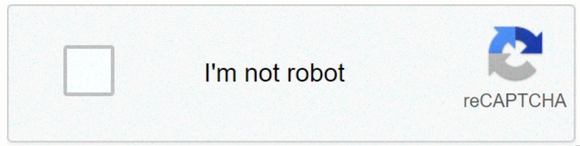

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Dusto chem h

Go to basic content For other purposes, see <a0><a1> Switch to primary content </a1><a2>< Organochloride known for its insecticidal properties DDT Names Preferred IUPAC name 1-chloro-4-[2,2,2-trichloro-1-(4-chlorophenyl)ethyl]benzene Identifiers CAS Number 50-29-3 Y 3D model (JSmol) Interactive image ChEBI CHEBI:16130 Y ChEMBL ChEMBL416898 Y ChemSpider 2928 Y ECHA InfoCard 100.000.023 KEGG D07367 Y PubChem CID 3036 UNII CIW5S16655 Y CompTox Dashboard (EPA) DTXSID4020375 InChI InChI=1S/C14H9Cl5/c15-11-5-1-9(2-6-11)13(14(17,18)19)10-3-7-12(16)8-4-10/h1-8,13H YKey: YVGGHNCTFXOJCH-UHFFFAOYSA-N YInChI=1/C14H9Cl5/c15-11-5-1-9(2-6-11)13(14(17,18)19)10-3-7-12(16)8-4-10/h1-8,13HKey: YVGGHNCTFXOJCH-UHFFFAOYAJ SMILES Clc1ccc(cc1)C(c2ccc(Cl)cc2)C(Cl)(Cl)Cl Properties Chemical formula C14H9Cl5 Molar mass 354.48 g·mol−1 Density 0.99 g/cm3 Melting point 108.5 °C (227.3 °F; 381.6 K) Boiling point 260 °C (500 °F; 533 K) (decomposes) Solubility in water 25 µg/L (25 °C)[1] Hazards Main hazards Toxic , hazardous to the environment, probably carcinogenic GHS pictograms GHS Signal word Danger GHS dangerous statements H301, H351, H372, H410 GHS precautionary statements P201, P202, P260, P264, P270, P273, P281, P301+310, P308+313, P314, P321, P330, P391, P405, P501 NFPA 704 (fire diamond) 2 2 0 Flash point 72–77 °C; 162–171 °F; 345–350 K [3] Lethal dose or concentration (LD, LC): LD50 (median dose) 113–800 mg/kg (rat, oral)[1] 250 mg/kg (rabbit, oral)135 mg/kg (mouse, orally)150 mg/kg (guinea pig, oral)[2] NIOSH (LIMITATION OF U.S. HEALTH EFFECTS):[4] PEL (permissible) TWA 1 mg/m3 [skin] REL (Recommended) Ca TWA 0.5 mg/m3 IDLH (Immediate Hazard) 500 mg/m3 Except as otherwise stated, the data is provided for materials in standard condition (at 25 °C [77 °F], 100 kPa). Y check (what is YN ?) Infobox refers to Dichlorodifenyltrichloretan, commonly known as DDT, is colorless, tasteless and almost odorless of the crystalline chemical compound,[5] organochlor. Originally developed as an insecticide, it became notorious for its environmental impact. DDT was first synthesized in 1874 by Austrian chemist Otmar Seider. The insecticide action of DDT was discovered by Swiss chemist Paul Hermann Mueller in 1939. DDT was used in the second half of World War II to limit the spread of diseases born to insects, malaria and tif among civilians and troops. Mueller 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. By October 1945, DDT was available for public sale in the United States. While it has been boosted by the government and industry for use as an agricultural and household pesticide, there have also been concerns about its use from the start. [7] Opposition to DDT was focused on publication Rachel Carson's Silent Spring 1962. It talked about influences correlated with the utily use of DDT in the United States agriculture, and this calls into question the logic of broadcasting potentially hazardous chemicals into the environment with little preliminary investigation into their environmental and health impacts. The book cites claims that DDT and other pesticides caused cancer and that their agricultural use posed a threat to wildlife, particularly birds. Even though Carson never directly called for an outright ban on the use of DDT, its publication was a semifinal event for the environmental movement and led to much public outrage that ultimately led, in 1972, to a ban on agricultural use of DDT in the United States. [8] The worldwide ban on agricultural use has been formalized under the Stockholm Convention on Permanent Organic Pollutants, which has been in force since 2004. DDT still has limited use of vector control of the disease due to its effectiveness in killing mosquitoes and thus reducing malaria infections, but this use is controversial due to environmental and health problems. [9] Along with the passage of the Endangered Species Act, the United States' ban on DDT is a major factor in the return of the bald eagle (U.S. national bird) and the peregrine falcon from near extinction in the contiguous United States. [11] The properties and chemistry of DDT are similar in structure to insecticide metoxychlor and dicophol acaricide. It is very hydrophobic and almost insolvable in water, but has good solubility in most organic solvents, fats and oils. DDT does not occur naturally and is synthesized by successive Friedel-Crafts reactions between chlorine (CCl3CHO) and two equivalents of chlorobenzene (C6H5Cl), in the presence of an acidic catalyst. DDT traded under trade names including Anofex, Cezarex, Chlorophenothane, Dicophane, Dinocide, Gesarol, Guesapon, Guesarol, Gyron, Ixodex, Neocid, Neocidol and Zerdane; INN is clofenotan. [5] Commercial DDT isomers and related compounds are a mixture of several closely related compounds. Due to the nature of the chemical reaction used to synthesize DDT, several combinations of ortho and para-ateristic patterns are formed. The main component (77%) is the desired p, p' isomer. Isometric impurity is also present in significant volumes (15%). Dichlorodifenichloretilen (DDE) and dichlorodifenichloretan (DDD) make up the balance of impurities in commercial samples. DDE and DDD are also major metabolites and products of ecological decay. [5] DDT, DDE, and DDD are sometimes collectively called DDX. [13] Components