Dichloro Diphenyl Trichloroethane (detailed)

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DDT ("dichlorodiphenyltrichloroethane") is a colorless, crystalline, tasteless and almost odorless organochloride known for its insecticidal properties. DDT has been formulated in almost every conceivable form, including solutions in xylene or petroleum distillates, emulsifiable concentrates, water-wettable powders, granules, aerosols, smoke candles and charges for vaporizers and lotions.[2]

First synthesized in 1874, DDT's insecticidal action was discovered by the Swiss chemist Paul Hermann Müller in 1939. It was then used in the second half of World War II to control malaria and typhus among civilians and troops. After the war, DDT was made available for use as an agricultural insecticide and its production and use duly increased.[3] Müller was awarded the Nobel Prize in Physiology or Medicine "for his discovery of the high efficiency of DDT as a contact poison against several arthropods" in 1948.[4]

In 1962, the book Silent Spring by American biologist Rachel Carson was published. It catalogued the environmental impacts of indiscriminate DDT spraying in the United States and questioned the logic of releasing large amounts of chemicals into the environment without a sufficient understanding of their effects on ecology or human health. The book claimed that DDT and other pesticides had been shown to cause cancer and that their agricultural use was a threat to wildlife, particularly birds. Its publication was a seminal event for the environmental movement and resulted in a large public outcry that eventually led, in 1972, to a ban on the agricultural use of DDT in the United States.[5] A worldwide ban on its agricultural use was later formalised under the Stockholm Convention, but its limited use in disease vector control continues to this day and remains controversial,[6][7] because of its effectiveness in reducing deaths due to malaria, countered by environmental and health concerns.

Along with the passage of the Endangered Species Act, the US ban on DDT is cited by scientists as a major factor in the comeback of the bald eagle (the national bird of the United States) and the peregrine falcon from near-extinction in the contiguous United States.[8][9]

DDT is similar in structure to the insecticide methoxychlor and the acaricide dicofol. Being highly hydrophobic, it is nearly insoluble in water but has good solubility in most organic solvents, fats and oils. DDT does not occur naturally, but is produced by the reaction of chloral (CCl

3CHO) with chlorobenzene (C

6H

5Cl) in the presence of sulfuric acid as a catalyst. Trade names that DDT has been marketed under include Anofex (Geigy Chemical Corp.), Cezarex, Chlorophenothane, Clofenotane, Dicophane, Dinocide, Gesarol (Syngenta Corp.), Guesapon, Guesarol, Gyron (Ciba-Geigy Corp.), Ixodex, Neocid (Reckitt & Colman Ltd), Neocidol (Ciba-Geigy Corp.) and Zerdane.[3]

Commercial DDT is a mixture of several closely–related compounds. The major component (77%) is the p,p' isomer which is pictured at the top of this article. The o,p' isomer (pictured to the right) is also present in significant amounts (15%). Dichlorodiphenyldichloroethylene (DDE) and dichlorodiphenyldichloroethane (DDD) make up the balance. DDE and DDD are also the major metabolites and breakdown products in the environment.[3] The term "total DDT" is often used to refer to the sum of all DDT related compounds (p,p'-DDT, o,p'-DDT, DDE, and DDD) in a sample.

From 1950 to 1980, DDT was extensively used in agriculture – more than 40,000 tonnes were used each year worldwide[10] – and it has been estimated that a total of 1.8 million tonnes have been produced globally since the 1940s.[1] In the U.S., where it was manufactured by some 15 companies including Monsanto,[11] Ciba,[12] Montrose Chemical Company, Pennwalt[13] and Velsicol Chemical Corporation,[14] production peaked in 1963 at 82,000 tonnes per year.[3] More than 600,000 tonnes (1.35 billion lbs) were applied in the U.S. before the 1972 ban. Usage peaked in 1959 at about 36,000 tonnes.[15]

In 2009, 3314 tonnes were produced for the control of malaria and visceral leishmaniasis. India is the only country still manufacturing DDT, with China having ceased production in 2007.[16] India is the largest consumer.[17]

In insects it opens sodium ion channels in neurons, causing them to fire spontaneously, which leads to spasms and eventual death. Insects with certain mutations in their sodium channel gene are resistant to DDT and other similar insecticides. DDT resistance is also conferred by up-regulation of genes expressing cytochrome P450 in some insect species, [18] as greater quantities of some enzymes of this group accelerate metabolism of the toxin into inactive metabolites.

DDT was first synthesized in 1874 by Othmar Zeidler under the supervision of Adolf von Baeyer.[3][19] It was further described in 1929 in a dissertation by W. Bausch and in two subsequent publications in 1930.[20][21] The insecticide properties of "multiple chlorinated aliphatic or fat-aromatic alcohols with at least one trichloromethane group" were described in a patent in 1934 by Wolfgang von Leuthold.[22] DDT's insecticidal properties were not, however, discovered until 1939 by the Swiss scientist Paul Hermann Müller, who was awarded the 1948 Nobel Prize in Physiology and Medicine for his efforts.[4]

DDT is the best-known of several chlorine-containing pesticides used in the 1940s and 1950s. With pyrethrum in short supply, DDT was used extensively during World War II by the Allies to control the insect vectors of typhus – nearly eliminating the disease in many parts of Europe. In the South Pacific, it was sprayed aerially for malaria and dengue fever control with spectacular effects. While DDT's chemical and insecticidal properties were important factors in these victories, advances in application equipment coupled with a high degree of organization and sufficient manpower were also crucial to the success of these programs.[23] In 1945, it was made available to farmers as an agricultural insecticide,[3] and it played a role in the final elimination of malaria in Europe and North America.[24][25][6]

In 1955, the World Health Organization commenced a program to eradicate malaria in countries with low to moderate transmission rates worldwide, relying largely on DDT for mosquito control and rapid diagnosis and treatment to reduce transmission.[26] The program was initially highly successful, eliminating the disease in "Taiwan, much of the Caribbean, the Balkans, parts of northern Africa, the northern region of Australia, and a large swath of the South Pacific"[27] and dramatically reducing mortality in Sri Lanka and India.[28] However failure to sustain the program, increasing DDT mosquito DDT resistance, and increasing parasite resistance led to a resurgence. In many areas, early victories partially or completely reversed and, in some cases, rates of transmission even increased.

[29] The program was successful in eliminating malaria only in areas with "high socio-economic status, well-organized healthcare systems, and relatively less intensive or seasonal malaria transmission".[30]

DDT was less effective in tropical regions due to the continuous life cycle of mosquitoes and poor infrastructure. It was not applied at all in sub-Saharan Africa due to these perceived difficulties. Mortality rates in that area never declined to the same dramatic extent, and now constitute the bulk of malarial deaths worldwide, especially following the disease's resurgence as a result of resistance to drug treatments and the spread of the deadly malarial variant caused by Plasmodium falciparum[citation needed] . The goal of eradication was abandoned in 1969, and attention was focused on controlling and treating the disease. Spraying programs (especially using DDT) were curtailed due to concerns over safety and environmental effects, as well as problems in administrative, managerial and financial implementation, but mostly because mosquitoes were developing resistance to DDT.[29] Efforts shifted from spraying to the use of bednets impregnated with insecticides and other interventions.[30][31]

As early as the 1940s, scientists in the U.S. had begun expressing concern over possible hazards associated with DDT, and in the 1950s the government began tightening some of the regulations governing its use.[15] However, these early events received little attention, and it was not until 1957, when the New York Times reported an unsuccessful struggle to restrict DDT use in Nassau County, New York, that the issue came to the attention of the popular naturalist-author, Rachel Carson. William Shawn, editor of The New Yorker, urged her to write a piece on the subject, which developed into her famous book Silent Spring, published in 1962. The book argued that pesticides, including DDT, were poisoning both wildlife and the environment and were also endangering human health.[5]

Silent Spring was a best seller, and public reaction to it launched the modern environmental movement in the United States. The year after it appeared, President Kennedy ordered his Science Advisory Committee to investigate Carson's claims. The report the committee issued "add[ed] up to a fairly thorough-going vindication of Rachel Carson’s Silent Spring thesis," in the words of the journal Science,[32] and recommended a phaseout of "persistent toxic pesticides".[33] DDT became a prime target of the growing anti-chemical and anti-pesticide movements, and in 1967 a group of scientists and lawyers founded the Environmental Defense Fund (EDF) with the specific goal of winning a ban on DDT. Victor Yannacone, Charles Wurster, Art Cooley and others associated with inception of EDF had all witnessed bird kills or declines in bird populations and suspected that DDT was the cause. In their campaign against the chemical, EDF petitioned the government for a ban and filed a series of lawsuits.[34] Around this time, toxicologist David Peakall was measuring DDE levels in the eggs of peregrine falcons and California condors and finding that increased levels corresponded with thinner shells.

In response to an EDF suit, the U.S. District Court of Appeals in 1971 ordered the EPA to begin the de-registration procedure for DDT. After an initial six-month review process, William Ruckelshaus, the Agency's first Administrator rejected an immediate suspension of DDT's registration, citing studies from the EPA's internal staff stating that DDT was not an imminent danger to human health and wildlife.[15] However, the findings of these staff members were criticized, as they were performed mostly by economic entomologists inherited from the United States Department of Agriculture, who many environmentalists felt were biased towards agribusiness and tended to minimize concerns about human health and wildlife. The decision not to ban thus created public controversy.[23]

The EPA then held seven months of hearings in 1971–1972, with scientists giving evidence both for and against the use of DDT. The hearings produced a 113-page decision, in which Hearing Examiner Edmund Sweeney wrote: “DDT is not a carcinogenic, mutagenic, or

teratogenic hazard to man. The uses under regulations involved here do not have a deleterious effect on fresh water fish, estuarine organisms, wild birds, or other wildlife. The evidence in this proceeding supports the conclusion that there is a present need for essential uses of DDT." [35]

In the summer of 1972, Ruckelshaus announced the cancellation of most uses of DDT – an exemption allowed for public health uses under some conditions.[15] He later wrote, in a letter to American Farm Bureau President Allan Grant, that "in such decisions the ultimate judgement remains political."[36] Immediately after the cancellation was announced, both EDF and the DDT manufacturers filed suit against the EPA, with the industry seeking to overturn the ban, and EDF seeking a comprehensive ban. The cases were consolidated, and in 1973 the U.S. Court of Appeals for the District of Columbia ruled that the EPA had acted properly in banning DDT.[15]

Some uses of DDT continued under the public health exemption. For example, in June 1979, the California Department of Health Services was permitted to use DDT to suppress flea vectors of bubonic plague.[37] DDT also continued to be produced in the US for foreign markets until as late as 1985, when over 300 tons were exported.[1]

In the 1970s and 1980s, agricultural use was banned in most developed countries, beginning with Hungary in 1968[38] then in Norway and Sweden in 1970, Germany and the United States in 1972, but not in the United Kingdom until 1984. By 1991 total bans on the use of DDT, including in disease control, were in place in at least 26 countries; for example Cuba in 1970, Singapore in 1984, Chile in 1985 and the Republic of Korea in 1986.[39]

The Stockholm Convention, which took effect in 2004, outlawed several persistent organic pollutants, and restricted DDT use to vector control. The Convention has been ratified by more than 170 countries and is endorsed by most environmental groups. Recognizing that total elimination in many malaria-prone countries is currently unfeasible because there are few affordable or effective alternatives, the convention exempts public health use within World Health Organization (WHO) guidelines from the ban.[40] Resolution 60.18 of the World Health Assembly commits the World Health Organization to the Stockholm Convention's aim of reducing and ultimately eliminating the use of DDT.[41] Malaria Foundation International states, "The outcome of the treaty is arguably better than the status quo going into the negotiations. For the first time, there is now an insecticide which is restricted to vector control only, meaning that the selection of resistant mosquitoes will be slower than before."[42]

Despite the worldwide ban, agricultural use continued in India,[43] North Korea, and possibly elsewhere as of 2008.[17]

Today, about 3,000 to 4,000 tonnes of DDT are produced each year for disease vector control.[16] DDT is applied to the inside walls of homes to kill or repel mosquitoes. This intervention, called indoor residual spraying (IRS), greatly reduces environmental damage. It also reduces the incidence of DDT resistance.[44] For comparison, treating 40 hectares (99 acres) of cotton during a typical U.S. growing season requires the same amount of chemical as roughly 1,700 homes.[45]

DDT is a persistent organic pollutant that is readily adsorbed to soils and sediments, which can act both as sinks and as long-term sources of exposure contributing to terrestrial organisms.[2] Depending on conditions, its soil half life can range from 22 days to 30 years. Routes of loss and degradation include runoff, volatilization, photolysis and aerobic and anaerobic biodegradation. Due to hydrophobic properties, in aquatic ecosystems DDT and its metabolites are absorbed by aquatic organisms and adsorbed on suspended particles, leaving little DDT dissolved in the water itself. Its breakdown products and metabolites,

DDE and DDD, are also highly persistent and have similar chemical and physical properties.[1] DDT and its breakdown products are transported from warmer regions of the world to the Arctic by the phenomenon of global distillation, where they then accumulate in the region's food web.[46]

Because of its lipophilic properties, DDT has a high potential to bioaccumulate, especially in predatory birds.[47] DDT, DDE, and DDD magnify through the food chain, with apex predators such as raptor birds concentrating more chemicals than other animals in the same environment. They are very lipophilic and are stored mainly in body fat. DDT and DDE are very resistant to metabolism; in humans, their half-lives are 6 and up to 10 years, respectively. In the United States, these chemicals were detected in almost all human blood samples tested by the Centers for Disease Control in 2005, though their levels have sharply declined since most uses were banned in the US.[48] Estimated dietary intake has also declined,[48] although FDA food tests commonly detect it.[49]

Marine macroalgae (seaweed) help reduce soil toxicity by up to 80% within six weeks.[50]

DDT is toxic to a wide range of living organisms, including marine animals such as crayfish, daphnids, sea shrimp and many species of fish. DDT, through its metabolite DDE (dichlorodiphenyldichloroethylene), caused eggshell thinning and resulted in severe population declines in multiple North American and European bird of prey species.[51] Eggshell thinning lowers the reproductive rate of certain bird species by causing egg breakage and embryo deaths. DDE related eggshell thinning is considered a major reason for the decline of the bald eagle,[8] brown pelican,[52] peregrine falcon, and osprey.[1] However, different groups of birds vary greatly in their sensitivity to these chemicals.[2] Birds of prey, waterfowl, and song birds are more susceptible to eggshell thinning than chickens and related species, and DDE appears to be more potent than DDT.[1] Even in 2010, more than forty years after the U.S. ban, California condors which feed on sea lions at Big Sur which in turn feed in the Palos Verdes Shelf area of the Montrose Chemical Superfund site seemed to be having continued thin-shell problems. Scientists with the Ventana Wildlife Society and others are intensifying studies and remediations of the condors' problems.[53]

The biological thinning mechanism is not entirely known, but there is strong evidence that p,p'-DDE inhibits calcium ATPase in the membrane of the shell gland and reduces the transport of calcium carbonate from blood into the eggshell gland. This results in a dose-dependent thickness reduction.[1][54][55][56] There is also evidence that o,p'-DDT disrupts female reproductive tract development, impairing eggshell quality later.[57] Multiple mechanisms may be at work, or different mechanisms may operate in different species.[1] Some studies show that although DDE levels have fallen dramatically, eggshell thickness remains 10–12 percent thinner than before DDT was first used.[58]

DDT is an endocrine disruptor[citation needed]. It is considered likely to be a human carcinogen although the majority of studies suggest it is not directly genotoxic.[59][60][61] The DDT metabolite DDE acts as an antiandrogen, but not as an estrogen. p,p'-DDT, DDT's main component, has little or no androgenic or estrogenic activity.[62] The minor component o,p'-DDT has weak estrogenic activity.

