also insecticidal. The current proliferation of chemical insecticides dates from World War II, until this time the insecticides available were based on: arsenicals, petroleum oils, sulphur, hydrogen cyanide gas, cryolite and on extracts from plants such as pyrethrum, nicotine and rotenone. Table 2: Classification of Insecticides gives a summary of the main chemical classes of insecticide and the main chemicals in each class. The characteristics of the main classes of insecticide: the organochlorines, organophosphates, carbamates and pyrethroids are summarised below. Organochlorines Also called: chlorinated hydrocarbons

A large and varied group that has a particularly high public profile because of the environmental problems they have caused. They were mostly discovered in 1942-56 and were very important in the early success of synthetic insecticides. They are mostly Insecticides with a very wide range of actions, they can be divided into three main groups: DDT and related compounds including rhothane (DDD) and methoxychlor. Widely used during World War II for control of disease vectors (such as mosquitoes) and subsequently much used on agricultural pests such as ectoparasites of farm animals and insect disease vectors and also widely used against insects in domestic and industrial premises. chlorinated cyclodiene insecticides such as aldrin, dieldrin and heptachlor. ost widely used as seed dressings and soil insecticides. hexachlorocyclohexanes (HCHs), such as lindane used against pests and parasites of farm animals, also in insecticidal seed dressings. Organochlorine insecticides are very stable solids with: limited vapour pressure, very low water solubility and high lipophilicity. They may be very

persistent in their original form or as stable metabolites. They tend to be stored in body fats and are particularly hazardous because they are so stable and tend to accumulate in successive organisms in thefoodchain. DDT and the HCHs a regarded as only moderately toxic to mammals while the chlorinated cyclodienes are highly toxic.

Action: all organochlorine insecticides are nerve poisons but DDT has a different action to the chlorinated cyclodienes and HCHs. DDT acts on the sodium channels in the nervous system so that the passage of an 'action potential' along the nerve is disrupted. It causes uncontrolled repetitive spontaneous discharges along the nerve. Uncoordinated muscle tremors and twitches are characteristic symptoms. The chlorinated cyclodienes and HCHs act on the GABA receptors which function as a channel for Cl - ions through the nerve membranes. They bind to the GABA receptors and reduce the flow of Cl - ions. Typical symptoms include convulsions. Organophosphates Also called: organic esters of phosphorus acid.

Such as bromophos, chlorpyrifos, diazinon, dichlorvos, fenitrothion, malathion, parathion and phorate. The same basic constituents are combined with many additional chemicals to give a wide range of products with very different properties. Organophosphates were developed during the second world war and have two main uses: as insecticides and as nerve gases (chemical warfare agents). They are mostly liquids, liphophilic, with some volatility and a few are solids. Generally, they are less stable and more readily broken down than organochlorines and are relatively short-lived in the environment, hence most of their hazard is associated with short-term (acute) toxicity.

The water solubility of the various organophosphate compounds is very variable and they are prepared in numerous formulations: as emulsifiable concentrates for spraying and to control ectoparasites of farm animals (particularly sheep dips) and sometimes internal parasites (such as ox warble fly); as seed dressings and as granular formulations particularly used for the most toxic organophosphates (e. g. disyston and phorate) as the active ingredient is effectively 'locked up' in the granule and is safer to handle and only slowly released into the environment. Organophosphates are also used to control vertebrate pests such as Quelea in Africa. Action: like organochlorines, organophosphates also act as a neurotoxin. They combine with the enzyme acetylcholinesterase and prevent conduction of nerve impulses at junctions in the nervous system where acetylcholine is the natural transmitter.

As a result, acetylcholine builds up in the nerve synapse and eventually leads to synaptic block when the acetylcholine can no longer relay signals across the synapse. In neuro-muscle junctions this leads to tetanus, the muscle is in a fixed state, unable to contract or relax in response to nerve stimulation. Carbamates e. g. aldicarb, carbaryl, carbofuran, methiocarb, methomyl, pirimicarb and propoxur Carbamates are a more recent development than organochlorines or organophosphates, they are all derivatives of carbamic acid. The basic carbamate group is combined with different chemicals to produce insecticides with a wide range of properties (in particular they vary greatly in their water solubility) and actions.