of commercial DDT p,p'-DDT(desired connection) o, p'-DDT(isometric impurities) p, p'-DDE(impurities) Production and use of DDT has been formulated in several forms, including solutions in xylene or oil distillates, concentrates, water powders, water-ing perunin powders. Powders. aerosols, smoke candles and charges for fumes and lotons. From 1950 to 1980, DDT was widely used in agriculture — more than 40,000 tons annually worldwide[15] and an estimated total of 1.8 million tons have been produced worldwide since the 1940s. [1] In the United States, it was manufactured by about 15 companies, including Monsanto, Ciba, Montrose Chemical Company, Pennwalt,[17] and Velsicol Chemical Corporation. [18] Production peaked in 1963 at 82,000 tonnes per year. [5] More than 600,000 tons (1.35 billion pounds) were applied in the United States before the 1972 ban. Usage peaked in 1959 at around 36,000 tonnes. In 2009, 3,314 tons were produced to control malaria and visceral leishmaniasis. India is the only country that is still producing DDT, and is the largest consumer. [20] China ceased production in 2007. The mechanism of action of insecticide In insects, DDT opens sodium ion channels in neurons, causing them to fire spontaneously, leading to cramps and possible death. Insects with certain mutations in the sodium canal gene are resistant to DDT and similar insecticides. [22] Resistance to DDT is also provided by increased regulation of genes expressing P450 cytochrome in some insect species,[23] as a larger number of some enzymes in this group accelerate the metabolism of the toxin into inactive metabolites. (The same enzyme family is also regulated in mammals, for example in response to ethanol consumption.) Genominous studies in the drosophila melanogaster genetic body model showed that high levels of DDT resistance are polygenic, involving multiple resistance mechanisms. [24] The history of commercial concentrate of products containing 50% DDT, circa 1960s Commercial product (Powder Box, 50 g), containing 10% DDT; A suicide. Ciba Heygi DDT; Destroys parasites such as fleas, lice, ants, beds, cockroaches, flies, etc. The neocid is sprinkled with a cache of vermin and places where there are insects and their places of passage. Leave the powder in place for as long as possible. Destroy human parasites and its dwellings. Death is not instantaneous, it inevitably follows sooner or later. French production; harmless to humans and warm-blooded animals confident and lasting effect. No smell. External Audio Episode 207: DDT, DDT Institute for The History of Science was first synthesized in 1874 by Otmar Seider under Adolf von Bayer. [25] He was described in 1929 in V. Baush's dissertation and in two subsequent editions in 1930. [27] [28] The properties of the insecticide multiple chlorinated alifatic or fat-aromatic alcohols with at least one group of trichlorometans were described in Wolfgang von Leithold's patent in 1934. [29] The insecticide properties of DDT were not, however, discovered until 1939 by Swiss scientist Paul Hermann Mueller, who was awarded the 1948 Nobel Prize in Physiology A cure for his efforts. [6] The use in the 1940s and 1950s of the DDT spraying aircraft over Baker County, Oregon as part of the Spruce Worm Management Project, the 1955 DDT spray magazine in Bosa (Sardinia) DDT is the most prominent of several chlorine-containing pesticides used in the 1940s and 1950s. With pyrethrum in short circulation, DDT was widely used during World War II by the Allies to control the vectors of tif insects - almost eliminating the disease in many parts of Europe. In the South Pacific, it was sprayed airborne to control malaria and dengue with impressive effects. While the chemical and insecticide properties of DDT were important factors in these victories, advances in applied engineering combined with a competent organization and sufficient manpower were also crucial to the success of these programs. In 1945, DDT was available to farmers as an agricultural insecticide[5] and played a role in the temporary elimination of malaria in Europe and North America. [9] [31] In 1955, the World Health Organization began a program to eradicate malaria in low- and moderate transmission countries around the world, relying mainly on DDT to fight mosquitoes and rapid diagnosis and treatment to reduce transmission. [33] The program eliminated the disease in North America, Europe, the former Soviet Union, as well as in Taiwan, much of the Caribbean, the Balkans, parts of north Africa, the northern region of Australia, and the great south Pacific scrolls,[35] and dramatically reduced mortality in Sri Lanka and India. However, failure to support the program, increasing mosquito tolerance for DDT and increasing parasite tolerance have led to a resurgence. In many areas, early successes are partially or completely reversed, and in some cases the pace of transmission has increased. [37] The program succeeded in eliminating malaria only in areas with high socioeconomic status, well-organized healthcare systems, and relatively less intense or seasonal malaria transmission. DDT was less efficient in tropical regions due to the continuous life cycle of mosquitoes and poor infrastructure. It was not used in sub-Saharan Africa at all because of these perceived difficulties. Mortality in this area has never decreased to the same dramatic extent, and