DDT is classified as "moderately toxic" by the United States National Toxicology Program (NTP)[63] and "moderately hazardous" by the World Health Organization (WHO), based on the rat oral LD of 113 mg/kg.[64] DDT has on rare occasions been administered orally as a treatment for barbiturate poisoning.[65]

DDT and DDE have been linked to diabetes. A number of studies from the US, Canada, and Sweden have found that the prevalence of the disease in a population increases with serum

DDT or DDE levels.[medical citation needed]

DDT and DDE, like other organochlorines, have been shown to have xenoestrogenic activity, meaning they are chemically similar enough to estrogens to trigger hormonal responses in animals. This endocrine disrupting activity has been observed in mice and rat toxicological studies, and available epidemiological evidence indicates that these effects may be occurring in humans as a result of DDT exposure. The US Environmental Protection Agency states that DDT exposure damages the reproductive system and reduces reproductive success. These effects may cause developmental and reproductive toxicity:

Occupational exposure in agriculture and malaria control has been linked to neurological problems (for example, Parkinson's disease)[medical citation needed] and asthma.[67][dead link] A 2014 study in JAMA Neurology reported that DDT levels were elevated 3.8 fold in Alzheimer's disease patients compared with healthy controls.[medical citation needed]

In 2002, the Centers for Disease Control reported that "Overall, in spite of some positive associations for some cancers within certain subgroups of people, there is no clear evidence that exposure to DDT/DDE causes cancer in humans."[1] The NTP classifies it as "reasonably anticipated to be a carcinogen," the International Agency for Research on Cancer classifies it as a "possible" human carcinogen, and the EPA classifies DDT, DDE, and DDD as class B2 "probable" carcinogens. These evaluations are based mainly on the results of animal studies.[1][31]

A Lancet review of epidemiological studies concluded that that DDT causes cancers of the liver, and pancreas, that there is mixed evidence that it causes cancers of the testes, and that it probably does not contribute to cancers of the rectum, prostate, endometrium, lung, or stomach.[31] A second review, whose co-authors included persons engaged in DDT-related litigation, reached broadly similar conclusions, but also found possible associations with breast cancer, leukemia, lymphoma, and testicular cancer.[48]

The question of whether DDT or DDE are risk factors in breast cancer has not been conclusively answered. Several meta analyses of observational studies have concluded that there is no overall relationship between DDT exposure and breast cancer risk.[68][69] The United States Institute of Medicine reviewed data on the association of breast cancer with DDT exposure in 2012 and concluded that a causative relationship could neither be proven nor disproven.[70]

A 2007 case control study using archived blood samples found that breast cancer risk was increased 5-fold among women who were born prior to 1931 and who had high serum DDT levels in 1963. Reasoning that DDT use became widespread in 1945 and peaked around 1950, they concluded that the ages of 14-20 were a critical period in which DDT exposure leads to increased risk. This study, which suggests a connection between DDT exposure and breast cancer that would not be picked up by most studies, has received variable commentary in third party reviews. One review suggested that "previous studies that measured exposure in older women may have missed the critical period."[48][71] A second review suggested a cautious approach to the interpretation of these results given methodological weaknesses in the study design.[72] The National Toxicology Program notes that while the majority of studies have not found a relationship between DDT exposure and breast cancer that positive associations have been seen in a "few studies among women with higher levels of exposure and among certain subgroups of women"[73]

Malaria remains a major public health challenge in many countries. 2008 WHO estimates were 243 million cases, and 863,000 deaths. About 89% of these deaths occur in Africa, and mostly to children under the age of 5.[74] DDT is one of many tools that public health officials use to fight the disease. Its use in this context has been called everything from a

"miracle weapon [that is] like Kryptonite to the mosquitoes,"[75] to "toxic colonialism."[76]

Before DDT, eliminating mosquito breeding grounds by drainage or poisoning with Paris green or pyrethrum was sometimes successful in fighting malaria. In parts of the world with rising living standards, the elimination of malaria was often a collateral benefit of the introduction of window screens and improved sanitation.[27] Today, a variety of usually simultaneous interventions is the norm. These include antimalarial drugs to prevent or treat infection; improvements in public health infrastructure to quickly diagnose, sequester, and treat infected individuals; bednets and other methods intended to keep mosquitoes from biting humans; and vector control strategies[74] such as larvaciding with insecticides, ecological controls such as draining mosquito breeding grounds or introducing fish to eat larvae, and indoor residual spraying (IRS) with insecticides, possibly including DDT. IRS involves the treatment of all interior walls and ceilings with insecticides, and is particularly effective against mosquitoes, since many species rest on an indoor wall before or after feeding. DDT is one of 12 WHO–approved IRS insecticides.

WHO's anti-malaria campaign of the 1950s and 1960s relied heavily on DDT and the results were promising, though temporary. Experts tie the resurgence of malaria to multiple factors, including poor leadership, management and funding of malaria control programs; poverty; civil unrest; and increased irrigation. The evolution of resistance to first-generation drugs (e.g. chloroquine) and to insecticides exacerbated the situation.[17][77] Resistance was largely fueled by often unrestricted agricultural use. Resistance and the harm both to humans and the environment led many governments to restrict or curtail the use of DDT in vector control as well as agriculture.[29] In 2006 the WHO reversed a longstanding policy against DDT by recommending that it be used as an indoor pesticide in regions where malaria is a major problem.[78]

Once the mainstay of anti-malaria campaigns, as of 2008 only 12 countries used DDT, including India and some southern African states,[74] though the number is expected to rise.[17]

When it was first introduced in World War II, DDT was very effective in reducing malaria morbidity and mortality.[23] The WHO's anti-malaria campaign, which consisted mostly of spraying DDT and rapid treatment and diagnosis to break the transmission cycle, was initially very successful as well. For example, in Sri Lanka, the program reduced cases from about one million per year before spraying to just 18 in 1963[79][80] and 29 in 1964. Thereafter the program was halted to save money and malaria rebounded to 600,000 cases in 1968 and the first quarter of 1969. The country resumed DDT vector control but the mosquitoes had evolved resistance in the interim, presumably because of continued agricultural use. The program switched to malathion, but despite initial successes, malaria continued its resurgence into the 1980s.[81][28]

Today, DDT remains on the WHO's list of insecticides recommended for IRS. Since the appointment of Arata Kochi as head of its anti-malaria division, WHO's policy has shifted from recommending IRS only in areas of seasonal or episodic transmission of malaria, to also advocating it in areas of continuous, intense transmission.[82] The WHO has reaffirmed its commitment to eventually phasing out DDT, aiming "to achieve a 30% cut in the application of DDT world-wide by 2014 and its total phase-out by the early 2020s if not sooner" while simultaneously combating malaria. The WHO plans to implement alternatives to DDT to achieve this goal.[83]

South Africa is one country that continues to use DDT under WHO guidelines. In 1996, the country switched to alternative insecticides and malaria incidence increased dramatically. Returning to DDT and introducing new drugs brought malaria back under control.[84] Malaria cases increased in South America after countries in that continent stopped using

DDT. Research data shows a significantly strong negative relationship between DDT residual house sprayings and malaria rates. In a research from 1993 to 1995, Ecuador increased its use of DDT and resulted in a 61% reduction in malaria rates, while each of the other countries that gradually decreased its DDT use had large increase in malaria rates.[45] [85][86]

In some areas resistance has greatly reduced DDT's effectiveness. WHO guidelines require that absence of resistance must be confirmed before using the chemical.[87] Resistance is largely due to agricultural use, in much greater quantities than required for disease prevention.

Resistance was noted early in spray campaigns. Paul Russell, a former head of the Allied Anti-Malaria campaign, observed in 1956 that "resistance has appeared after six or seven years."[27] Resistance has been detected in Sri Lanka, Pakistan, Turkey and Central America, and it has largely been replaced by organophosphate or carbamate insecticides, e.g. malathion or bendiocarb.[88]

In many parts of India, DDT has also largely lost its effectiveness.[89] Agricultural uses were banned in 1989 and its anti-malarial use has been declining. Urban use has halted completely.[90] Nevertheless, DDT is still manufactured and used,[91] and one study had concluded that "DDT is still a viable insecticide in indoor residual spraying owing to its effectivity in well supervised spray operation and high excito-repellency factor."[92]

Studies of malaria-vector mosquitoes in KwaZulu-Natal Province, South Africa found susceptibility to 4% DDT (the WHO susceptibility standard), in 63% of the samples, compared to the average of 86.5% in the same species caught in the open. The authors concluded that "Finding DDT resistance in the vector An. arabiensis, close to the area where we previously reported pyrethroid-resistance in the vector An. funestus Giles, indicates an urgent need to develop a strategy of insecticide resistance management for the malaria control programmes of southern Africa."[93]

DDT can still be effective against resistant mosquitoes,[94] and the avoidance of DDT-sprayed walls by mosquitoes is an additional benefit of the chemical.[92] For example, a 2007 study reported that resistant mosquitoes avoided treated huts. The researchers argued that DDT was the best pesticide for use in IRS (even though it did not afford the most protection from mosquitoes out of the three test chemicals) because the others pesticides worked primarily by killing or irritating mosquitoes – encouraging the development of resistance to these agents.[94] Others argue that the avoidance behavior slows the eradication of the disease.[95] Unlike other insecticides such as pyrethroids, DDT requires long exposure to accumulate a lethal dose; however its irritant property shortens contact periods. "For these reasons, when comparisons have been made, better malaria control has generally been achieved with pyrethroids than with DDT."[88] In India, with its outdoor sleeping habits and frequent night duties, "the excito-repellent effect of DDT, often reported useful in other countries, actually promotes outdoor transmission."[96] Genomic studies in the model genetic organism Drosophila melanogaster have revealed that high level DDT resistance is polygenic, involving multiple resistance mechanisms.[97]

IRS is effective if at least 80% of homes and barns in a residential area are sprayed.[87] Lower coverage rates can jeopardize program effectiveness. Many residents resist DDT spraying, objecting to the lingering smell, stains on walls, and the potential exacerbation of problems with other insect pests.[88][95][98] Pyrethroid insecticides (e.g. deltamethrin and lambda-cyhalothrin) can overcome some of these issues, increasing participation.[88]

Compared to contemporaries living where DDT is not used, South Africans living in sprayed homes have levels that are several orders of magnitude greater.[48] Breast milk from South

African mothers contains high levels of DDT and DDE.[48] There is some evidence that these levels are associated with neurological abnormalities in babies.[88]

Most studies of DDT's human health effects have been conducted in developed countries where DDT is not used and exposure is relatively low. Many experts urge that alternatives be used instead of IRS.[31][48] Epidemiologist Brenda Eskenazi argues, "We know DDT can save lives by repelling and killing disease-spreading mosquitoes. But evidence suggests that people living in areas where DDT is used are exposed to very high levels of the pesticide. The only published studies on health effects conducted in these populations have shown profound effects on male fertility. Clearly, more research is needed on the health of populations where indoor residual spraying is occurring, but in the meantime, DDT should really be the last resort against malaria rather than the first line of defense."[99]

Illegal diversion to agriculture is also a concern as it is almost impossible to prevent and its subsequent use on crops is uncontrolled. For example, DDT use is widespread in Indian agriculture,[100] particularly mango production,[101] and is reportedly used by librarians to protect books.[102] Other examples include Ethiopia, where DDT intended for malaria control is reportedly being used in coffee production,[103] and Ghana where it is used for fishing."[104][105] The residues in crops at levels unacceptable for export have been an important factor in recent bans in several tropical countries.[88] Adding to this problem is a lack of skilled personnel and supervision.[95]

Critics argue that limitations on DDT use for public heath purposes have caused unnecessary morbidity and mortality from vector borne diseases, with some claims of malaria deaths ranging as high as the hundreds of thousands,[106] and millions. Robert Gwadz of the US National Institutes of Health said in 2007, "The ban on DDT may have killed 20 million children."[107] In his novel State of Fear, author Michael Crichton wrote "Banning DDT killed more people than Hitler."[108] These arguments have been dismissed as "outrageous" by former WHO scientist Socrates Litsios. May Berenbaum, University of Illinois entomologist, says, "to blame environmentalists who oppose DDT for more deaths than Hitler is worse than irresponsible."[75] Investigative journalist Adam Sarvana and others characterize this notion as a "myth" promoted principally by Roger Bate of the pro-DDT advocacy group Africa Fighting Malaria (AFM).[109][110]

Criticisms of a DDT "ban" often specifically reference the 1972 US ban (with the erroneous implication that this constituted a worldwide ban and prohibited use of DDT in vector control). Reference is often made to Rachel Carson's Silent Spring, even though she never pushed for a ban on DDT specifically. John Quiggin and Tim Lambert wrote, "the most striking feature of the claim against Carson is the ease with which it can be refuted."[111]

It has also been alleged that donor governments and agencies have refused to fund DDT spraying, or made aid contingent upon not using DDT. According to a report in the British Medical Journal, use of DDT in Mozambique "was stopped several decades ago, because 80% of the country's health budget came from donor funds, and donors refused to allow the use of DDT."[112] Roger Bate asserts, "many countries have been coming under pressure from international health and environment agencies to give up DDT or face losing aid grants: Belize and Bolivia are on record admitting they gave in to pressure on this issue from [USAID]."[113]

The United States Agency for International Development (USAID) has been the focus of much criticism. While the agency is currently funding the use of DDT in some African countries,[114] in the past it did not. When John Stossel accused USAID of not funding DDT because it wasn't "politically correct," Anne Peterson, the agency's assistant administrator for global health, replied that "I believe that the strategies we are using are as effective as spraying with DDT ... So, politically correct or not, I am very confident that what

we are doing is the right strategy."[115] USAID's Kent R. Hill states that the agency has been misrepresented: "USAID strongly supports spraying as a preventative measure for malaria and will support the use of DDT when it is scientifically sound and warranted."[116] The Agency's website states that "USAID has never had a 'policy' as such either 'for' or 'against' DDT for IRS. The real change in the past two years [2006/07] has been a new interest and emphasis on the use of IRS in general – with DDT or any other insecticide – as an effective malaria prevention strategy in tropical Africa."[114] The website further explains that in many cases alternative malaria control measures were judged to be more cost-effective that DDT spraying, and so were funded instead.[117]

Organophosphate and carbamate insecticides, e.g. malathion and bendiocarb, respectively, are more expensive than DDT per kilogram and are applied at roughly the same dosage. Pyrethroids such as deltamethrin are also more expensive than DDT, but are applied more sparingly (0.02–0.3 g/m2 vs 1–2 g/m2), so the net cost per house is about the same over 6 months.[30]

Before DDT, malaria was successfully eliminated or curtailed in several tropical areas by removing or poisoning mosquito breeding grounds and larva habitats, for example by filling or applying oil to standing water. These methods have seen little application in Africa for more than half a century.[118] According to the United States CDC, such methods are not practical in Africa because "Anopheles gambiae, one of the primary vectors of malaria in Africa, breeds in numerous small pools of water that form due to rainfall.... It is difficult, if not impossible, to predict when and where the breeding sites will form, and to find and treat them before the adults emerge."[119]

The relative effectiveness of IRS (with DDT or alternative insecticides) versus other malaria control techniques (e.g. bednets or prompt access to anti-malarial drugs) varies greatly and is highly dependent on local conditions.[30]

A WHO study released in January 2008 found that mass distribution of insecticide-treated mosquito nets and artemisinin–based drugs cut malaria deaths in half in Rwanda and Ethiopia, countries with high malaria burdens. IRS with DDT did not play an important role in mortality reduction in these countries.[120][121]

Vietnam has enjoyed declining malaria cases and a 97% mortality reduction after switching in 1991 from a poorly funded DDT-based campaign to a program based on prompt treatment, bednets, and pyrethroid group insecticides.[122]

In Mexico, effective and affordable chemical and non-chemical strategies against malaria have been so successful that the Mexican DDT manufacturing plant ceased production due to lack of demand.[123]