Carbamates are not only used as insecticides but also molluscicides and nematicides. Carbamates are also used as herbicides and fungicides but

these have a different mode of action and are described elsewhere. Carbamates are mainly used to control insect pests in agriculture and horticulture, they have abroad spectrum of activity and usually act by contact or stomach action although a few possess systemic activity (aldicarb, carbofuran). Action: basically the same as organophosphates, inhibiting the action of acetylcholine at the nerve synapses. Doses of carbamates are not accumulative and carbamate poisoning is more easy to reverse than that caused by organophosphates.

They are generally regarded as representing a short-term hazard. Pyrethroids Such as cypermethrin, deltamethrin, permethrin, phenothrin, resmethrin. Pyrethrin insecticides were developed from naturally occurring chemicals found in the flower heads of Chrysanthenum sp. and these provided the model for the production of synthetic pyrethroid insecticides. Pyrethroids are generally more stables than natural pyrethrins. The development of pyrethroids can be traced over 4 main phases (Ware 1991). The first generation allethrin was a synthetic duplicate of a natural pyrethrum, cinerin I. The second generation included bioallethrin, phenothrin, resmethrin and bioresmethrin.

These were marginally more effective than natural pyrethrums but were neither effective enough nor photostable enough to be used extensively in agriculture. However, they are still used in pest control formulations for the home. The third generation of pyrethroids included fenvalerate and permethrin which were stable in sunlight and only slightly volatile and could be used successfully in agriculture. Finally, the fourth and current generation of pyrethroids can be used at much lower concentrations (one-fifth to one-

tenth) that those in generation 3 and are all photostable. Overall, most pyrethroids are not sufficiently soluble in water to be used a systemic insecticides. They are mainly formulated as emulsifiable concentrates for spraying.

They control a wide range of agricultural and horticultural insect pests and are used extensively to control insect vectors of disease (e. g. tsetse fly in Africa) Action: pyrethroids are generally solids with very low water solubility and they act as neruotoxins in a very similar way to DDT. They are readily biodegradable but can bind to particles in soils and sediments and can be persistent in these locations. They are particularly toxic to insects as opposed to mammals and birds and the main environmental concerns are over their effects on fish and non-target invertebrates. Table 2: Classification of Insecticides Data from: Whitehead, R (1995) The UK Pesticide Guide. CAB International & BCPC. Chemical group | Compound | Action | Notes | | AMIDINES | | | | | amitraz | | also ACARICIDE | | BOTANICAL | | | | | | azadirachtin | insect growth regulator | extracted from Neem | | | nicotine | contact, non-persistent general | extracted from tobacco | | | purpose, | | | | pyrethrin | contact, non-persistent | extracted from Pyrethrum | | | rotenone | contact | extracted from Derris and Lonchocarpus | | CARBAMATES | | | | | aldicarb | systemic | also NEMATICIDE | | | bendiocarb | contact & ingested | | | | | carbaryl | contact | also WORM KILLER, FRUIT THINNER | | | carbofuran | systemic | also NEMATICIDE | | | methiocarb | stomach acting | also MOLLUSCICIDE | | | methomyl | fly bait | | | | pirimicarb | contact & fumigant aphids only | | | | propoxur | fumigant, maimainly in | | | | | glasshouses | | | | thiocarb | pelleted bait | also MOLLUSCICIDE | | ORGANOCHLORINES | | | | |