now constitute the bulk of malaria deaths worldwide, especially after the resurgence of the disease as a result of resistance to drug treatment and the spread of the deadly malaria variant caused by Plasmodium falciparum. Eradication was abandoned in 1969 and attention was instead focused on controlling and treating the disease. Spraying programs (especially using DDT) have been curtailed due to concerns about safety and environmental impact, as well as problems in administrative, and financial realization. [37] Efforts have shifted from spraying to using bedding soaked in insecticides and and Activities. [34] [38] The United States Prohibition Until October 1945, DDT was available for public sale in the United States, used as an agricultural pesticide and as a household insecticide. Even though its use has contributed to the government and the agricultural industry, U.S. scientists such as FDA pharmacologist Herbert O. Culvery expressed concern about possible dangers associated with DDT back in 1944. [39] In 1947, Dr. Bradbury Robinson, a physician and nutritionist practicing in St. Louis, Michigan, warned of the dangers of using the pesticide DDT in agriculture. DDT was researched and manufactured in St. Louis by the Michigan Chemical Corporation, later acquired by Velsicol Chemical Corporation,[40] and became an important part of the local economy. [41] Referring to research done by Michigan State University[42] in 1946, Robinson, past president of the local Conservation Club,[43] predicted that: ... perhaps the biggest danger from the D.D.T. is that its widespread use in agricultural areas is likely to upset natural residues by not only killing beneficial insects in large numbers, but also resulting in the deaths of fish, birds and other forms of wild life or by feeding insects killed by D.D.T. or directly by eating poison. [44] As its production and use increased, public resonance was mixed. At the same time that DDT was affected as part of the world of tomorrow, concerns were expressed about its potential to kill harmless and beneficial insects (especially pollinators), birds, fish and eventually humans. The issue of toxicity was complex, partly because the effects of DDT ranged from species to species, and partly because successive impacts could accumulate, causing damage comparable to large doses. A number of states have tried to regulate DDT. [7] In the 1950s, the federal government began tightening regulations governing its use. [19] These events received little attention. Women like Dorothy Colson and Mamie Ella Pleller of Claxton, Georgia, gathered evidence about the effects of DDT and wrote to the Georgia Department of Public Health, the National Health Council in New York and other organizations. In 1957, The New York Times reported on a failed fight to limit the use of DDT in Nassau County, New York, and the issue caught the attention of popular naturalist author Rachel Carson. William Sean, editor of The New Yorker, encouraged her to write a piece on the subject that grew into her 1962 book Silent Spring. The book argued that pesticides including DDT poison both wildlife and the environment and endanger human health. Silent Spring was a best seller, and public reaction to it launched a modern environmental movement in the United States. A year after it has emerged President John F. Kennedy has ordered his Scientific Advisory Committee to investigate Carson's claims. V.O. V.O. The report add to Rachel Carson's rather thorough vindication of silent spring dissertations, according to the journal Science,[46] and recommended phased resistant toxic pesticides. [47] In 1965, the U.S. military removed DDT from the military supply system due in part to the development of body lice resistance to DDT; he was replaced by Lindan. [48] DDT became the main target of the growing antichemical and anti-pesticide movement, and in 1967 a group of scientists and lawyers established the Environmental Defense Fund (EDF) for the specific purpose of imposing a ban on DDT. Victor Jannacone, Charles Urster, Art Cooley and others in the group witnessed bird killings or declining bird populations and suspected that DDT was the cause. In its campaign against chemicals, EDF petitioned for the government's ban and filed lawsuits. [49] Around this time, toxicologist David Pickall measured DDE levels in peregrine falcon eggs and California condors and found that elevated levels matched thinner shells. [50] In response to the EDF lawsuit, the U.S. District Court of Appeals in 1971 ordered the EPA to begin a de-escalation procedure for DDT. After an initial six-month review process, William Ruckelshaus, the agency's first administrator, rejected the immediate suspension of DDT registration, citing research by internal EPA employees, saying DDT posed no imminent danger. [19] However, these findings were criticized because they were performed largely by economic entomologists inherited from the United States Department of Agriculture, who, on their basis, were biased towards agribusiness and understated concerns about human health and wildlife. Thus, the decision created controversy. [30] The EPA held seven months of hearings in 1971–1972 by scientists who testified for and against DDT. In the summer of 1972, Ruckelshaus announced the abolition of most applications of DDT - an exemption from the use of public health under some