A review of fourteen studies on the subject in sub-Saharan Africa, covering insecticide-treated nets, residual spraying, chemoprophylaxis for children, chemoprophylaxis or intermittent treatment for pregnant women, a hypothetical vaccine, and changing front–line drug treatment, found decision making limited by the gross lack of information on the costs and effects of many interventions, the very small number of cost-effectiveness analyses available, the lack of evidence on the costs and effects of packages of measures, and the problems in generalizing or comparing studies that relate to specific settings and use different methodologies and outcome measures. The two cost-effectiveness estimates of DDT residual spraying examined were not found to provide an accurate estimate of the cost-effectiveness of DDT spraying; furthermore, the resulting estimates may not be good predictors of cost-effectiveness in current programs.[124]

However, a study in Thailand found the cost per malaria case prevented of DDT spraying

($1.87 US) to be 21% greater than the cost per case prevented of lambda-cyhalothrin–treated nets ($1.54 US),[125] at very least casting some doubt on the unexamined assumption that DDT was the most cost-effective measure to use in all cases. The director of Mexico's malaria control program finds similar results, declaring that it is 25% cheaper for Mexico to spray a house with synthetic pyrethroids than with DDT.[123] However, another study in South Africa found generally lower costs for DDT spraying than for impregnated nets.[126]

A more comprehensive approach to measuring cost-effectiveness or efficacy of malarial control would not only measure the cost in dollars of the project, as well as the number of people saved, but would also consider ecological damage and negative aspects of insecticide use on human health. One preliminary study regarding the effect of DDT found that it is likely the detriment to human health approaches or exceeds the beneficial reductions in malarial cases, except perhaps in malarial epidemic situations. It is similar to the earlier mentioned study regarding estimated theoretical infant mortality caused by DDT and subject to the criticism also mentioned earlier.[127]

A study in the Solomon Islands found that "although impregnated bed nets cannot entirely replace DDT spraying without substantial increase in incidence, their use permits reduced DDT spraying."[128]

A comparison of four successful programs against malaria in Brazil, India, Eritrea, and Vietnam does not endorse any single strategy but instead states, "Common success factors included conducive country conditions, a targeted technical approach using a package of effective tools, data-driven decision-making, active leadership at all levels of government, involvement of communities, decentralized implementation and control of finances, skilled technical and managerial capacity at national and sub-national levels, hands-on technical and programmatic support from partner agencies, and sufficient and flexible financing."[129]

DDT resistant mosquitoes have generally proved susceptible to pyrethroids. Thus far, pyrethroid resistance in Anopheles has not been a major problem.[88]

What is dichloro diphenyl trichloroethane (DDT)? definition and meaning

http://www.businessdictionary.com/definition/dichloro-diphenyl-trichloroethane-DDT.html December 09, 2014

Definition of dichloro diphenyl trichloroethane (DDT): Non-water soluble chlorinated hydrocarbon in use since Second World War (1939-1945) ...

Non-water soluble chlorinated hydrocarbon in use since Second World War (1939-1945) as an insecticide for the control of lice (that spread typhus) and mosquitoes (that spread malaria and yellow fever). But its primary use was in agriculture to control plant pests , specially those affecting cotton and tobacco. Being non-biodegradable it persists in nature and climbs up the food chain by accumulating in body fat (in which it is soluble), causing widespread poisoning of birds and other small animals and plants . Even small concentrations of DDT (0.01 part per million) retard photosynthesis in plankton by 20 percent , and one part per billion ppb) has been known to kill 39 percent of the sea water shrimp in three weeks . Since 1970s, its manufacture and use is restricted in most countries and banned in some. Although DDT was synthesized in 1874 by the German chemist Othmar Zeidler, its pesticidal properties were discovered only in 1939 by the Swiss chemist Paul Mueller who won the 1951 Nobel Prize in physiology & medicine. Chemical formula : (ClC6H4)2CHCCl3.

Dichloro-diphenyl-trichloroethane and newer insecticides

PAUL H. MÜLLER Dichloro-diphenyl-trichloroethane and newer insecticides Nobel Lecture, December 11, 1948 Ladies and Gentlemen. Since Perkin synthesized the first ...

P AUL H . M Ü L L E R

Dichloro-diphenyl-trichloroethane and newer insecticides

Nobel Lecture, December 11, 1948

Ladies and Gentlemen.

Since Perkin synthesized the first dyestuff in 1856, about 90 years have elapsed. During this period the synthesis of artificial dyestuffs has gone through enormous development.

The discovery of fuchsines by Emanuel Verguin in 1859, unlocked the triphenylmethane dyestuffs for the dyestuffs chemist.

The very important discovery of the diazo compounds by Peter Griess, in 1858, certainly brought about the most significant reaction in the chemistry of dyestuffs, a quite unique impetus to the development of this field of organic chemistry.

The most important milestones in this development are: the preparation of Bismarck brown by C. Martius in 1863, of Chrysoidine by O. N. Witt in 1876, and Congo Red, the first dyestuff capable of direct application to cotton, by P. Böttiger in 1884.

This large-scale development and the vast mass of experimental material available have called for a variety of attempted explanations. The relationships between composition and colour on the one hand, and fastness on the other, were the subjects of several attempts at theoretical explanation.

Particularly fruitful in this respect was the theory of O. N. Witt on chromophores and auxochromes, and the theory - valid only in special cases - of E. Schirm, J. Prakt. Chem., 144 (1936) 69, concerning the connexion between composition and affinity to cellulose fibre.

Thanks to these results the dyestuffs chemist today, after about 90 years of untiring detailed study, is in the fortunate position to possess certain points of reference enabling him to set up his programme of work in bold outlines.

The field of synthetics is already very much more complicated in the case of the pharmaceutical chemist.

Today, certain basic substances and formulae are known, as for instance salicylic acid, the barbiturates and the sulfonamides which are able to release certain physiological activity.

Long years of patient detailed study have produced explanations of the constitution of important vitamins, hormones and bacteriostatic substances such as penicillin, streptomycin, etc., and later these were, in part, synthesized. Yet in spite of all these results we are still far removed from being able to predict with any degree of reliability, the physiological activity to be expected from any given constitution. In other words, the connexion between constitution and action are so far still quite unexplained. In addition we have the particularly difficult conditions caused by the uncertainty of tests on living material.

More difficult still are the relationships in the field of pesticides, and in particular of synthetic insecticides.

First of all then, we could consider synthesizing natural insecticides such as pyrethrum and rotenone of known composition and obtained from plants, but quite irrespective of the excessive price entailed in providing a synthesis of such a highly complicated chemical substance there are, as we shall soon see, other grave disadvantages.

We must be clear on this point, namely that we are in fact, moving into unknown territory where there are no points of reference to begin with so that we can only proceed by feeling our way. To this too we must add the

grave difficulties of biological trials requiring multiple controls of the results.

When, in about 1935 and on behalf of my Company, J.R. Geigy A.G. in Basel, I began to study the field of insecticides, and in particular those insecticides of importance to agriculture, the situation looked desperate indeed. Already an immense amount of literature existed on the subject and a flood of patents had been taken out. Yet of the many patented pesticides there were practically none on the market and my own investigations showed that they were not comparable with known insecticides such as the arsenates, pyrethrum or rotenone.

This gave me the courage to press on. In other respects too, the chances were worse than poor; only a particularly cheap or remarkably effective insecticide had any prospects of being used in agriculture, since the demands put upon an agricultural insecticide must necessarily be strict. I relied upon my determination and powers of observation. I considered what my ideal insecticide should look like, and the properties it should possess.

I soon realized that a contact or "touch" insecticide would possess very much better prospects than an oral poison. The properties of this ideal insecticide should be as follows:

1. Great insect toxicity.

2. Rapid onset of toxic action.
3. Little or no mammalian or plant toxicity.
4. No irritant effect and no or only a faint odour (in any case not an unpleasant one).
5. The range of action should be as wide as possible, and cover as many Arthropoda as possible.
6. Long, persistent action, i.e. good chemical stability.
7. Low price (= economic application).

Known insecticides can be grouped as follows under these seven headings:

First of all, a substance had to be found with greater contact insecticidal properties and this was obviously not so easy. My biological trials were conducted in a large glass chamber, of the Peet-Grady type, in which I put Calliphora vomitoria treated with a fine spray of the substance to be tested, in a non-toxic solvent. Suitable non-toxic or relatively non-toxic solvents are ethyl alcohol and acetone. I conducted my early experiments myself in those days and still do so today since, in my view, nothing is better than making one’s own observations.

Such personally conducted biological investigations stimulate the chemist in his work, and at the same time he learns, by his observations, to understand the problems and uncertainties of biological tests and is thus better able to appreciate the difficulties facing his colleagues who work in biology. He will also test substances synthesized by himself with far more keenness and understanding than will a biologist working at a distance from him, who, unconsciously tends in time to lose interest because the tedious chemical formulae mean little to him.

Sometimes new and valuable discoveries may be made by small changes in methods of application, or again by the correct observation of apparently unimportant side-effects.

Our natural-science branches are today specialized in the extreme. This is

useful when some-small detail is to be examined; in my judgement, however, it becomes a fatal disadvantage when two branches - in our case chemical and biological - become intermingled.

After the fruitless testing of hundreds of various substances I must admit that it was not easy to discover a good contact insecticide. In the field of natural science only persistence and sustained hard work will produce results, and so I said to myself "Now, more than ever, must I continue with the

search. " This capacity I owe probably - apart from my inborn determination - to strict upbringing by my teacher, Professor Fichter, who taught us that in chemistry results can only be achieved by using the utmost patience.

From earlier trials I knew that compounds with the group -CH2Cl, for instance -CO-CH 2 Cl, often showed a certain activity. From the work done at the same time in our firm by Dr. H. Martin and his colleagues on toxic substances for the control of moths, in which I personally took no part, I knew that compounds of the general formula:

frequently showed considerable oral toxicity to moths.

In studying the literature, I came across an article by Chattaway and Muir in the J. Chem. Soc., 1934, p. 701, which described the preparation of diphenyl-trichloroethane

I remembered my earlier experiments with substances comprising the -CH2Cl group, for instance laurylchloroacetate, etc. and I was curious to know what effects the CCl3 groups would have on the activity of contact insecticides.

The substance was prepared in September 1939 and tests showed a very considerable contact-insecticidal activity on flies. I began to prepare derivates from this basic material and, probably influenced by the results in the field of moth control, I synthesized the p,p'-dichloro combination

This compound, originally made as long ago as 1873 by an Austrian student in the course of his thesis, now showed a strong insecticidal contact action such as I had to date never observed in any substance.

My fly cage was so toxic after a short period that even after very thorough cleaning of the cage, untreated flies, on touching the walls, fell to the floor. I could carry on my trials only after dismantling the cage, having it thoroughly cleaned and after that leaving it for about one month in the open air.

Similar observations were made by American scientists during their first tests of dichlorodiphenyl-trichloroethane :

"The toxic action of DDT is so strong that some of the scientists who first used it, ruined important experiments because they failed to clean their insect cages before using them again, and the small amounts of DDT remaining were sufficient to kill the new insects introduced." (From How Magic is DDT , by General James Stevens Simmons, 1945, p. 4.)

Later the material was tested on other insects such as aphids, gnats (Culex) and finally cockchafers, Colorado beetles, etc. In all cases the new compound acted, although it often killed only in a matter of hours or days. This is also the reason why initially the biologists were not very interested in the substance; pyrethrum and rotenone had accustomed them to expect a rapid knock-down and they did not understand that long residual activity far outweighed the slow toxic process.

Finally these laboratory results were confirmed by field trials conducted by the research stations at both Wadenswil and Oerlikon (Switzerland) and by our own field trials, and it was found that the effects on the Colorado beetle lasted from four to six weeks.

DDT insecticides have now been introduced into all possible fields of insect control, for instance in hygiene, textile protection, storage and plant protec-

tion. It is noteworthy that in Sweden the application of DDT for timber protection is also being studied, and has already had considerable success.

After the recognition of the strong contact action of dichloro-diphenyl-trichloroethane all possible derivates and analogues were synthesized both by us and later also abroad, and tested.

It is very surprising indeed how little has come of these trials, and it only indicates how strictly specific the action is. When the slighest, seemingly unimportant change is made in the dichloro-diphenyl-trichloroethane molecule, the whole wide range of activity possessed by the original substance is lost.

This is already shown by the isomers : e.g. the o,p'-compound

is present as an impurity to the extent of about 10% in the raw p,p'-dichloro-diphenyl-trichloroethane and its activity is, depending upon the insects treated, about 10-20 times less than that of the p,p'-combination.

The o,o'-combination, estimated at 1% of the raw substance, behaves in the same way; it is only very slightly active.

The m,p'-combination, which may be prepared from

on the other hand, again shows a relatively strong insecticidal action which does not, however, approach that of the p,p'-isomer.

A survey of the properties of the isomers known to date has been made by my colleague Dr. Gatzi.

Of the many analogues and related substances of dichlorodiphenyl-trichloroethane which were prepared, only a few have any real application as insecticides.

The most interesting perhaps is p,p'difluorodiphenyl-trichloroethane, which has shown a somewhat faster contact-insecticidal activity than p,p'-dichlorodiphenyl-trichloroethane but an insufficient residual activity, in addition to which the cost is too high for widespread use. Another interesting insecticide is p,p'dimethoxydiphenyl-trichloroethane, recommended particularly in America where, with some specific pests, it worked to some extent better than its older brother. It possesses the great advantage of having somewhat less mammalian toxicity than the DDT insecticide, but in the main its action is too specific.

A further specific insecticide is p,p'-ditolyl-trichloroethane

which is however too selective and has therefore found little application.

p,p'-Dichlorodiphenyl-dichloroethane, the so-called DDD

Cl

also has certain uses and is easily obtainable from dichloracetal and chloro-

benzol. It is said to be equally toxic to mosquito larvae as dichloro-diphenyl-trichloroethane but at the same time less toxic to mammals. After great initial optimism, little is heard of it nowadays; clearly the product has failed to satisfy in some respect.

Naturally, the synthesis of these closely related substances of dichloro-diphenyl-trichloroethane does not represent the end of the road, and work continues uninterrupted.

The industrial chemist is, in this field, not in the happy position of his academic colleague of being able to freely publish his findings. In view of possible patents which might be taken out, he must bide his time until it has been confirmed as quite certain that no other application is possible.

Since the discovery of dichloro-diphenyl-trichloroethane a number of other insecticides have been discovered.

γ -Hexachlorocyclohexane, again a substance long known to man (Michael Faraday first produced it in 1825 as the mixed isomers α, p, y,6) has found its uses as an insecticide for specific applications. Its very clinging, unpleasant smell militates against widespread use.

During the war, I. G. Farben developed an aphicide based on hexaethyl-tetraphosphate and having the complicated formula:

Today, the Americans (Victor Chemical Co.) have superseded this with their synthesized tetraethyl pyrophosphate having the formula :

A chlorinated camphene, the formula for which is not firmly established but is probably:

was developed in America by the Hercules Powder Co. and was successfully used against certain cotton pests.

"Velsicol 1068" or "Chlordane", also called "Octachlor" is manufactured by the Velsicol Company and the Julius Hyman Corporation. The general formula for this compound is C10H 6Cl 8. We seem here to be dealing with chlorinated dicyclopentadiene.

The most active insecticide in every respect, surpassing in most cases even dichlorodiphenyl-trichloroethane, is a phosphorus compound developed during the War by the German firm I.G. Farben. In Germany it is known as "E 605 ", while in America where large-scale trials have already been held it goes under the name of "Parathion"; it consists of p-nitrophenyldiethyl-thiophosphoric acid ester having the formula:

In the beginning all these insecticides were greeted with great optimism, and people prophesied only a short life for dichloro-diphenyl-trichloroethane.

Some of these insecticides have found a special application for certain purposes, and will probably continue to be used for these. E. 605 is possibly the most interesting, but it must be excluded in cases demanding the least possible toxicity to humans and maximum residual activity. E. 605 is a substance having a very high mammalian toxicity and decomposes rapidly on exposure to air and light, and because of this it is of little use in the fight against epidemic diseases.