diphenyl aliphatic derivatives | DDT | | | | rhothane (DDD) | | | | benzene derivatives | lindane ? amma HCH | contact, ingested & fumigant | | | cyclodiene derivatives | aldrin | persistent | UK revoked 1989 | | | dieldrin | persistent | UK revoked 1989 | | | endosulfan | contact & ingested | also ACARICIDE | | ORGANOPHOSPHATES | | | | aliphatic derivatives | dichlorvos | contact, fumigant | | | dimethoate | contact, systemic | also ACARICIDE | | | disulfoton | systemic, granules | | | | malathion | contact | also ACARICIDE | | | phorate | systemic | | | phenyl derivatives | fenitrothion | contact, broad spectrum | | | | parathion | | | | heterocyclic derivatives | chlorpyrifos | contact & ingested | also ACARICIDE | | | diazinon | contact | | | ORGANOTINS | | | | | fenbutatin-oxide | | ONLY ACARICIDE | | PYRETHROIDS | | | | | generation 1 | allethrin | | | | generation 2 | bioresmethrin | contact, residual | also ACARICIDE | | | phenothrin | contact & ingested | | | | resmethrin | contact | | | | tetramethrin | contact | | | generation 3 | fenvalerate | contact | | | | permethrin | contact & ingested, broad | | | | spectrum | | | generation 4 | bifenthrin | contact, residual | also ACARICIDE | | | cypermethrin | contact & ingested | | | | cyfluthrin | | | | | fenpropathrin | contact & ingested | also ACARICIDE | [pic] Ecotoxicology - Pesticide Classification - Herbicides [pic] It is really only in the last 50 years that use of chemical weedkillers or herbicides has become widespread. Prior to this, the control of weeds in crops was carried out largely by manual weeding, crop rotation, ploughing and various ways of stopping weed seeds being dispersed in crop seed. Today, the heavy use of herbicides is confined to those countries that practice highly intensive, mechanised farming.

In 1971 it was estimated that more energy was expended on weeding crops than on any other single human task (Brain 1971). Herbicides are also used extensively in non-crop and amenity situations such as industrial sites, roadsides, ditch banks, recreational areas etc. Herbicides can be classified in a number of different ways. The main classification used is often according to chemical class but they can also be classified according to their selectivity, the way that they affect the plant, the timing of application and the area covered by an application. Herbicides are classed as selective if they kill some plant species but not others, for instance they may kill the weeds but not the crop and as non-selective if they kill all vegetation.

Herbicides may be intrinsically selective in that they are active against some species of weed but not others but they may also be used selectively, that is in such a way that they only come into contact with the weeds and not the crop. There are two main ways in which herbicides affect the plants they are applied to: contact herbicides kill parts of the plant that they come into contact with. These are generally used against annual weeds and if they are to be effective need complete coverage of the target weed with the chemical. Systemic or translocated herbicides are absorbed either by the roots or foliage of the plant and then move within the plants system to areas remote from the site of application.

Translocated herbicides tend to be slower acting than contact ones and while they can be used against annual weeds they are more commonly aimed at perennial weeds. With translocated herbicides a uniform, although not necessarily complete, coverage of the target weeds is necessary. Finally, herbicides can be classified according to the timing of application in relation

to the crop they are being used in. Pre-plant, or pre-sowing herbicides must be applied to an area before the crop is planted. Pre-emergence herbicides are applied before the crop has emerged, this may allow an added level of selectivity as a herbicide can be applied to growing weeds while the crop itself is still protected by the oil. Finally, post-emergence herbicides are applied after the crop has emerged from the soil.

Again, a level of selectivity may be introduced by applying a germination inhibitor to prevent further germination of weed seeds - after the crop itself has germinated. Phenoxy Herbicides e. g. 2, 4-D, MCPA, 2, 4, 5-T All derivatives of phenoxyalkane carboxylic acids that act as plant growth regulator herbicides. Phenoxy herbicides were the first safe, selective herbicides discovered and they are still used in huge quantities. They act by simulating the action of natural hormones and produce uncoordinated plant growth. Their action is selective as they are toxic to dicotyledonous but not monocotyledonous plants. Hence they can be used to control 'dicot' weeds (broad leaved weeds) in 'monocot' crops (e. g. cereals, grass). Their physical properties vary greatly according to formulation.