conditions. [19] Again, it caused controversy. Immediately after the announcement, both EDF and DDT manufacturers filed a lawsuit against the EPA. Many in the agricultural community were concerned that food production would be severely impacted, while pesticide advocates warned of increased breakthroughs in insect-borne diseases and questioned the accuracy of providing animals with high amounts of pesticides for cancer potential. The industry sought to overturn the ban, while EDF wanted a comprehensive ban. The cases were consolidated, and in 1973, the United States Court of Appeals for the District of Columbia Circuit ruled that the EPA acted appropriately in banning DDT. [19] In the late 1970s, the EPA also began banning organoquines, pesticides that were chemically similar to DDT. They included aldrin, dildrin, chlordan, heptachlor, texafen and mirex. [51] Some of them DDT continued under public health exemption. For example, in June 1979, the California Department of Public Health was allowed to use DDT to suppress flea vectors of bubonic plague. [52] DDT continued to be produced in the United States for foreign markets until 1985, when more than 300 tons were exported. [1] International restrictions on use In the 1970s and 1980s, agricultural use was banned in most developed countries, starting in Hungary in 1968,[53] followed by Norway and Sweden in 1970, West Germany and the United States in 1972, but not in the United Kingdom until 1984. By 1991, total bans, including those for disease control, could be in effect in at least 26 countries; For example, Cuba in 1970, the United States in the 1980s, Singapore in 1984, Chile in 1985 and the Republic of Korea in 1986. [54] The Stockholm Convention on Permanent Organic Pollutants, which came into force in 2004, absorbed a global ban on several sustainable organic pollutants, and restricted the use of DDT for vector control. The Convention was ratified by more than 170 countries. Recognizing that complete elimination in many malaria-prone countries is being unenviable for missing affordable/effective alternatives, the convention exempts the use of public health as part of World Health Organization (WHO) guidelines from prohibition. Resolution 60.18 of the World Health Assembly commits the WHO to the goal of the Stockholm Convention to reduce and ultimately eliminate DDT. [56] The International Malaria Foundation states: The outcome of the treaty is perhaps better than the status quo going into negotiations. For the first time now, there is an insecticide that is limited only to vector control, meaning that the choice of drug-resistant mosquitoes will be slower than before. Despite the worldwide ban, agricultural use continued in India,[58] North Korea and possibly elsewhere. [20] As of 2013, an estimated 3,000 to 4,000 tons of DDT were produced to control disease vectors, including 2,786 tons in India. DDT applies to the inner walls of houses to kill or repel mosquitoes. This intervention, called residual indoor spraying (IRS), greatly reduces environmental damage. It also reduces the frequency of DDT resistance. By comparison, processing 40 hectares of cotton during a typical veal period in the U.S. requires the same amount of chemicals to handle approximately 1,700 homes. [61] Degrading the environmental impact of DDT to form DDE (by eliminating HCl, left) and DDD (by reproductive dechlorination, right) DDT is a persistent organic pollutant that is easily adsorbed to soils and sediments that can act as both sinks and long-term sources of exposure affecting organisms. [14] Depending on the conditions half-life can range from 22 days to 30 years. Ways of loss and degradation include runoff, volatility, photolysis and aerobic and anaerobic biodegradations. Due to hydrophobic properties, the [62] DDT ecosystems and its metabolites are absorbed by aquatic organisms and adsorbed to hinged particles, leaving little DDT dissolved in water (however, its semi-muscular life in aquatic environments is listed by the National Center for Information on Pesticides as 150 years old. Its fission and metabolite products, DDE and DDD, are also resistant and have similar chemical and physical properties. DDT and the products of its breakdown are transported from warmer areas to the Arctic by the phenomenon of global distillation, where they then accumulate in the region's food web. [63] Medical researchers in 1974 found a measurable and significant difference in the presence of DDT in human milk between mothers who lived in New Brunswick and mothers who lived in Nova Scotia, possibly due to the greater use of insecticide sprays in the past. [64] Due to its lipophilic properties, DDT can bio-accumulate, especially in birds of prey. DDT is toxic to a wide range of living organisms including marine animals such as crayfish, daphnides, sea shrimp and many fish species. DDT, DDE and DDD increase through the food chain, with apex predators such as rapist birds concentrate more chemicals than other animals in the same environment. They are stored mainly in fat in the body. DDT and DDE are resistant to metabolism; in humans their half-wine is 6 and up to 10 years respectively. In the United States, these chemicals were found in nearly all human blood samples tested by the Centers for Disease Control in 2005, though their levels dropped dramatically because most applications were banned. Food consumption