On the whole there is now less talk of this new insecticide and DDT has, particularly in the field of hygiene, maintained and even improved its dominating position.

Ladies and gentlemen, we are today on the threshold of a new develop-

ment in the field of pest control. Whilst before we had to depend on inorganic poisons such as the arsenates or the perishable active substances of

plants such as pyrethrum or rotenone, we now have, besides dichloro-diphenyl-trichloroethane, a whole series of substances capable of being synthesized. This development appears to be paving a way similar to that in the field of dyestuffs, so that gradually insecticides and other pest-control agents will be available providing a whole range of specific properties.

In many respects this development can perhaps better be compared to that followed for several decades by the synthesis of medical products as outlined by S. Frankel in his Die Arzneimittel-Synthese (Synthesis of Medical Products), p. 9. In a somewhat more succinct and clear form, he says:

"The artist sees as his purpose not the slavish imitation of Nature degraded by art to simple reproduction. He rather employs his subjective conception of beauty in order to give birth to a new beauty which Nature does not offer in precisely that form. He creates this by using a natural form of representation which is none the less characteristic of the artist.

Similarly the synthetics chemist must create new types of substances, inspired by the action of substances found in Nature and guided by his chemical and pharmaco-dynamic knowledge of active groups.

In so doing, his imagination, too, must be given full rein, just as the artist creates from contemplation of what, subjectively, appears to him as being beautiful."

But at this stage it is of course presumptuous to wish to look into the future; the new fields are still hidden and decades of years ofpatient, painstaking study by biologists and chemists still lie ahead before one day we will perhaps finally be able to recognize the relationships between chemical constitution and mode of action.

Most certainly it was premature, when the insecticidal activity of dichloro-diphenyl-trichloroethane had only just been recognized and scant experimental material was made available, for theories regarding this relationship to be advanced from various quarters. Owing to the lack of appropriate bases these were found to be wrong, and further development has since confirmed this to be so.

Even the mode of action of DDT in poisoning an insect is far from fully explained. We may indeed assume with a fair degree of certainty that the poison enters the insect's body via the sense-organs and somehow travels along the nervous system. (In other words, dichloro-diphenyl-trichloroethane is a nerve poison, like pyrethrum.) However, the real cause of death

is today still unknown in spite of the fact that all over the world, particularly in America and here in Europe, numerous laboratories are trying to find the answer to this question. The problem is not made easier by the fact that we now know that certain insects such as bees which develop a higher temperature in their hives and presumably also have a higher body-temperature than other insects, by virtue of this develop immunity to dichloro-diphenyl-trichloroethane.

The field of pest control is immense, and many problems impatiently await a solution. A new territory has opened up for the synthetics chemist, a territory which is still unexplored and difficult, but which holds out the hope that in time further progress will be made.

I am grateful and glad that I have been permitted to lay a first foundation stone in this puzzling and apparently endless domain,

Teen Essay About Endangered/extinct species

http://www.teenink.com/hot_topics/environment/article/20292/Dichloro-Diphenyl-Trichloroethane/ December 09, 2014

Dichloro-Diphenyl-Trichloroethane – by itself, this chemical compound means nothing to the average person. However, when you realize that its abbreviation is DDT ...

Dichloro-Diphenyl-Trichloroethane – by itself, this chemical compound means nothing to the average person. However, when you realize that its abbreviation is DDT, you may recall the controversy surrounding this pesticide a couple of decades ago, which led to a ban in the United States. Concern over the detrimental effects of DDT on the environment and

people's health was the main reason for the ban. Yet, for some reason, historically reputable organizations like the World Health Organization are now endorsing its use in developing countries.

After World War II, DDT became a household item for most Americans. It was used on lawns – an area where children and pets spend much of their time. It was also used on crops to control insects. However, DDT is far from selective. In fact, it killed not only insects, but also birds and fish. These animals often suffered seizures and blindness before they died. DDT was later associated with a significant decrease in the bald eagle population, causing America's national symbol to become endangered.

Initially, Americans failed to realize that DDT was harmful. Several years after it became prevalent, people began to notice it was adversely affecting wildlife, even beyond immediate deaths. They observed that the remaining birds had difficulty reproducing. Their eggs would not hatch, or if they did, the chicks died almost immediately. Scientists realized the problem was a result of extremely thin and fragile shells. They soon discovered a strong inverse correlation between the level of DDE (the breakdown product of DDT) in the eggs and shell thickness. This proved that DDT was now not only responsible for the direct deaths of many birds, but was also indirectly driving species to extinction by interfering with reproduction.

Recent studies have suggested that DDT could be affecting humans in a similar way. DDT and DDE have been found to act as endocrine-mimicking compounds. In other words, DDT and DDE are so chemically similar to the hormone estrogen that they can trigger hormonal responses in the body, wreaking havoc by upsetting the body's delicate balance. This disruption in the body's hormones has been linked to breast, prostate, and ovarian cancers. Research suggests that exposure to DDT can make it difficult for women to give birth and can cause reproductive difficulties for men as well.

Yet, in spite of all this knowledge, many still advocate the use of DDT in developing nations, especially Africa. They claim that DDT will help fight malaria by killing disease-carrying mosquitos. History has clearly proved, however, that DDT harms everything in its path – including humans. In addition, it has been shown many times that a target species often - becomes resistant to the chemical and its population can experience a strong rebound. Thus, not only would DDT harm other plants and animals, but it could still fail to eradicate malaria in Africa in the long run.

What is even more distressing is that DDT biodegrades very slowly, with a half-life as long as 15 years. Traces of DDT can still be found in our bodies, and there is every reason to suspect that there are many other undiscovered ways in which DDT is adversely affecting us. Therefore, it makes no sense to continue polluting our planet with a chemical that has been responsible for many species becoming endangered or downright extinct. What's to say that the same thing couldn't eventually happen to us?

This piece has been published in Teen Ink's monthly print magazine.

Dichloro-diphenyl-trichloroethane (DDT/DDE)

http://www.breastcancerfund.org/clear-science/radiation-chemicals-and-breast-cancer/ddt.html December 09, 2014

Dichloro-diphenyl-trichloroethane (DDT/DDE) CATEGORY*: IARC possible carcinogen, NTP reasonably anticipated carcinogen, Endocrine disruptor FOUND IN: Banned in many ...

FOUND IN: Banned in many countries, still used for malaria control in 17 countries, persists in the environment even where it is not used

THE GIST: Rachel Carson made DDT famous in 1961 with the publication of Silent Spring, a groundbreaking book that spurred the environmental movement—and the passage of important legislation to regulate environmental toxicants. Because of Carson's work, the United States banned DDT in 1972. The world followed suit—and the chemical was banned for agricultural use under the Stockholm Convention. But it continues to be used in Africa to control malaria. In spite of the ban, the chemical remains in our systems.

DDT was the first widely used synthetic pesticide. It is credited on the one hand with the eradication of malaria in the United States and Europe and on the other with long-term devastating effects on reproductive success in wildlife and with adverse health effects in humans (Beard, 2006). Banned in most countries for agricultural use, DDT is still used for malaria control in many countries, especially in sub-Saharan Africa (WHO, 2007).

Because of its continued use and its persistence in the environment, DDT is found worldwide. Most animals, including humans, ingest DDT-contaminated foods and retain the chemical and its main metabolite, DDE. Substantial concentrations of DDT and DDE are still found in the body fat of humans and animals as well as in human breast milk and placenta, even in areas where it has not been used for a long time (Rogan, 2007; Shen, 2007; Zheng, 1999).

Epidemiological data are mixed regarding the effects of DDT/DDE on breast cancer risk (Brody, 2007). For example, one study from the Long Island Breast Cancer Study Project did not find an association between DDT/DDE (or polychlorinated biphenyls, PCBs) and breast cancer (Gammon, 2002). However, like many such studies that did not find reliable connections between DDT or DDE levels and risk for breast cancer, this project measured contaminant levels near the time of breast cancer diagnosis, without regard to possible exposures during critical early periods of breast development, and did not consider the effect of chemical mixtures nor assess key metabolites. As we now know, exposures to toxic chemicals during critical periods of mammary tissue development may lead to effects on breast cancer rates that are not observed when exposures take place exclusively or primarily in adulthood (Cohn, 2011).

An important study explored women's estimated DDT levels based on aggregate data from their year of birth (to ascertain historic exposures to DDT levels), as well as blood DDT levels at the time the women gave birth to their first children. Researchers then followed the women over the next two decades, noting cases when women either were diagnosed with breast cancer (invasive or non-invasive) before the age 50, or had died from breast cancer before the age of 50. Results show that exposure to DDT during childhood and early adolescence (younger than age 14) was associated with a fivefold increase in risk of developing breast cancer before the age of 50. The younger the women were during the period of heavy DDT use (1945 to 1972), the greater the effect. At the time of the study, the authors noted, "Many U.S. women heavily exposed to DDT in childhood have not yet reached age 50. The public health significance of DDT exposure in early life may be large" (Cohn, 2007).

Supporting the premise that exposures to DDT are associated with increased risk of breast cancer is a recent review comparing the association between disease risk and DDT use in developed countries, where DDT has been banned for several decades, and in developing countries, where DDT use is still prevalent. The association between DDT levels and breast cancer was much stronger in developing countries, where women of the age to be diagnosed with breast cancer also would have been exposed to DDT during critical periods of development (Shakeel, 2010).

Laboratory studies have found that, in addition to being directly genotoxic or carcinogenic (Canales-Aguirre, 2011), the estrogen-like form of DDT enhances the growth of estrogen-

receptor-positive (ER+) mammary tumors (Robison, 1985; Scribner, 1981; Uppala, 2005). ER+ tumors are the most common type of breast cancer. The percentage of breast tumors in the United States that are ER+ rose from 73 percent in 1973 to 78 percent in 1992. This is the period when women who were young girls during the highest use of DDT in the 1950s were reaching the age that post-menopausal breast cancer begins to be diagnosed (Pujol, 1994). Another study, looking at chemical levels in breast fat tissue, did not find an association of DDT/DDE with ER+ tumors. However, data from this study indicated a statistically significant association of higher concentrations of these compounds in breast tissue with tumors that were more aggressive and that had poorer prognoses (Woolcott, 2001).

*For chemicals that have been shown to be carcinogens, we provide classifications from two authoritative bodies: the International Agency for Research on Cancer (IARC, an international body) and the National Toxicology Program (NTP, a division of the U.S. Department of Health and Human Services). We have categorized endocrine-disrupting compounds where the body of peer-reviewed research indicates a strong foundation for doing so.

Dichloro-diphenyl-trichloroethane definition of Dichloro-diphenyl-trichloroethane in the Free Online Encyclopedia.

http://encyclopedia2.thefreedictionary.com/Dichloro-diphenyl-trichloroethane December 09, 2014

Army's Office of Malaria Control in War Areas began to use dichloro-diphenyl-trichloroethane (DDT) to combat the mosquitos that carried malaria.

or 2,2-bis(p-chlorophenyl)-1,1,1,-trichloroethane, chlorinated hydrocarbon compound used as an chemical, biological, or other agents used to destroy insect pests; the term commonly refers to chemical agents only. Chemical Insecticides

..... Click the link for more information. . First introduced during the 1940s, it killed insects that spread disease and fed on crops, and Swiss scientist Paul , 1899–1965, Swiss chemist, Ph.D. Univ. of Basel, 1925. He worked as a research scientist with J. R. Geigy A.G. in Basel, Switzerland.

..... Click the link for more information. was awarded the 1948 Nobel Prize in Physiology or Medicine for discovering (1939) DDT's insecticidal properties. DDT, however, is toxic to many animals, including humans, and it is not easily degraded into nonpoisonous substances and can remain in the environment and the food chain for prolonged periods. By the 1960s its harmful effects on the reproductive systems of fish and birds were apparent in the United States, where the insecticide had been heavily used for agricultural purposes. After the United States banned its use in 1972, the affected wildlife population recovered, particularly the bald eagle and the osprey. Nevertheless, DDT use continues in parts of the world, particularly in tropical regions, to control the mosquitoes that spread infectious parasitic disease that can be either acute or chronic and is frequently recurrent. Malaria is common in Africa, Central and South America, the Mediterranean countries, Asia, and many of the Pacific islands.

..... Click the link for more information. . In 2001 the Stockholm Convention on Persistent Organic Pollutants called for the phasing out of DDT once a cost-effective alternative becomes available. or 2,2-bis(-chlorophenyl)-1,1,1,-trichloroethane, chlorinated hydrocarbon compound used as an insecticide . First introduced during the 1940s, it killed insects that spread disease and fed on crops, and Swiss scientist Paul Müller was awarded the 1948 Nobel Prize in Physiology or Medicine for discovering (1939) DDT's insecticidal properties. DDT, however, is toxic to many animals, including humans, and it is not easily

degraded into nonpoisonous substances and can remain in the environment and the food chain for prolonged periods. By the 1960s its harmful effects on the reproductive systems of fish and birds were apparent in the United States, where the insecticide had been heavily used for agricultural purposes. After the United States banned its use in 1972, the affected wildlife population recovered, particularly the bald eagle and the osprey. Nevertheless, DDT use continues in parts of the world, particularly in tropical regions, to control the mosquitoes that spread malaria . In 2001 the Stockholm Convention on Persistent Organic Pollutants called for the phasing out of DDT once a cost-effective alternative becomes available. See D. Kinkela, DDT and the American Century (2011). dichlorodiphenyltrichloroethane; a colourless odourless substance used as an insecticide. It is toxic to animals and is known to accumulate in the tissues. It is now banned in the UK Common name for an insecticide; melting point 108.5°C, insoluble in water, very soluble in ethanol and acetone, colorless, and odorless; especially useful against agricultural pests, flies, lice, and mosquitoes. Also known as dichlorodiphenyltrichloroethane. DDT - Generic term for a program that assists in debugging other programs by showing individual machine instructions in a readable symbolic form and letting the user change them. In this sense the term DDT is now archaic, having been widely displaced by "debugger" or names of individual programs like "adb", "sdb", "dbx", or "gdb". DDT - Under MIT's fabled ITS operating system, DDT (running under the alias HACTRN) was also used as the shell or top level command language used to execute other programs. DDT - Any one of several specific debuggers supported on early DEC hardware. The DEC PDP-10 Reference Handbook (1969) contained a footnote on the first page of the documentation for DDT that illuminates the origin of the term:

Historical footnote: DDT was developed at MIT for the PDP-1 computer in 1961. At that time DDT stood for "DEC Debugging Tape". Since then, the idea of an on-line debugging program has propagated throughout the computer industry. DDT programs are now available for all DEC computers. Since media other than tape are now frequently used, the more descriptive name "Dynamic Debugging Technique" has been adopted, retaining the DDT abbreviation. Confusion between DDT-10 and another well known pesticide, dichloro-diphenyl-trichloroethane (C14-H9-Cl5) should be minimal since each attacks a different, and apparently mutually exclusive, class of bugs.

(The "tape" referred to was, incidentally, not magnetic but paper.) Sadly, this quotation was removed from later editions of the handbook after the suits took over and DEC became much more "businesslike".

The history above is known to many old-time hackers. But there's more: Peter Samson, compiler of the original TMRC lexicon, reports that he named "DDT" after a similar tool on the TX-0 computer, the direct ancestor of the PDP-1 built at MIT's Lincoln Lab in 1957. The debugger on that ground-breaking machine (the first transistorised computer) rejoiced in the name FLIT (FLexowriter Interrogation Tape).

Dichloro-diphenyl-trichloroethane

http://www.cyclopaedia.de/wiki/Dichloro-diphenyl-trichloroethane December 09, 2014

Der Begriff Dichloro-diphenyl-trichloroethane ist im englisch-sprachigen Wikipedia aufgeführt. Dort heißt es dazu: DDT (dichlorodiphenyltrichloroethane) is a ...