For instance, as alkali salts they are highly water soluble (can be formulated as aqueous solutions) whereas when as simple esters they have low water solubility and are lipophilic (generally formulated as emulsifiable concentrates). The main hazard they present is mainly posed by unwanted spray drift but they have also sometimes been contaminated with the highly toxic compound TCDD (or dioxin). Other related compounds, also with plant growth regulating properties include phenoxypropionic acids (e. g. CMPP) and phenoxybutyric acids (e. g. 2, 4DB). Table 3: Classification of Herbicides

Data from: Whitehead, R (1995) The UK Pesticide Guide. CAB International & BCPC. Chemical group | Compound | Uptake/action | Persistence | Timing/site of | Other uses | | | | | application | | | ACETANILIDES | | | | | | | alachlor | via roots, | residual | pre/post-emergence | | | | translocated | | | | AMIDES or substituted amides | | | | | | | napropamide | | | pre-emergence | | | propachlor | | | pre-emergence | | BENZOICS or arylaliphatic acids | | | | | | | dicamba | translocated | | soil/foliar | | | BENZONITRILES or substituted nitriles| | | | | | | dichlobenil | | residual | soil | | | DIAZINONES | | | | | | | bentazone | contact | | post-emergence | | | BIPYRIDYLIUMS | | | | | | | diquat | contact | non-residual | foliar | pre-harvest, CROP | | | | | | DESICCANT | | | paraquat | contact | non-residual | | | | CARBAMATES or carbanilates | | | | | | | asulam | translocated | | foliar | | | | chlorpropham | | residual | soiltubers | POTATO SPROUT SUPPRESSANT| | | phenmedipham | contact | | foliar | | | CHLOROALKANOIC ACIDS or chlorinated | | | | | | | aliphatic acids | | | | | | | dalapon | | persistent | soil? | | DINITROANILINES or nitroanilines | | | | | | | pendimethalin | | residual | pre-emergence, soil| | | | | trifluralin | | | soil-incorporated | | | HBNs | | | | | | | bromoxynil | contact | | post-emergence | | | | ioxynil | contact | | post-emergence | | | IMIDAZOLINONES or imidazoles | | | | | | | imazapyr | translocated | residual | foliar, soil | | | | imazaquin | | | | | | OXIMES or cyclohexenones | | | | | | | cycloxydim | translocated | | post-emergence | | | | sethoxydim | | | post-emergence | | | PHENOXYACETIC ACIDS | | | | | | | MCPA | translocated | | post-emergence | | | PHENOXYBUTYRIC ACIDS | | | | | | | MCPB | translocated | | post-emergence | | | PHENOXYPROPRIONIC ACIDS | | | | | | | diclofop-methyl | translocated | | post-emergence | | | | fenoxaprop-P-ethyl

| | | post-emergence | | | fluazifop-P-butyl | | | post-emergence | | | | mecoprop | translocated | | | | | mecoprop-P | translocated | | post-emergence | | | PHOSPONIC ACIDS or phosphona amino | | | | | | acids or phosphates | | | | | | | glufosinate-ammonium | contact | non-residual | foliar | | | | glyphosate | translocated | non-residual | foliar | | | PICOLINIC ACIDS | | | | | | | picloram | translocated | persistent | foliar, soil | | | PYRIDINOXY ACIDS | | | | | | | fluroxypur | | | post-emergence | | | triclopyr | | | foliar | | | QUATERNARY AMMONIUM | | | | | | | difenzoquat | | | post-emergence | | | SULFONYLUREAS | | | | | | | metsulfuron-methyl | contact | residual | post-emergence | | | | triasulfuron | | | post-emergence | | | THIOCARBAMATES | | | | | | | tri-allate | | | soil-acting, | | | | | | pre-emergence | | | TRIAZINES | | | | | | | atrazine | | residual | pre/post emergence | | | | cyanazine | contact | residual | pre-emergence | | | | metribuzin | contact | residual | pre-/post-emergence| | | | simazine | root uptake | | | soil