is estimated to have declined,[66] although FDA food tests typically detect it. Despite being banned for years, in 2018 research showed that DDT remains are still present on European soils and Spanish rivers. [68] The thinning of the eggshell Chemical and its DDE and DDD fission products caused thinning of the eggshell, with populations declining in several North American and European species of birds of prey. [1] [70] [11] [71] [72] [73] The effect was first finally proven on Bellow Island in Lake Michigan during research funded by the University of Michigan on american osver-1960s seagulls. [74] Thinning of the DDE-related eggshell is considered the main cause of the decline of the bald eagle,[11] a brown pelican,[75] peregrine falcon and osprey. However, birds vary in sensitivity to these chemicals, with birds of prey, waterducts and birds songs more susceptible than chickens and related species. [1] Even in 2010, California condors feeding on sea lions in Big Sur, which in turn feed on the Palos Verdes shelf area of the Montrose chemical super fund, exhibited long-lasting subtle shell problems,[76][77] although DDT's role in the decline of the California condor [73] [72] The biological thinnery mechanism is not entirely clear, but DDE appears to be larger than DDT,[1] and strong evidence suggests that p,p'-DDE inhibits calcium ATPase in the membrane of the gland shell and reduces the transportation of calcium carbonate from the blood into the eggshell of the gland. This leads to a decrease in dose-dependent thickness. [1] [78] [79] [71] Other evidence suggests that o,p'-DDT disrupts the development of the female reproductive tract, later impairing the quality of the eggshell. [80] Several mechanisms can be at work, or different mechanisms can work in different kinds. [1] Human health American soldier demonstrates equipment for spraying hands with DDT. DDT was used to control the spread of tifu-bearing lice. Spraying hospital beds with DDT, Paigc Hospital Ziguinshor, 1973 Biomagnification is a build-up of toxins in the food chain. The concentration of DDT is in parts per million. As trophic levels rise in the food chain, the amount of toxic build-up is also increasing. X represent the amount of toxic build-up of accumulation as trophic levels rise. Toxins accumulate in the body's tissues and fats. Predators accumulate higher toxins than prey. DDT is an endocrine destroyer. [81] It is considered human carcinogen, although most studies suggest that it is not directly genotoxic. [83] [84] DDE acts as a weak androgen receptor antagonist, but not as an estrogen. [86] p,p'-DDT, the main component of DDT, has little or no androgenic or estrogenic activity. [87] A minor o,p'-DDT component has weak estrogenic activity. Acute DDT toxicity is classified by the WHO as moderately toxic by the U.S. National Toxicology Program (NTP) and moderately dangerous based on rat mouth LD50 of 113 mg/kg. [88] Indirect influence is considered relatively non-toxic to humans. [89] Chronic toxicity primarily due to DDT's tendency to build up in areas of the lipid-high body, chronic exposure can affect reproductive capabilities and the embryo or fetus. [89] A review of an article in The Lancet states that studies have shown that exposure to DDT in the amount that would be required when controlling malaria can lead to premature birth and early displacement ... toxicology data show endocrine destructive properties; human data also indicates possible impaired sperm quality, menstruation, gestational length, and lactation duration. [38] Other studies document reduces sperm quality among men with high exposure (usually from internal spray residue). [90] Studies are inconsistent as to whether high levels of DDT or DDE increase before pregnancy. In mothers with high serum DDE, daughters may have up to a 32% increase in the likelihood of conception, but elevated DDT was associated with a 16% decrease in a single study. [91] Indirect exposure to mothers through employees directly in contact with DDT is associated with an increase in [89] Other studies have shown that DDT or DDE interfere with proper function of the thyroid gland during pregnancy and childhood. [66] [92] Mothers with high levels of DDT circulating in their blood during pregnancy were found to be more likely to give birth to children who would develop autism. [93] [94] Carcinogenicity In 2015, the International Agency for Research on Cancer classified DDT as group 2A, probably carcinogenic to humans. [95] Preliminary assessments of the U.S. National Toxicology Program classified it as reasonably expected as a carcinogen and the EPA classified DDT, DDE and DDD as probable class B2 carcinogens; these estimates were mainly based on animal studies. [1] [38] A 2005 Lancet review stated that professional exposure to DDT was associated with an increased risk of pancreatic cancer in 2 case control studies, but another study showed a lack of association of DDE dos-effects. The results of a possible link with liver cancer and biliary cancer conflict: workers who have not had direct professional contact with DDT have shown an increased risk. White men had an increased risk, but not white women or black men. Results on connectivity with multiple moths, prostate and testicular cancer, endometrial cancer, and colorectal cancer were to no avail or generally do not support association. [38] A review of