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tasteless and almost odorlessknown for its insecticidal properties. [Technical DDT][further explanation needed] has been formulated in almost every conceivable form, including solutions in xylene or petroleum distillates , emulsifiable, water-, granules, aerosols,and charges for vaporisers and lotions.First synthesized in 1874, DDT's insecticidal action was discovered by the Swiss chemist Paul Hermann Müller in 1939. It was then used with great success in the second half of World War II to control malaria and typhus among civilians and troops. After the war, DDT was made available for use as an agricultural insecticide and its production and use duly increased. Müller was awarded the Nobel Prize in Physiology or Medicine "for his discovery of the high efficiency of DDT as a contact poison against several arthropods" in 1948.In 1962, the book Silent Spring by American biologist Rachel Carson was published. It catalogued the environmental impacts of indiscriminate DDTin the United States and questioned the logic of releasing large amounts of chemicals into the environment without a sufficient understanding of their effects on ecology or human health. The book suggested that DDT and other pesticides may cause cancer and that their agricultural use was a threat to wildlife, particularly birds. Its publication was a seminal event as regards theand resulted in a large public outcry that eventually led, in 1972, to a ban on the agricultural use of DDT in the United States. A worldwide ban on its agricultural use was later formalised under the, but its limited use in disease vectorcontinues to this day and remains controversial.Along with the passage of the, the US ban on DDT is cited by scientists as a major factor in the comeback of the(the) and thefrom near- extinction in the contiguous United States.Dies ist ein Auszug aus dem Artikel Dichloro-diphenyl-trichloroethane der freien Enzyklopädie Wikipedia. In der Wikipedia ist eine Liste der Autoren verfügbar.

DDT (dichlorodiphenyltrichloroethane)

http://pmep.cce.cornell.edu/profiles/extoxnet/carbaryl-dicrotophos/ddt-ext.html December 09, 2014

DDT is no longer registered for use in the United States, ... Initial degradates in mammalian systems are 1,1-dichloro-2,2-bis(p-dichlorodiphenyl)ethylene ...

It is available in several different forms: aerosols, dustable powders, emulsifiable concentrates, granules and wettable powders (1, 2). It is reported to be compatible with many other pesticides and incompatible with alkaline substances (1). Many insect pests may have developed resistance to DDT (1).

Unless otherwise specified, the toxicological, environmental effects and environmental fate and chemistry data presented here refer to the technical product DDT. Technical grade DDT is actually a mixture of three isomers of DDT, principally the p,p'-DDT isomer (ca. 85%), with the o,p'-DDT and o,o'-DDT isomers typically present in much lesser amounts (3).

One-time administration of DDT to rats at doses of 50 mg/kg led to decreased thyroid function and a single dose of 150 mg/kg led to increased blood levels of liver-produced enzymes and changes in the cellular chemistry in the central nervous system of monkeys (3). Single doses of 50-160 mg/kg produced tremors in rats, and single doses of 160 mg/kg produced hind leg paralysis in guinea pigs (3). Mice suffered convulsions following a one-time oral dose of 200 mg/kg. Single administrations of low doses to developing 10- day old mice are reported to have caused subtle effects on their neurological development (3).

DDT is slightly to practically non-toxic to test animals via the dermal route, with reported dermal LD50s of 2,500-3,000 mg/kg in female rats (1-3), 1000 in guinea pigs (3) and 300 in rabbits (3). It is not readily absorbed through the skin unless it is in solution (3).

It is thought that inhalation exposure to DDT will not result in significant absorption through the lung alveoli (tiny gas-exchange sacs) but rather that it is probably trapped in mucous

secretions and swallowed by exposed individuals following the tracheo-bronchial clearance of secretions by the cilia (3).

Acute effects likely in humans due to low to moderate exposure may include nausea, diarrhea, increased liver enzyme activity, irritation (of the eyes, nose or throat), disturbed gait, malaise and excitability; at higher doses, tremors and convulsions are possible (3, 6). While adults appear to tolerate moderate to high ingested doses of up to 280 mg/kg, a case of fatal poisoning was seen in a child who ingested one ounce of a 5% DDT:kerosene solution (3).

The main effect on the liver seen in animal studies was localized liver damage. This effect was seen in rats given 3.75 mg/kg/day over 36 weeks, rats exposed to 5 mg/kg/day over 2 years and dogs at doses of 80 mg/kg/day over the course of 39 months (3). In many cases lower doses produced subtle changes in liver cell physiology, and in some cases higher doses produced more severe effects (3). In mice doses of 8.33 mg/kg/day over 28 days caused increased liver weight and increased liver enzyme activity (3). Liver enzymes are commonly involved in detoxification of foreign compounds, so it is unclear whether increased liver enzyme activity in itself would constitute an adverse effect. In some species (monkeys and hamsters), doses as high as 8-20 mg/kg/day caused no observed adverse effects over exposure periods as long as 3.5-7 years (3).

Kidney effects observed in animal studies include adrenal gland hemorrhage in dogs at doses of 138.5 mg/kg/day over 10 days and adrenal gland damage at 50 mg/kg day over 150 days in dogs (3). Kidney damage was also seen in rats at doses of 10 mg/kg/day over 27 months (3).

Immunological effects observed in test animals include: reduced antibody formation in mice following administration of 13 mg/kg/day for 3-12 weeks and reduced levels of immune cells in rats at doses of 1 mg/kg/day (3). No immune system effects were observed in mice at doses of 6.5 mg/kg/day for 3-12 weeks (3).

Dose levels at which effects were observed in test animals are very much higher than those which may be typically encountered by humans (4). The most significant source of exposure to individuals in the United States is occupational, occurring only to those who work or worked in the production or formulation of DDT products for export (5). Analysis of U. S. market basket surveys showed approximately a 30-fold decrease in detected levels of DDT and metabolites in foodstuffs from 1969-1974, and another threefold drop from 1975-1981, with a final estimated daily dose of approximately 0.002 mg/person/day (3). Based on a standard 70-kg person, this results in a daily intake of approximately 0.00003 mg/kg/day. Due to the persistence of DDT and its metabolites in the environment, very low levels may continue to be detected in foodstuffs grown in some areas of prior use (3). It has been suggested that, depending on patterns of international DDT use and trade, it is possible that dietary exposure levels may actually increase over time (3). Persons eating fish contaminated with DDT or metabolites may also be exposed via bioaccumulation of the compound in fish (3).

Even though current dietary levels are quite low, past and current exposures may result in measurable body burdens due to its persistence in the body (3). More information on the metabolism and storage of DDT and its metabolites in mammalian systems is provided below (Fate in Humans and Animals).

Adverse effects on the liver, kidney and immune system due to DDT exposure have not been demonstrated in humans in any of the studies which have been conducted to date (3).

Available epidemiological evidence from two studies does not indicate that reproductive

effects have occurred in humans as a result of DDT exposure (3). No associations between maternal blood levels of DDT and miscarriage nor premature rupture of fetal membranes were observed in two separate studies (3, 7, 8). One study did report a significant association between maternal DDT blood levels and miscarriage, but the presence of other organochlorine chemicals (e.g., PCBs) in maternal blood which may have accounted for the effect make it impossible to attribute the effect to DDT and its metabolites (9).

In humans, blood cell cultures of men occupationally exposed to DDT showed an increase in chromosomal damage. In a separate study, significant increases in chromosomal damage were reported in workers who had direct and indirect occupational exposure to DDT (3). Thus it appears that DDT may have the potential to cause genotoxic effects in humans, but does not appear to be strongly mutagenic. It is unclear whether these effects may occur at exposure levels likely to be encountered by most people.

In other studies, however, no carcinogenic activity was observed in rats at doses less than 25 mg/kg/day; no carcinogenic activity was seen in mice with at doses of 3-23 mg/kg/day over an unspecified period, and in other hamster studies there have been no indications of carcinogenic effects (3).

The available epidemiological evidence regarding DDT's carcinogenicity in humans, when taken as a whole, does not suggest that DDT and its metabolites are carcinogenic in humans at likely dose levels (3). In several epimiological studies, no significant associations were seen between DDT exposure and disease, but in one other study, a weak association was observed (3, 10). In this latter study, which found a significant association between long-term, high DDT exposures and pancreatic cancers in chemical workers, there were questions raised as to the reliability of the medical records of a large proportion of the cancer cases (3, 10).

DDT is very slowly transformed in animal systems (4). Initial degradates in mammalian systems are 1,1-dichloro-2,2-bis(p-dichlorodiphenyl)ethylene (DDE) and 1,1-dichloro-2,2-bis(p-chlorophenyl)ethane (DDD), which are very readily stored in fatty tissues (3). These compounds in turn are ultimately transformed into bis(dichlorodiphenyl) acetic acid (DDA) via other metabolites at a very slow rate (3). DDA, or conjugates of DDA, are readily excreted via the urine (3).

Available data from analysis of human blood and fat tissue samples collected in the early 1970s showed detectable levels in all samples, but a downward trend in the levels over time (3). Later study of blood samples collected in the latter half of the 1970s showed that blood levels were declining further, but DDT or metabolites were still seen in a very high proportion of the samples (3). Levels of DDT or metabolites may occur in fatty tissues (e.g. fat cells, the brain, etc.) at levels of up to several hundred times that seen in the blood (3). DDT or metabolites may also be elminated via mother's milk by lactating women (3).

There has been much concern over chronic exposure of bird species to DDT and effects on reproduction, especially eggshell thinning and embryo deaths (12). The mechanisms of eggshell thinning are not fully understood. It is thought that this may occur from the major metabolite, DDE, and that predator species of birds are the most sensitive to these effects (12). Laboratory studies on bird reproduction have demonstrated the potential of DDT and DDE to cause subtle effects on courtship behavior, delays in pairing and egg laying and decreases in egg weight in ring doves and Bengalese finches (12). The implications of these for long-term survival and reproduction of wild bird species is unclear.

There is evidence that synergism may be possible between DDT's metabolites and organophosphate (cholinesterase-inhibiting) pesticides to produce greater toxicity to the nervous system and higher mortality (12). Aroclor (polychlorinated biphenyls, or PCBs) may

result in additive effects on eggshell thinning (12).

DDT is very highly toxic to fish species as well. Reported 96-hour LC50s are less than 10 ug/L in coho salmon (4.0 ug/L), rainbow trout (8.7 ug/L), northern pike (2.7 ug/L), black bullhead (4.8 ug/L), bluegill sunfish (8.6 ug/L), largemouth bass (1.5 ug/L), and walleye (2.9 ug/L) (13). The reported 96-hour LC50s in fathead minnow and channel catfish are 21.5 ug/L and 12.2 ug/L respectively (13). Other reported 96-hour LC50s in largemouth bass and guppy were 1.5 ug/L and 56 ug/L respectively (12). Observed toxicity in coho and chinook salmon was greater in smaller fish than in larger (12). It is reported that DDT levels of 1 ng/L in Lake Michigan were sufficient to affect the hatching of coho salmon eggs (14). DDT may be moderately toxic to some amphibian species and larval stages are probably more susceptible than adults (11, 12).

In addition to acute toxic effects, DDT may bioaccumulate significantly in fish and other aquatic species, leading to long-term exposure. This occurs mainly through uptake from sediment and water into aquatic flora and fauna, and also fish (12). Fish uptake of DDT from the water will be size-dependent with smaller fish taking up relatively more than larger fish (12). A half- time for elimination of DDT from rainbow trout was estimated to be 160 days (12).

The reported bioconcentration factor for DDT is 1,000 to 1,000,000 in various aquatic species (15), and bioaccumulation may occur in some species at very low environmental concentrations (13). Bioaccumulation may also result in exposure to species which prey on fish or other aquatic organisms (e.g., birds of prey).

Earthworms are not susceptible to acute effects of DDT and its metabolites at levels higher than those likely to be found in the environment, but they may serve as an exposure source to species that feed on them (12). DDT is non-toxic to bees; the reported topical LD50 for DDT in honeybees is 27 ug/bee (12). Laboratory studies indicate that bats may be affected by DDT released from stored body fat during long migratory periods (12).

Due to its extremely low solubility in water, DDT will be retained to a greater degree by soils and soil fractions with higher proportions of soil organic matter (12). It may accumulate in the top soil layer in situations where heavy applications are (or were) made annually; e.g., for apples (2). Generally DDT is tightly sorbed by soil organic matter, but it (along with its metabolites) has been detected in many locations in soil and groundwater where it may be available to organisms (12, 15). This is probably due to its high persistence; although it is immobile or only very slightly mobile, over very long periods of time it may be able to eventually leach into groundwater, especially in soils with little soil organic matter.

Residues at the surface of the soil are much more likely to be broken down or otherwise dissipated than those below several inches (14). Studies in Arizona have shown that volatilization losses may be significant and rapid in soils with very low organic matter content (desert soils) and high irradiance of sunlight, with volatilization losses reported as high as 50% in 5 months (17). In other soils (Hood River and Medford) this rate may be as low as 17- 18% over 5 years (17). Volatilization loss will vary with the amount of DDT applied, proportion of soil organic matter, proximity to soil-air interface and the amount of sunlight (12).

DDT has been widely detected in ambient surface water sampling in the United States at a median level of 1 ng/L (part per trillion) (3, 6).

water, effects, environmental, pollutants, United States, impact, pesticide, toxic, world, human, use, health

http://www.pollutionissues.com/Co-Ea/DDT-Dichlorodiphenyl-Trichloroethane.html December 09, 2014

DDT, dichlorodiphenyl trichloroethane, was synthesized in 1874, but its insecticidal properties were first identified in 1939 by P.H. Mueller.

DDT, dichlorodiphenyl trichloroethane, was synthesized in 1874, but its insecticidal properties were first identified in 1939 by P.H. Mueller. He received the Nobel Prize for his discovery, which coincided with the outbreak of World War II, when DDT was used extensively to keep soldiers free of head and body lice. DDT also proved very effective against mosquitoes, which transmit a serious global human disease, malaria, as well as yellow fever. After the war, DDT was developed extensively as an agricultural pesticide.

DDT has an extremely low volatility and may be the least soluble chemical known, which makes it extremely persistent in soils and aquatic sediments. It has relatively low acute mammalian toxicity and is toxic to a wide range of insects. It kills insects by affecting the transmission of nerve impulses, probably by influencing the delicate balance of sodium and potassium within the neuron.

More than four billion pounds of DDT have been used throughout the world since 1940. Production in the United States peaked in 1961 when 160 million pounds were manufactured. Large economic benefits have resulted from the control of many serious agricultural and forestry pests, including Colorado potato beetle, cotton boll weevil, and pests of fruit, vegetables, corn, and tobacco. In forestry, its greatest success occurred in combating

spruce budworm and gypsy moth. However, its major impact lay in the control of mosquitoes that transmit malaria, as well as body lice and fleas; many millions of lives have been saved through these uses.

DDT's potentially adverse environmental effects were brought to public attention by Rachel Carson in her book Silent Spring (1963). Carson emphasized the great persistence of DDT in soils and river sediments and focused on the bioconcentration of DDT through the trophic levels of food chains. One result of the bioaccumulation of DDT was the thinning of the eggshells of predatory birds such as bald eagles, peregrine falcons, golden eagles, hawks, and pelicans, resulting in embryonic death and decreasing populations of these species. DDT bioconcentrates because it has low water solubility and high fat solubility, that is, a high lipid-to-water partition coefficient (e.g., it can concentrate into fatty tissues from water). In the 1960s large DDT residues in human tissues and human milk began to be reported, probably from the consumption of food containing traces of DDT. DDT in body fat was reported to cause convulsions in laboratory rats; it also reached human fetuses by crossing the placenta. However, few serious effects on human health were officially recorded.

Many pests began to develop resistance to DDT, necessitating the progressive use of more of the pesticide to control such pests. In 1972 the use of DDT in the United States was banned on environmental grounds, including the widespread contamination of the environment with DDT, its ability to bioconcentrate, and its effects on endangered bird species.

Suitable alternatives to DDT were found in the United States and other industrialized countries that also banned its use in the 1970s. However, tropical developing countries that used inexpensive DDT extensively to control malaria and other pests faced a significant dilemma. Moreover, although the United States no longer used DDT, it continued to manufacture and export very large quantities to developing countries and how much DDT is still used. It is difficult to say with accuracy exactly which countries still use DDT. Some

countries use it illegally, others only in small quantities. And information is often impossible to obtain because questionnaires from an organization like the World Health Organization (WHO) generally have only a 50 to 60 percent response rate. Nonetheless, it is known that poorer countries in Central and South America, Africa, and Asia, as well as the large nation of China, continue to utilize sizable quantities of DDT.

Dichloro-diphenyl-trichloroethane

http://www.science.gc.ca/default.asp?Lang=En&n=730D78B4-1 December 09, 2014

DDT - Dichloro-diphenyl-trichloroethane. Watch in Windows Media | Real Media | Video Player Help 4:01 min. Teacher's Guide. Summary. Science can help us to take ...

Science can help us to take action to improve our health and the environment. Banning DDT in 1972 is considered one of the most important steps in protecting wildlife and people from the effects of pesticides and has set the course for future research into the effects of the chemicals we use.

Gillian Deacon

This week on Earth Tones, we're looking at some of the environmental advances that affect our daily lives. Tonight, a development with an unexpectedly negative and lingering impact on earth.

Jay Ingram

At one time, DDT was probably the most commonly used pesticide in North America. By the early '70's, more than one billion kilograms had been released into the environment. DDT was so popular because it's a one size fits all pesticide - kills many different kinds of bugs. But DDT doesn't just kill bugs... it's harmful to all kinds of creatures, especially birds.

Dr. Chip Weseloh

DDT affected the egg shell on birds. Normal eggshells are quite thick. The birds sit on them, then incubate them - but DDT made the egg shells thinner, so when the birds sat on them the eggshell broke, and there were no young produced, so the populations declined.

Jay Ingram

Some bird populations declined more than others. They were more sensitive to DDT's harmful effects.

Dr. Chip Weseloh

Uh... the birds that were affected most by DDT were the ones at the top of the food chain - herring gulls and some of the other fish eating birds, cormorants and brown pelicans - some of the raptors - peregrine falcon in particular. And birds weren't affected equally. Some are much more sensitive than others. The peregrine is very, very sensitive. The double crested cormorant is quite sensitive. The herring gull isn't very sensitive. But the birds at the top of the food chain were the ones that were most affected.

Jay Ingram

That's because DDT is what's called a very persistent chemical. It hangs around in the environment for a long time. For instance, DDT may persist in water for as long as 150 years! It stays in the tissues of all the animals that ingest it. That's why birds at the top of

the food chain get a bigger dose. It's called bio-magnification or bio-accumulation.

Dr. Chip Weseloh

... and those are terms that refer to the buildup of a compound as you go up the food chain. DDT, for instance, is in the water and it goes into the small organisms, slightly bigger organisms, and it gets up into the top of the food chain... and by that time it's magnified, where it becomes a greater concentration than it was down at the bottom of the food chain.

Jay Ingram

Birds weren't the only ones beings affected. Mounting evidence of DDT's harmful effects led our government to ban it for most uses in 1969.

Dr. Chip Weseloh

It's not nearly as big a problem as it used to be. Back 10 or 15 or 20 years ago it was a much greater problem because there was more in the atmosphere and more in the environment. It's still bio-accumulating and bio-concentrating, but it's not doing as much as it used to. So the values, the concentrations, are lower now than they used to be, but yes, it's still accumulating.

Jay Ingram

But the news isn't all bad. The ban on DDT has had an effect.

Dr. Chip Weseloh

Oh I think there's lots of cause for optimism. I mean, things are not nearly as bad as they used to be and they're getting better all the time. We have a program going where we've been measuring chemicals in herring gull eggs for 25 years and uh... the levels are much, much less than they used to be. They're about 10 percent of what they were 25 years ago. So they've declined about 90 percent and that's good. I mean, we've made big strides, but we've got a little bit left to go. I mean, eventually we'd like to get to absolutely no chemicals in these birds, and so there's a ways to go. We have to keep working to keep the regulations there and to try and get rid of the bit that's left in the environment.

Earth Tones is produced in co-operation with Environment Canada.

History: the ban of Dichloro Diphenyl Trichloroethane (DDT)

http://faircompanies.com/news/view/history-ban-dichloro-diphenyl-trichloroethane-ddt/ December 09, 2014

He became one of the most famous Modernist architects- responsible for many of Barcelona’s most famous monuments-, but ...

A synthetic compound used as an insecticide and pesticide to fight human illnesses and agricultural pests, until in the seventies it was proven toxic and dangerous.

Despite being prohibited as a pesticide, its medical use continues, especially to control malaria.

Dicloro-Difenil-Tricloroetano (DDT) is a compound soluble in oils and organic solvents, although insoluble in water.

Despite having been synthesized in 1874, its amazing insecticide properties (it was defined as the "miracle compound", given its effects) weren't discovered until 1939, coinciding with

the beginning of World War II, where it was used successful to combat typhus, malaria and other illnesses transmitted by mosquitos and other insects, among both the troops and the civilian population.

After the war, the efficiency of DDT extended its use as a pesticide and its production increased dramatically. DDT was used in a generalized manner to protect crops from pests worldwide, until the 1970s when the public learned about the environmental effects of using this synthetic compound.

In her successful book Silent Spring (1962), Rachel Carson explained the environmental damages derived from the use of DDT. Carson predicted that, if the use of DDT continued, all the birds in the world would disappear.

With the help of other scientific personalities, Rachel Carson alerted the world of the of the danger of irreversible contamination of food, since the compound accumulates in food chains.

Once Carson's studies were ratified by other biologists and scientists, the U.S. Environmental Protection Agency (EPA) banned the use of DDT in 1972.

Its prohibition extended to other countries (the first to adopt a ban where Norway and Sweden; the U.S., and in the eighties European countries like the United Kingdom). However, in countries like India its use hasn't been banned.

Despite an order in 1971 from the U.S. District Court of Appeals to begin the de-registration process for DDT, EPA administrator William Ruckelshaus initially rejected an outright ban stating it to be proven safe and non-toxic to humans and animals. Due to public outcry, in the summer of 1972 Ruckelshaus announced a ban on most uses of DDT:

To prevent malaria without endangering people, animals and plants

In 2006 the World Health Organization announced that it would return to using DDT as an insecticide to eradicate malaria (and to kill the mosquitos that transmit the disease).

Although this international organization considers that its use isn't harmful to nature and, instead, effective in the prevention of this disease, its use continues to be controversial.

The United Nations Environment Program (UNEP) called for the elimination of 12 compounds that "can kill people, damage the nervous and immune systems, cause cancer and reproductive disorders and interfere with normal infant and child development," among those was DDT.

Due to its characteristics, according to the Stockholm Convention on Persistent Organic Pollutants (POPs) from May of 2005, DDT was classified as one of these compounds:

Those in favor of the use of DDT to combat malaria argue that:

However, the environmental community and part of the scientific community don't believe that DDT is benign:

The effects of DDT on human health are controversial, although many studies present varying results:

Among the diverse effects of DDT on the health of animals, many highlight:

Dichloro-diphenyl-trichloroethane

http://www.cyclopaedia.fr/wiki/Dichloro-diphenyl-trichloroethane December 09, 2014

Résultats pour "Dichloro-diphenyl-trichloroethane" sur Internet, dans les universités et dans les œuvres littéraires cyclopaedia.net

Le terme Dichloro-diphenyl-trichloroethane est cité dans le Wikipedia de langue anglaise. Il est défini comme suit:DDT (dichlorodiphenyltrichloroethane) is a colorless,, tasteless and almost odorlessknown for its insecticidal properties. [Technical DDT][further explanation needed] has been formulated in almost every conceivable form, including solutions in xylene or petroleum distillates, emulsifiable concentrates, water-, granules, aerosols,and charges for vaporisers and lotions.First synthesized in 1874, DDT's insecticidal action was discovered by the Swiss chemist Paul Hermann Müller in 1939. It was then used in the second half of World War II to control malaria and typhus among civilians and troops. After the war, DDT was made available for use as an agricultural insecticide and its production and use duly increased. Müller was awarded the Nobel Prize in Physiology or Medicine "for his discovery of the high efficiency of DDT as a contact poison against several arthropods" in 1948.Ceci est un extrait de l'article Dichloro-diphenyl-trichloroethane de l'encyclopédie libre Wikipedia. La liste des auteurs est disponible sur Wikipedia.

DDT Dichloro Diphenyl Trichloroethane

http://www.ukessays.com/essays/environmental-studies/ddt-dichloro-diphenyl-trichloroethane.php December 09, 2014

DDT Dichloro Diphenyl Trichloroethane. A Review of the Potential Threat of DichloroDiphenylTrichloroethane (DDT) on the Environment. Abstract. The case of DDT is a ...

A Review of the Potential Threat of DichloroDiphenylTrichloroethane (DDT) on the Environment

The case of DDT is a classic example of our ignorance in failing to predict the effect of the widespread use of this highly persistent pesticide on the environment (Shaw I, Chadwick J 1998). Initially DDT was spectacularly successful particularly in the control of malaria, as well as against agricultural pests. However numerous studies have highlighted a number of serious environmental effects associated with the indiscriminate use of DDT and its derivatives DDD and DDE. These effects including high level of persistence in the environment, biomagnification, bioaccumulation, and its potential to mimic hormones and thereby disrupt endocrine systems in wildlife and possibly humans, have led to wide-ranging restrictions on its use worldwide. This paper highlights the known effects of DDT on the environment.

DichloroDiphenyl Trichloroethane (DDT) was first discovered over 130 years ago by a German graduate student, and the formula lay forgotten for over 70 years until was rediscovered by a Swiss entomologist Dr. Paul Muller when researching for an insecticide to use against the common clothes moths (J Chadwick, I C Shaw, 1998). In the 1950s and 1960s large amounts of DDT were released into environment (air, soil and water) when it was sprayed on crops and forests to control insects including use to control malaria causing mosquitoes. DDT highly acclaimed as a miracle for its success and commonly used with some 4 x 105 tons of DDT being manufactured worldwide in 1964. However its production decreased as its toxic effects got to be understood.

DDT and its related insecticides, eldrin, dieldrin and aldrin are termed as organochlorine (OC) pesticides. They are all heavily chlorinated hydrophobic ion channel inhibitors and are very toxic (Shaw I, Chadwick J 1998). DDT is a also classified under a wider group of

toxicants commonly known as a Persistent Organic Pollutant (POPs). POPs are

The fate and behavior of DDT in the environment is directly related to its chemical and physical properties.

DDT's chemical formula is C14H9Cl5. So, for every molecule of DDT, there are 14 carbon atoms, 9 hydrogen atoms, and 5 chlorine atoms.

Before its ban in 1973 in the U.S.A and other developed countries, the main sources of DDT and its entry into the environment were its widespread production and use as a pesticide or insecticide. It must be noted that although many countries have subscribed to the banning of the importation or use DDT, there are number of countries still using DDT, including China, some countries in Africa, and central and South Africa. Other sources of DDT include leaching from waste sites, soils and other media as result of past use. DDD dichlorodiphenyldichloroethane and DDE dichlorodiphenyldichloroethylene are chemicals derivatives of DDT enter environment as a result of breakdown of DDT. DDE has no commercial use, while DDD was also used as a pesticide, but its use has also been banned. Table 1 below shows the global decline in production of DDT since 1964.

Table 2 Global production of DDT since 1964, showing its decline following the realization that it was extremely toxic to the environment after prolonged sue

g This figure includes DDT plus other OCs

Figure 2 Countries which still permit the import of DDT

Data from Problems with POPs, Towards better alternatives, Consumers International, Pesticides Trust [now PAN UK], WWF, 1996

Due to its chemical properties DDT, and its derivatives DDD and DDE, once released into the environment, can be transported from one medium to another over time and space. DDT can evaporate from initial sources into the air and can then be deposited on land or surface water. This cycle of evaporation and deposition may be repeated many times. As a result, DDT, DDE, and DDD may be carried long distances in the atmosphere. These chemicals have been found in bogs, snow, and animals in the Arctic and Antarctica regions, far from where they have been used. The half-life (that is, the time it takes for one-half of the chemical to turn into something else) of these chemicals in the atmosphere as vapors has been calculated to be approximately 1.5-3.0 days. However, in reality, this half-life estimate is too short to account for the ability of DDT, DDE, and DDD to be carried long distances in the atmosphere.

Further to the above, in order to properly understand the pathways, persistence and toxicity of DDT in the environment, one must understand the concept of bioaccumulation and biomagnifications. Knowledge of feeding mechanisms can enable you to predict the extent of the potential toxic effect of a chemical on the environment (Shaw I, Chadwick J 1998). Biomagnification or bioaccumulation is the accumulation of a substance up the food chain by transfer of residues of the substance in smaller organisms that are food for larger organisms in the chain. It generally refers to the sequence of processes that result in higher concentrations in organisms at higher trophic levels in the food chain (Spacie, A., McCarty, L.S., and Rand, G.M., 1995). The table below illustrates bioaccumulation up a food chain.

Table 3 Concentration of DDT in east coast estuary in U.S.A

Persistence of DDT in the Environment

The chemical stability of DDT and its fat solubility are the key factors that lead to its persistence in the environment. DDT is not metabolized very rapidly by animals; instead, it is deposited and stored in the fatty tissues. The biological half-life of DDT is about eight years; that is, it takes about eight years for an animal to metabolize half of the amount it assimilates. If ingestion continues at a steady rate, DDT builds up within the animal over time. DDT is known to be highly persistence in other environment media including soils, water, and vegetation.

Persistence of DDT in soils and groundwater

DDT is very highly persistent in the environment, with a reported half life of between 2-15 years and is immobile in most soils. Routes of loss and degradation include runoff, volatilization, photolysis and biodegradation (aerobic and anaerobic). These processes generally occur only very slowly. Breakdown products in the soil environment are DDE and DDD, which are also highly persistent and have similar chemical and physical properties. Due to its extremely low solubility in water, DDT will be retained to a greater degree by soils and soil fractions with higher proportions of soil organic matter. It may accumulate in the top soil layer in situations where heavy applications are (or were) made annually; e.g., for apples. Generally DDT is tightly adsorbed by soil organic matter, but it (along with its metabolites) has been detected in many locations in soil and groundwater where it may be available to organisms. This is probably due to its high persistence; although it is immobile or only very slightly mobile, over very long periods of time it may be able to eventually leach into groundwater, especially in soils with little soil organic matter. Residues at the surface of the soil are much more likely to be broken down or otherwise dissipated than those below several inches.

DDT may reach surface waters primarily by runoff, atmospheric transport, drift, or by direct application (e.g. to control mosquito-borne malaria). The reported half-life for DDT in the water environment is 56 days in lake water and approximately 28 days in river water. The main pathways for loss are volatilization, photo-degradation, adsorption to water-borne particulates and sedimentation. Aquatic organisms, as noted above, also readily take up and store DDT and its metabolites. Field and laboratory studies in the United Kingdom demonstrated that very little breakdown of DDT occurred in estuary sediments over the course of 46 days. DDT has been widely detected in ambient surface water sampling in the United States at a median level of 1 ng/L (part per trillion).

DDT does not appear to be taken up or stored by plants to a great extent. It was not translocated into alfalfa or soybean plants, and only trace amounts of DDT or its metabolites were observed in carrots, radishes and turnips all grown in DDT-treated soils. Some accumulation was reported in grain, maize and rice plants, but little translocation occurred and residues were located primarily in the roots.