liver cancer research in 2017 concluded that orchanochlor pesticides, including DDT, can increase the risk of hepatocelic carcinoma. [96] A 2009 review co-authored by individuals associated with DDT reached widely similar conclusions, with an unequivocal link to testicular cancer. Case-control studies have not maintained a link to leukemia or lymphoma. [66] Breast cancer Questions about whether DDT or DDE risk factors for breast cancer were not definitively answered. Several metaanalysis observational studies have concluded that there is no overall link between DDT exposure and breast cancer risk. [97] [98] The U.S. Institute of Medicine reviewed breast cancer association data with DDT contact in 2012 and concluded that the pathogen could neither be proven nor provoked. [99] A 2007 case control study using archival blood samples found that the risk of breast cancer was increased by 5 times among women born before 1931 and who had high serum DDT levels in 1963. Arguing that the use of DDT was most informally widespread in 1945 and peaked around 1950, they concluded that the age of 14-20 years was a critical period in which the impact of DDT leads to increased risk. This study, which suggests the link between DDT contact and breast cancer, which won't pick up most studies, has received variable comments in third-party reviews. One review suggested that previous studies that measured exposure in older women may have critical period. [66] [100] The National Toxicology Program notes that while studies found no link between DDT exposure and breast cancer that positive associations were seen in several studies among women with higher levels of exposure and among certain subgroups of women. [84] A

2015 case control study identified a link (coefficient of 3.4) between exposure to in-utero (estimated archival maternal blood samples) and breast cancer diagnosis in daughters. The findings support the classification of DDT as an endocrine disruptor, breast cancer predictor, and high-risk marker. [101] Malaria malaria control remains a major public health challenge in many countries. In 2015, there were 214 million reported cases of malaria worldwide, leading to an estimated 438,000 deaths, 90% of which occurred in Africa. [102] DDT is one of many tools to fight the disease. Its use in this context was so-called by everyone, from miracle weapons[i.e.] as Kryptonite to mosquitoes, [103] to toxic colonialism. [104] Prior to DDT, the elimination of mosquito breeding sites by drainage or poisoning by Paris green or pyrethrum was sometimes successful. In some parts of the world with rising living standards, eliminating malaria has often been a side benefit of introducing window screens and improving sanitation. [35] A variety of typically simultaneous interventions are best practice. These include anti-marilarary drugs for the prevention or treatment of infection; improving public health infrastructure for diagnosis, sequestration and treatment of infected individuals; bed nets and other methods designed to ensure that mosquitoes do not bite in humans; Vector control strategies[105] such as insecticide larvae, environmental controls such as draining mosquito breeding sites or introducing fish to eat larvae and internal spraying residues (IRS) by insecticides, possibly including DDT. The IRS provides for the treatment of internal walls and ceilings with insecticides. It is particularly effective against mosquitoes, as many species rest on the wall indoors before or after feeding. DDT is one of 12 WHO-approved IRS insecticides. [34] The WHO's anti-malarial campaign of the 1950s and 1960s relied heavily on DDT, and the results were promising, albeit temporary in developing countries. Experts have linked the recovery of malaria to several 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 insecticides has exacerbated the situation. [20] [106] Resistance was largely fueled by unlimited agricultural use. Resistance and harm to both humans and the environment have led many governments to curtail the use of DDT in vector control and agriculture. [37] In 2006, WHO long-standing policy against DDT recommending that it be used as an indoor pesticide in regions where malaria is As of 2008, only 12 countries have used DDT, including India and some southern African states,[105] although the number was expected to rise. Initial efficacy When it was introduced in World War II, DDT was effective in reducing malaria morbidity and mortality. [30] The WHO's anti-malarial campaign, consisting mainly of DDT spraying and rapid treatment and diagnosis to disrupt the transmission cycle, was initially also successful. In Sri Lanka, for example, the program reduced cases from about one million a year before being sprayed to just 18 in 1963[108][109] and 29 in 1964. After that, the program was stopped to save money and malaria rebounded to 600,000 cases in 1968 and the first quarter of 1969. The country regained control of DDT vectors, but mosquitoes evolved resistance in the interim, presumably due to continued agricultural use. [110] The program switched to malathion, but despite initial successes, malaria continued its revival in the 1980s. After arata Kochi was appointed head of its anti-malarial unit, the WHO policy shifted from the IRS recommendation only in areas of seasonal or episodic malaria transmission, to promoting it in areas of continuous, intensive