A common measure of toxicity of any toxicant is the lethal dose (LD) and/or lethal concentration (LC) that causes death resulting from a single or limited exposure in 50% of the treated the population. LD is generally expressed as the dose, in milligrams (mg) of chemical per kilogram (kg) of body weight. LC is often expressed as mg of chemical per volume (e.g., litre (L) of medium (i.e., air or water) the organism is exposed to. Chemicals are considered highly toxic when the LD /LC is small and practically non-toxic when the figure is large. However, the LD /LC does not reflect any effects from long term exposure (i.e., cancer, birth defects or reproductive toxicity) that may occur at levels below those which cause death. In order to properly d the discussion of the toxicological effect of DDT; one must understand LD /LC

In birds Reported dietary LD s range from greater than 2,240 mg/kg in mallard, 841 mg/kg in Japanese quail and 1,334 mg/kg in pheasant. In birds, exposure to DDT occurs mainly

through the food web through predation on aquatic and/or terrestrial species having body burdens of DDT, such as fish, earthworms and other birds. However there has been much concern over chronic exposure of bird species to DDT and effects on reproduction, especially eggshell thinning and embryo deaths. The idea that DDT was responsible for the decline in population of much bird species because it thinned and weakened their egg shells was a central tenet of Rachel Carson's Silent Spring (1962).

The toxicity of DDT in aquatic species ranges from moderate in some amphibian larval stages to very highly toxic in some aquatic invertebrates (see table 4). In addition to acute toxic effects, DDT may bioaccumulate significantly in fish and other aquatic species, leading to long-term exposure. This occurs mainly through uptake from sediment and water into aquatic flora and fauna, and also fish. Fish uptake of DDT from the water will be size-dependent with smaller fish taking up relatively more than larger fish. A half-life for elimination of DDT from rainbow trout was estimated to be 160 days (. The reported bioconcentration factor for DDT is 1,000 to 1,000,000 in various aquatic species, and bioaccumulation may occur in some species at very low environmental concentrations. Bioaccumulation may also result in exposure to species which prey on fish or other aquatic organisms (e.g., birds of prey).

Table 4 Toxicity of DDT in various aquatic invertebrates

Effects of DDT on other animals

Some detritus feeders including earthworms are not known to be susceptible to acute effects of DDT and its metabolites at levels higher than those likely to be found in the environment, but they may serve as an exposure source to species that feed on them. DDT is non-toxic to bees; the reported topical LD for DDT in honeybees is 27ug/bee. Laboratory studies indicate that bats may be affected by DDT released from stored body fat during long migratory periods.

DDT and its potential effects on the environment have been by far the longest studied and most understood among all toxicants especially since Rachel Carson's book Silent Spring (1962). Rachel Carson caught the world's attention when she documented detrimental effects of pesticides particularly DDT on the environment, especially its effects on birds. DDT has been found to cause thinning of egg shells and result in reproductive problems in bird species such as the Peregrine Falcon, which has led to the near extinction of these birds in some parts of the world.

Although DDT had tremendous successes in combating pests and the deadly malaria causing mosquitoes, its known cumulative effects in the environment far outweigh its advantages and this has led to the worldwide call for restrictions and banning of the production and use of DDT and other similar organochlorine pesticides and other Persistent Organic Pollutants.

dichloro-diphenyl-trichloroethane « Angelbabe43's Blog

https://angelbabe43.wordpress.com/category/dichloro-diphenyl-trichloroethane/ December 09, 2014

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Heralded as a scientific miracle, the pesticide Dichloro-diphenyl-trichloroethane (DDT) was used to control malaria during World War II. By the mid 1950s, Americans were duped into allowing over 31 million acres of land to be sprayed with this “harmless” poison. Years later congressional reports tell a totally different story.

Yet scientists of the day knew DDT stored in fatty tissue "…at least as early as 1945 that DDT presents a potential residue problem." [1] Stammers, et al. noted in 1947, "The possibilities of cumulative effects from storage of DDT in milk and tissues of sheep and cattle require further investigation." [2] In the late 1940's, Schechter and colleagues developed methods to detect contamination, "of such products as milk, butter, eggs, meat, and fats when farm animals consume DDT-treated feed," and show that DDT, "can be excreted in milk." [3]

Remember those dates, folks, because claiming ignorance that "we didn't know back then" doesn't quite work anymore. Moreover, it was hoped the DDT contamination problem would "just go away" and those provisions were inserted in section 331(c) of the Economic Opportunity Act of 1964.

"Until recently there was reasonable prospect that the problem [pesticide residues] would have diminished to a point by June 30, 1967, that further extension of the authority would not be necessary. However, in recent months, several large producers in the Rio Grande Valley have had their milk removed from the market because of DDT residue. The local dairymen, Texas and New Mexico State agencies, dairy and cotton associations, and our Agricultural Research Service, have undertaken to determine the causes by tests in Arizona."

It's pretty apparent the public never was supposed to know about that happening.

Got DDT?

When it was obvious pesticide residue levels weren't dropping as hoped, the Dairy Indemnity Payment Program, or DIPP, officially was created through public law in 1968 to reimburse farmers whose milk was contaminated with pesticides. However, in reading the congressional Dairy Indemnity Payments reports that talk candidly about the widespread contamination, it is then that we see the full extent of the cavalier agricultural practices promoted by the U.S. Government.

"The problem which our dairy farmers are facing has been brought about by the use of chemicals approved by the Federal Government to dust crops. Some of these chemicals have been found to contaminate feeds. The contamination passes on into the milk and when the residues of pesticides is [sic] found to be of too high a level, the farmers are forced to dump their milk, taking it out of commercial channels. The result has been disastrous to the dairy farmers involved, some of which have had to go into bankruptcy."

"There had been at that time, reasonable prospect that the problem would have diminished to a point that further extension of the authority would not have been necessary. However, as the Department testified then, several large producers had their milk removed from the market because of DDT residue. The problem had in fact not been solved. The local dairymen, the State governments involved, dairy and cotton associations, and the U.S. Department of Agriculture have been cooperating in an effort to rid us of this problem. The problem, however, still continues and it appears that it will continue for the forseeable (sic) years to come."

What does DDT have to do with vaccines? Well, I'm glad you asked.

A Spoonful of MediSIN…

To prove the safety of DDT, apologists often point anecdotally to Merck Chemist, Joseph J. Jacobs, slurping spoonfuls of the white stuff before public speeches. This act, to them, apparently validates the chemical's safety across the board for all persons. Fortunately,

Joseph wasn't lactating at the time.

Wait a minute! Did I just say Merck? Who do you think was a producer of DDT?

Not only did Merck mass-produce DDT for controlling malaria in Italy during WWII, Merck also formulated and manufactured a polio vaccine in response to the 1952 polio epidemic. The detection of the cancer-causing virus (SV40) in polio vaccine by Merck scientist, Maurice Hilleman, will be the focus of a future article. (http://en.wikipedia.org/wiki/Maurice_Hilleman)

Henry Kumm worked at the International Health Division of the Rockefeller Foundation for Medical Research in 1928. During World War II, Kumm experimented with larvicides containing DDT to control the spread of malaria in Italy. [4]

Additionally, according to the Medical Archives at John Hopkins,

"In 1951 [a year before the polio outbreak], he resigned from the Rockefeller Foundation to accept a position as assistant director of research at the National Foundation for Infantile Paralysis. He conducted field trials in the study of gamma globulin and the Salk vaccine and became the director of research in 1954. Rejoining the Rockefeller Foundation in 1959, Kumm retired as an associate professor in 1964." [5]

To tie up a few loose ends, you should know the National Foundation for Infantile Paralysis (NFIP) was supposed to investigate the cause of polio. That foundation also had invested millions of dollars into researching a possible polio vaccine prior to the 1952 polio epidemic.

Years after the polio epidemic in 1979, researchers Gabliks and Utz reported, "Studies in cell cultures with insecticides…indicated increased replication of poliovirus in human cells exposed to Kelthane, Karathane, and DDT. Furthermore an activation of a latent virus was also observed in primary rabbit kidney cells grown in the presence of DDT." [6]

Do you think it would be a cold day in hell before Good Old Henry Kumm would make a possible correlation between a polio outbreak and the spraying of pesticides? However, there were more profits to be made from polio vaccines than recommending people stop drinking DDT-contaminated milk, wasn't there?

"The phenomenal growth in production and use of chemical control agents is illustrated by the fact that in 1940 these products had a wholesale value of about $40 million. Today (August 12th, 1959) it is $290 million and is estimated to reach $1 billion by 1975. One-sixth of all croplands and millions of acres of forests, rangelands, and marshlands are treated annually with these chemicals…"

"…Before 1940, relatively small amounts of such chemicals as nicotine, rotenone, pyrethrum, and the aresenicals (sic) were used for insect control. During and following World War II a rapid changeover to DDT, heptachlor, dieldrin, TEPP, malathion, and related compounds occurred."

SOURCE: Authorizing Research on Insecticides, Herbicides, Fungicides, and Other Pesticides by the Secretary of the Interior. 85th Congress, 2nd Session. House of Representatives Report No. 2181. July 16, 1958.

In order to keep the American people hoodwinked about the toxicity of DDT, attempts are still made by corporate interests to dispel the dangers. Yet, each new piece of information brings us shockingly closer to the truth. At the end of the line, the core message still remains stubbornly in-line with the corporate sickness care agenda: take more vaccines,

consume more pills, and wind up getting sicker!

As a result of DDT application, can we just be imagining more cancer, more autism, and more chronic illnesses? Despite superficial attempts by government agencies to 'get to the bottom' of a problem, taxpayers shouldn't be suckered into waiting twenty years to get answers. I certainly don't want to wait at all, since toxic products should not be permitted into the food chain at any phase of production. P.T. Barnum said, "There's a sucker born every minute," and that's what they apparently are relying on.

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1. Wayland, J. Present Status of Our Knowledge of DDT Intoxication. Am. J. of Public Health, (1955) 45: 478-485. 2. Stammers, F.M.G., and Whitefield, F.G.S. The Toxicity of DDT to Men and Animals. Bull. Ent. Res., (1947) 38:1-73.

3. Schechter, M.S., Pogerelskin, M.A., and Haller, H.L. Colorimetric Determination of DDT in Milk and Fatty Materials. U.S. Dept. of Ag. (1947) 19:1.

4. Snowden, F.M. (2006). The Conquest of Malaria: Italy, 1900-1962, (pp. 200). Frederick W. Hilles Publication Fund of Yale University.

5. Medical Archives of The John Hopkins Medical Institutions. Henry W. Kumm Collection. Retrieved March 25th , 2011 from http://www.medicalarchives.jhmi.edu/papers/kumm.html

6. Gabliks, J., and Utz, C. DDT and Inflammatory Responses; 1. Influenza Infection in Mice Fed DDT. Pesticide Biochemistry and Physiology (1979) 12, 257-263.

definition of Dichloro-diphenyl-trichlothroethane by Medical dictionary

http://medical-dictionary.thefreedictionary.com/Dichloro-diphenyl-trichlothroethane December 09, 2014

DDT dichloro-diphenyl-trichloroethane, a powerful insect poison; used in dilution as a powder or in an oily solution as a spray. DDT (d d-t) n ...

dichloro-diphenyl-trichloroethane, a powerful insect poison; used in dilution as a powder or in an oily solution as a spray. n. Dichlorodiphenyltrichloroethane; a colorless contact insecticide, toxic to humans and animals when swallowed or absorbed through the skin, that has been banned in the United States for most uses since 1972. a nonbiodegradable water-insoluble chlorinated hydrocarbon once used worldwide as a major insecticide, especially in agriculture. In recent years knowledge of its adverse impact on the environment has led to restrictions in its use. In addition, because tolerance in formerly susceptible organisms develops rapidly, DDT has been largely replaced by organophosphate insecticides in the United States, where DDT was banned by the FDA in 1971. It is still used as a pediculicide where epidemic-scale delousing is justified, as in barracks and refugee camps. Its value as a scabicide is marginal, because scabies and crab lice quickly become resistant to it. See also scabicide A gene on chromosome 22q11.23 that encodes an enzyme belonging to the MIF family which converts D-dopachrome into 5,6-dihydroxyindole. [ ] a moderately toxic chlorinated hydrocarbon pesticide, formerly widely used but now banned in the United States except for a few specialized purposes because its extremely long half-life causes ecological damage. Dichloro-diphenyl-trichloroethane Environment A highly hepatotoxic and potentially neurotoxic insecticide that accumulates in fat; DDT is non-biodegradable and concentrates up the food chain. See Pesticide dichloro-diphenyl-trichloroethane, a powerful insect poison; used in dilution as a powder or in an oily solution as a spray.

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What is the chemical formula for dichloro dipheyl tricloroethane?

https://in.answers.yahoo.com/question/index?qid=20060926221629AAYw1Co December 09, 2014

What is the chemical formula for dichloro dipheyl tricloroethane? ... (p-chlorophenyl)e... and dichloro-diphenyl-trichloroethane ...

Chemical name 4,4'-(2,2,2-trichloroethane-

1,1-diyl)bis(chlorobenzene)

Chemical formula C14H9Cl5

Molecular mass 354.49 g/mol

Melting point 108.5 째C

Boiling point 260 째C

CAS number 50-29-3

DDT was the first modern pesticide and is arguably the best known organic pesticide. It is a highly hydrophobic colorless solid with a weak, chemical odor that is nearly insoluble in water but has a good solubility in most organic solvents, fat, and oils. DDT is also known under the chemical names 1,1,1-trichloro-2,2-bis(p-chlorophenyl)e... and dichloro-diphenyl-trichloroethane (from which the abbreviation was derived).

DDT was developed as the first of the modern insecticides early in World War II. It was initially used with great effect to combat mosquitoes spreading malaria, typhus, and other insect-borne human diseases among both military and civilian populations, and as an agricultural insecticide. The Swiss chemist Paul Hermann M체ller of Geigy Pharmaceutical in Switzerland was awarded the Nobel Prize in Physiology or Medicine in 1948 "for his discovery of the high efficiency of DDT as a contact poison against several arthropods."

In 1962, American biologist Rachel Carson published the book Silent Spring, which alleged that DDT caused cancer and harmed bird reproduction by thinning egg shells.[1] The book resulted in a large public outcry which eventually led to the insecticide being banned for agricultural use in the USA, and was one of the signature events in the birth of the environmental movement. DDT was subsequently banned for agricultural use in many countries in the 1970s; there is still a great controversy regarding the impact of this decision on the use of DDT to fight disease vectors.

humans, body, used, water, plants, chemical, animals, parts, effects, cause, substance, principle, DDT as an insecticide

http://www.scienceclarified.com/Co-Di/DDT-dichlorodiphenyltrichloroethane.html December 09, 2014

DDT is a synthetic chemical compound once used widely in the United States and throughout the world as a pesticide (a chemical substance used to kill weeds ...

DDT is a synthetic chemical compound once used widely in the United States and throughout the world as a pesticide (a chemical substance used to kill weeds, insects, rodents, or other pests). It is probably best known for its dual nature: although remarkably effective in destroying certain living things that are harmful to plants and animals, it can also

be extremely dangerous to humans and the environment.

The abbreviation DDT stands for dichlorodiphenyltrichloroethane. DDT was first produced in the laboratory in 1873. For more than half a century, it was little more than a laboratory curiosity—a complicated synthetic (produced by scientists) compound with no apparent use.

Then, in 1939, Swiss chemist Paul Hermann Müller (1899–1965) discovered that DDT was highly poisonous to insects. The discovery was very important because of its potential for use in killing insects that cause disease and eat agricultural crops. For his work, Müller was awarded the Nobel Prize in medicine in 1948.

During and after World War II (1939–45), DDT became extremely popular among public health workers, farmers, and foresters. Peak production of the compound reached 386 million pounds (175 million kilograms) globally in 1970. Between 1950 and 1970, 22,204 tons (20,000 metric tons) of DDT was used annually in the former Soviet Union. The greatest use of DDT in the United States occurred in 1959, when 79 million pounds (36 million kilograms) of the chemical were sprayed.