transmission. [111] Who reaffirmed its commitment to phase out DDT, seeking to achieve a 30% reduction in DDT applications worldwide by 2014 and its overall phased output by the early 2020s, if not before, while combating malaria. Who plans to introduce alternatives to DDT to achieve this goal. [112] South Africa continues to use DDT in accordance with WHO guidelines. In 1996, the country switched to alternative insecticides and the incidence of malaria increased dramatically. Returning to DDT and administering new drugs brought malaria under control. [113] Malaria cases increased in South America after countries on this continent stopped using DDT. Research data showed a strong negative link between the remnants of DDT home spraying and malaria. In a study from 1993 to 1995, Ecuador increased DDT use and achieved a 61% reduction in malaria rates, while each of the other countries that gradually reduced DDT use had a significant increase. [61] [114] [115] Mosquito resistance In some areas, resistance reduces the effectiveness of DDT. WHO guidelines require that a lack of resistance be confirmed before using the chemical. [116] Resistance is largely due to agricultural use, in much larger quantities than is required for disease prevention. Resistance was noted at the beginning of the campaign spray. Paul Russell, former head of the allied anti-marion campaign, observed in 1956 that resistance emerged six to seven years later. was discovered in Sri Lanka, Pakistan, Turkey and Central America, and has been largely replaced by organophosphate or or insecticides, such as malaise or bendiocairb. [117] In many parts of India, DDT is ineffective. [118] Agricultural use was banned in 1989, and its anti-marijuana use decreased. Urban use is over. [119] One study concluded that DDT is still a viable insecticide in internal residual spraying due to its effectiveness in well-controlled spray work and a high arousal-repellence factor. Studies of malaria-vector mosquitoes in KwaZulu-Natal province, South Africa found susceptibility to 4% of DDT (WHO susceptibility standard), in 63% of samples, compared to an average of 87% in the same species caught in the open. The authors concluded that the Search for DDT resilience in the An. arabiensis vector, not far from the area where we previously reported pyretroid resistance in the An. funestus Giles vector, shows the urgent need to develop an insecticide resistance management strategy for South African malaria control programs. DDT can still be effective against resistant mosquitoes,[122] and avoiding DDT sprayers by mosquitoes is an added benefit of the chemical. [120] For example, a 2007 study reported that drug-resistant mosquitoes avoided treated huts. The researchers argued that DDT was the best pesticide to use in the IRS (even though it did not have the greatest protection against mosquitoes from three test chemicals) because other pesticides worked primarily by killing or irritating mosquitoes - encouraging the development of resistance. [122] Others argue that avoidance behavior slows eradication. Unlike other insecticides, such as pyretroids, DDT requires lasting exposure to the accumulation of the lethal dose; however, its irritation property reduces contact periods. For these reasons, when comparisons were made, better malaria control was generally achieved by pyretroids than with DDT. [117] In India, widespread outdoor sleep duties and night duties, which means that the excitable effect of DDT, often reported useful in other countries, actually contributes to outdoor transmission. [124] Concerns of Residents Main Article: Internal Residual Spraying § Opposition of Residents to the IRS IRS is effective if at least 80% of homes and sheds are sprayed in a residential building. [116] Reducing coverage could jeopardize the effectiveness of the program. Many residents resist spraying DDT, objecting to lingering odor, stains on walls and potential exacerbation of problems with other insect pests. [117] [123] Pyrethroid insecticides (e.g., deltametrine and lambda-cygalotyrine) can overcome some of these issues by increasing participation. [117] A 1994 human exposure study found that South Africans living in sawn-off houses have equal, several orders larger than the others. Breast milk from South African mothers contains high levels of DDT and DDE. [66] It is unclear to what extent these levels arise from spraying against leftovers. Evidence suggests that these levels are associated with neurological abnormalities of infants. [117] Most studies of human health effects of DDT have been conducted in developed countries where DDT is not used and exposure is relatively low. [38] [66] Illegal diversion into agriculture is also a concern because it is difficult to prevent and its further use on crops is uncontrolled. For example, the use of DDT is widespread in Indian agriculture,[127] especially mango production[128] and is reportedly used by librarians to protect books. [129] Other examples include Ethiopia, where DDT, designed to control malaria, is reportedly used in coffee production,[130] and Ghana, where it is used for fishing. [131] [132] The remains of crops at levels unacceptable for export were an important factor in bans in several tropical countries. [117] Adding to this problem is a lack of qualified personnel and management. Criticism of restrictions on the use of DDT Several