By the early 1970s, however, serious questions were being raised about the environmental effects of DDT. Reports indicated that harmless insects (such as bees), fish, birds, and other animals were being killed or harmed as a result of exposure to DDT. The pesticide was even blamed for the near-extinction of at least one bird, the peregrine falcon. Convinced that the environmental damage from DDT was greater than the compound's possible benefits, the U.S. Environmental Protection Agency banned the use of DDT in the United States in 1973. Its use in certain other countries has continued, however, since some nations face health and environmental problems quite different from those of the United States.

DDT's environmental problems arise because of two important properties: persistence and lipid-solubility. The term persistence refers to the fact that DDT does not break down very easily. Once the pesticide has been used in an area, it is likely to remain there for many years. In addition, DDT does not dissolve in water, although it does dissolve in fatty or oily liquids. (The term lipid-solubility is used because fats and oils are

members of the organic family known as lipids.) Since DDT is not soluble in water, it is not washed away by the rain, adding to its persistence in the environment. But since DDT is lipid-soluble, it tends to concentrate in the body fat of animals. The following sequence of events shows how DDT can become a problem for many animals in a food web.

DDT is used today in such African nations as Zimbabwe and Ethiopia to control mosquitoes and the tsetse fly. These two insects cause serious diseases, such as malaria and sleeping sickness. DDT saves lives when used on the tsetse fly in Lake Kariba in Zimbabwe. But once sprayed on

the lake, DDT does not disappear very quickly. Instead, it is taken up by plants and animals that live in the lake. Studies have shown that the concentration of DDT in the lake itself is only 0.002 parts per billion. But algae in the lake have a concentration of 2.5 parts per million. Other members of the food web also accumulate DDT from the organisms they eat. Fish that feed on the algae have DDT levels of 2 parts per million; tiger-fish and cormorants (both of whom live on the algae-eating fish) have levels of 5 and 10 parts per million, respectively; and crocodiles (who eat both tiger-fish and cormorants) have levels as high as 34 parts per million.

Bans on the use of DDT in the United States and some other nations have given

ecosystems in those countries a chance to recover. Populations of peregrine falcons, for example, have begun to stabilize and grow once again. Many other animal species are no longer at risk from DDT. Of course, poor nations continue to face a more difficult choice than does the United States, since they must balance the protection of the health of their human populations against the protection of their natural ecosystems.

In December 2000, in a convention organized by the United Nations Environment Program, 122 nations agreed to a treaty banning twelve very toxic chemicals. Included among the twelve was DDT. However, the treaty allowed the use of DDT to combat malaria until other alternatives become available. Before it can take effect, the treaty must be ratified by 50 of the nations that agreed to it in principle.

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PAUL H. MÜLLER Dichloro-diphenyl-trichloroethane and newer insecticides Nobel Lecture, December 11, 1948 Ladies and Gentlemen. Since Perkin synthesized the first ...

CHAPTER 9: Environmental Chemical Contaminants and Pesticides ... dichloro-diphenyl-trichloroethane (DDT) and polychlorinated biphenyls (PCBs)), many are very

Apac and Oyam, where indoor residual spray (IRS) was carried out. Uganda re-introduced the use of DDT for malaria control in the districts of Oyam and Apac in 2008

CHEMICAL AND PHYSICAL INFORMATION 4.1 CHEMICAL IDENTITY When we refer to DDT, ... -chlorophenyl)ethane; dichloro diphenyl trichloroethane; DDT; ...

DDT (dichloro-diphenyl-trichloroethane), an inexpensive and highly effective insecticide, had ... led to the banning of DDT in most industrialized countries in

MAMTA MODERN SR. SEC. SCHOOL HOLIDAYS HOMEWORK â Each one agreed they would have failed if they had worked alone, far behind the parents

DDT (50-29-3) Registrations canceled for following uses: Foliar preplant soil application, ... DDT (dichloro-diphenyl-trichloroethane), Federal Register

MINISTRY OF AGRICULTURE (Department of Agriculture and Cooperation) ... The use of Dichloro Diphenyl Trichloroethane for the domestic public health programme is

Kannada, situated near the centre of the hill chain of the ... molecule of Dichloro Diphenyl Trichloroethane (DDT) that was highly effective in control of mosquitoes.

Co-Metabolic Degradation of Dichloro Diphenyl Trichloroethane by a Defined Microbial Consortium

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Persistent organic pollutants such as Dichloro diphenyl trichloroethane (DDT) and related compounds are of particular environmental concern because of their toxicity ...

Pesticides have been widely used to protect and improve the quality and the quantity of food commodities, building materials, clothing, animal health and to combat certain disease transmitting insect vectors. Among the pesticides, chlorinated hydrocarbons such as hexachlorocylohexane (HCH), Dichloro diphenyl trichloroethane (DDT), endosulfan etc. have had their major share since their introduction in 1940s. However, indiscriminate use has caused serious concern about toxic effects of these compounds on non-target organisms and halogenation has been implicated as a reason for persistence and toxicity of these compounds (Neilson et al., 1985). These compounds enter the soil, water and food through several routes e.g., landfill, dumping of industrial wastes, by run-off from treated plant surfaces, spillage during application, use of contaminated manure, drift from aerial and ground applications, erosion of contaminated soil by wind and water into the aquatic system, accidents in transport of insecticides etc. (Johri et al., 1996). The residues of these compounds thus enter the human and animal body via food chain (Smith, 1991). Birds were found to either die or severely affected by DDT use (Cooper, 1991; Fry, 1995). DDT-breakdown products such as DDE were found to thin egg shells, thus significantly reducing the number of chicks that hatched. Although the use of DDT was banned in the western countries way back in 1970s, India continues to use DDT for public health programmes for the control of vectors causing malaria, typhus, dengue etc. Thus with continued production of DDT, the environmental contamination (soil, water, air) continues and it is imperative to develop technologies to degrade these recalcitrant and persistent pollutants. Studies on the degradation of HCH and DDT have been carried out using microbial isolates (Bidlan and Manonmani, 2002; Murthy and Manonmani, 2007). In the present study, we describe the co-metabolic degradation of DDT by microbial consortium in view to enhance the degradation of DDT.

Materials

DDT (99% pure) was procured from Sigma-Aldrich chemical company, Mo, USA. The co-substrates used were purchased from Hi-Media laboratories, Mumbai, India. The solvents

used in extraction of DDT and solvents used for GC were obtained from E. Merck, India. Other chemicals used in media preparations were purchased from standard chemical companies. All the chemicals were of either AR or HPLC grade.

Methods

Microbial Consortium

The defined microbial consortium used in this study was the HCH-degrading consortium developed in our laboratory by long-term enrichment technique (Murthy and Manonmani, 2007). The individual members of the consortium were grown separately in nutrient broth for 72 h under shaking conditions, mixed together at equal OD and were induced with the daily addition of 10 ppm of DDT for 7 days. This induced microbial consortium was used in the co-metabolic degradation of DDT.

Basal Medium

The basal medium, M used for biodegradation studies consisted of (per liter of distilled water) 0.675 g KH PO 5.455 g Na HPO and 0.25 g NH NO . The pH of the medium was 7.5. Medium was sterilized at 121°C for 20 min.

Degradation of DDT

DDT as acetone solution (10 mg mL-1 stock solution) was added at required level to sterile, dry 250 mL Erlenmeyer flasks inside the laminar hood. Acetone was allowed to evaporate and 50 mL of M medium was added to these flasks. The DDT pre-exposed microbial consortium was inoculated at 500 µg protein mL-1 level. The flasks were incubated in a rotary shaker (180 rpm) at ambient temperature (26-28°C). Samples were collected at regular intervals and used for the analysis of residual DDT.

To study the co-metabolic degradation of DDT, different co-substrates were supplemented at 0.5% level. The rest of the protocols were same as that used in degradation studies.

All the experiments were done in triplicates.

Analytical Methods

Extraction of Residual DDT

The sample removed after required period of incubation was acidified to pH 2.0. Residual DDT was extracted thrice from the acidified culture broth with equal volumes of dichloromethane in a separating funnel. Sample and solvent were shaken vigorously for 5 min and the two layers were allowed to separate out. The solvent layers were pooled, passed through anhydrous sodium sulphate and then through florisil. The solvent was allowed to evaporate and the residue was resuspended in a known volume of acetone for further analysis. The recovery of DDT by this method was 95±2%.

Growth

Growth of bacterial strains was determined by estimating the total protein in the biomass by modified method of Lowry et al. (1951). Cells were harvested from a suitable quantity of culture broth, washed with minimal medium, suspended in 3.4 mL distilled water and 0.6 mL of 20% NaOH. This was mixed and digested in a constant boiling water bath for 10 min. Total protein, in cooled sample of this hydrolysate, was estimated by using Folin-Ciocalteau reagent. A total of 0.5 mL of the hydrolysate was taken in a clean test tube. To this was added 5.0 mL of Lowrys C. After 10 min, 0.5 mL of Lowrys D (Folin-Ciocalteau reagent (1:2)

was added and mixed well. The colour was read at 660nm after 20.0 min of standing at room temperature, using a spectrophotometer (Shimadzu UV-160A, Japan). Total amount of protein was computed using the standard curve prepared with BSA (Bovine serum Albumin).

Gas Chromatography

Concentrated residual substrate, after passing through activated florisil, was resuspended in a known volume of HPLC grade acetone and gas chromatography was done using Chemito 1000 series gas chromatograph (Nasik, India). One microlitre of the extract suspension was injected in to a BP-5 capillary column (30x0.25 mm ID) set at 180°C and programmed as: 180°C for 10 min and a rise at the rate of 2°C min-1 up to 220°C and maintained there for 2 min. Injector was maintained at 250°C while electron capture detector (Ni63) was maintained at 280°C. Pure nitrogen gas was used as the carrier at the rate of 1 mL min-1. Under these conditions, the retention time for standard DDT was 28.16 min. Quantification of DDT in the sample was carried out using the area under the peak and the standard under same conditions.

The key to the assessment of the fate of organic chemicals in the environment is the realistic evaluation of their susceptibility to mineralization. The major factor determining the susceptibility of an organic compound to microbial attack is the length of the time it has been exposed, which can enforce the microorganisms to initiate mineralization. Then care must be taken in establishing a degrading population. With this in view, an organochlorine pesticide (HCH) degrading microbial consortium was acclimated with DDT by enrichment technique in shake flasks. After seven days of continuous pre-exposure to DDT, the microbial consortium was observed to possess the capacity to degrade 10 ppm of DDT under aerobic conditions. All the ten members of the microbial consortium survived during acclimation of DDT indicating that the presence of DDT as a sole did not cause deleterious effect on the survivability of the members of the consortial population. The problem of foreignness was not observed with these members because of their exposure to a new recalcitrant compound and at 10 ppm level DDT was observed to be non-toxic. This acclimated microbial consortium was used for studies on the degradation of DDT.

Degradation of 10 ppm of DDT

The microbial consortium was found to degrade 10 ppm of DDT upto 65% by 10 days (data not shown). Only 10% of DDT was found to be degraded by 24 h of incubation (Table 2). The degradation increased slowly with time and by 10 days of incubation, 65% of DDT was found to be degraded. With increase in incubation time, there was no substantial improvement in the degradation. The rate of degradation was 0.0034 µg mL-1 day-1. All the organisms were found to survive by the end of ten days of incubation period (Table 1) which indicated that all the isolates had the machinery to degrade DDT, which might be a synergistic action. There was practically very little degradation in abiotic controls (data not shown). Only 1.8% degradation was observed after 10 days of incubation.

The biodegradation of any compound will be a success if enzymes are synthesized in response to the presence of a recognisable substrate and the genetic capability of the microorganism(s) in action. As most of these enzymes are inducible enzymes, the degradation capability of any organism(s) depends on the extent of induction of these degrading enzymes i.e., the requisite quantity of the requisite enzyme. The degradation of 65% of 10 ppm DDT by 240 h of incubation without further improvement indicated that sufficient quantities of the enzymes might not have been produced to carry out complete degradation which could be due to insufficient quantity of microbial cells or there could have been inhibition of the enzyme activity by the intermediates formed during degradation. If the xenobiotic substrate, DDT is incapable of causing induction of the requisite quantity of

the enzyme(s), due to fact that microbial cells would act as resting cells and DDT-acting as non-growth substrate, the increase in biodegradation could be achieved by the addition of a growth substrate during the degradation of a non-growth substrate, DDT. With this in view, enhancement in DDT degradation was tried with the addition of few co-substrates.

Co-metabolic Degradation of DDT

Among twelve different co-substrates used along with 10 ppm of DDT, yeast extract was found to be the best helping with 74.56% degradation by 72 h (Table 3) at the rate of 0.0174 µg mL-1 day-1. The degradation was 2.319 times more than the control (without co-substrate). This was followed by beef extract and glucose which showed 55.98% degradation at 0.0110 µg mL-1 day-1 (Table 3). This was observed to be 1.74 times more than the control. Co-substrates such as glycerol and TSB showed inhibitory effects towards DDT-degradation. The degradation was found to decrease by 0.464 and 0.327 times, respectively compared to control, with the rate of degradation being 0.0023 and 0.0015 µg mL-1 day-1, respectively. However, the growth of the consortium in the presence of these co-substrates did not show any relation to degradation (Table 2).

That is, the growth of the consortium was highest in glycerol, which showed inhibitory effects towards degradation of DDT. Glucose showed less growth (around four times less than that in glycerol) but helped in better degradation of DDT (56%). Nitrogenous substances supported better growth of the consortium compared to simple sugars and s. It was clear that the presence of a growth substrate i.e., co-metabolite assisted in the better transformation or degradation of DDT i.e., the compound showing resistance to degradation. This kind of situation has been used to make a distinction between co-metabolism and gratuitous metabolism by Dalton and Stirling (1982), wherein substantial amount of degradation would not be observed with pre-grown cells because these cells could not extract energy from the subsequent metabolism of it to drive the first requiring step. Only when a growth substrate or other transformable compound was present that could yield energy to drive the initial reaction would transformation occur and this has been defined as co-metabolism (Dalton and Stirling, 1982).

When the composition of the microbial consortium was studied, all the cultures showed increase in their numbers during co-metabolic degradation whereas without co-substrates, isolate numbers 3 and 8 slightly decreased in number by 72 h (Table 1). These members of the consortium might have been necessary for synergistic complete degradation. However, the necessity of these isolates for completion of degradation of DDT needs to be worked out in detail. In the presence of co-metabolite, the increase in cell number of all members could help in concerted attack by many microbes within the community that can lead to complete degradation i.e., the co-metabolic metabolite by one or few species of the community could be degraded by another species of organism within the community (Bull, 1980). This clearly indicates significant coordinative interaction within the community wherein, degradation can be completed accurately by the mixed-culture system. Perhaps, difficulty is associated in proving the existence of specific interactions based on effects in microbial communities. In our studies, in the microbial consortium, all the species were found to be present. There were no dominants, associates or incidentals. It was not clear which organism(s) were primary utilizers and which were secondary utilizers. The community as a whole might be playing an important role in biodegradation.

Yeast extract at 1% level assisted in co-metabolic degradation. Concentration lesser to this showed lesser degradation. An increase in yeast extract concentration did not show any further increase in degradation (Fig. 1).

Co-metabolic degradation of DDT by microbial consortium was maximum at 72 h of incubation period (Fig. 2). Degradation was 94% by 72 h and reached 100% only by 120 h

i.e., disappearance of DDT after 94% of degradation was slow.

All microorganisms cannot utilize the xenobiotic compound as the sole source of carbon and energy. In such cases, co-metabolism enhances the degradation of the compound. Effect of different co-substrates on the degradation of DDT by the microbial consortium was studied. The consortium showed enhanced degradation with yeast extract at a level of 1%.

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