people and groups have argued that restrictions on the use of DDT for public health purposes have caused unnecessary morbidity and mortality from vector diseases, with some allegations of malaria deaths in the range of hundreds of thousands [133] and millions. [134] Robert Guajs of the U.S. National Institutes of Health declared in 2007, The ban on DDT may have killed 20 million children. [135] These arguments were dismissed as outrageous by former WHO scientist Socrates Litcios. [103] May Berenbaum, an entomologist at the University of Illinois, says that blaming environmentalists who oppose DDT for more deaths than Hitler is worse than irresponsible. [103] Most recently, Michael Palmer, a professor of chemistry at the University of Waterloo, noted that DDT is still used to prevent malaria, that its declining use is primarily due to increased production costs, and that in Africa, malaria control efforts were regional or local, not comprehensive. [136] The question is that ... Malaria control experts should ask not what's worse, malaria or DDT?, but rather What are the best tools to deploy to control malaria in a given situation, taking into account ground challenges and needs, efficacy, cost and side effects - both positive and negative - for human health and the environment, as well as the uncertainties associated with all these considerations? Hans Herren & Charles Mbogo[137] Criticism of the DDT ban often specifically references the 1972 United States ban (with the false assumption that it constituted a worldwide ban and prohibited the use of DDT in vector control). References are often to Silent Spring, though Carson has never pushed for a DDT ban. John Clawgin and Tim Lambert wrote: 'The clearest feature of the lawsuit against Carson is with which it can be disproved. [138] Investigative Journalist Adam Sarwana and Others these concepts as myths promoted mainly by Roger Bate of the pro-DDT advocacy group Africa Fighting Malaria (AFM). [139] Alternatives to Insecticides The main article: Internal residual spraying of Oranophosphate and insecticide carbamate, such as malathion and bendiodarb, respectively, cost more than DDT per kilogram and are used at approximately the same dosage. Pyrethropes, such as deltametrin, are also more expensive than DDT, but are used more economically (0.02–0.3 g/m2 vs. 1-2 g/m2), so the net cost of one house for treatment is approximately the same. DDT has one of the longest periods of residues of any IRS insecticide, lasting 6 to 12 months. Pyretroids will remain active for only 4 to 6 months, and organophosphates and carbamats remain active for 2-6 months. In many endemic countries, malaria transmission occurs year-round, meaning that high costs running a spray campaign (including hiring spray operators, procuring insecticides and conducting pre-spray campaigns to encourage people to be home and take intervention) should occur several times a year for these shorter long-lasting insecticides. [141] In 2019, the associated diffluorodifenyltrichlorate (DFDT) compound was described as a potentially more effective and therefore potentially safer alternative to DDT. [142] [143] Nonchemical vector control Before DDT, malaria was successfully eliminated or curtailed in several tropical areas by removing or poisoning mosquito breeding sites and larvae habitats, for example by eliminating standing water. These methods have seen little application in Africa for more than half a century. [144] According to the CDC, such methods are not practical in Africa because Anopheles gambiae, one of the main vectors of malaria in Africa, multiplies in numerous small pools of water that are formed through precipitation ... It is difficult, if not impossible, to predict when and where breeding sites will form, and to find and treat them before the advent of adults. The relative effectiveness of the IRS and other malaria control methods (e.g., bed beds or prompt access to anti-malarial drugs) varies and depends on local conditions. A WHO study published in January 2008 found that the mass spread of mosquito nets treated with insecticides and artemisinin-based drugs reduced malaria deaths in half in malaria-burdened Rwanda and Ethiopia. The IRS with DDT played no important role in reducing mortality in those countries. [146] Vietnam enjoys a 97% reduction in malaria cases and a 97% reduction in deaths after moving in 1991 from a poorly funded DDT-based campaign to a program based on rapid treatment, bed beds and pyretroid insecticide groups. [148] In Mexico, effective and affordable chemical and nonchemical strategies have been so successful that DDT manufacturing plant ceased production due to the lack of A review of fourteen studies in sub-Saharan Africa covering insecticide-treated nets, residual spraying, chemoprophylaxis for children, chemoprophylaxis or periodic treatment of pregnant women, hypothetical vaccine and change of frontline drug treatment, found that decision-making is limited to lack of information on the costs and consequences of many interventions, a small number of economic efficiency analyses, lack of evidence of costs and consequences of packages of measures and problems in generalizing or comparative studies specific use of various methodologies and measures for results. Two performance assessments of residual DDT spraying were not found to provide an accurate assessment of the effectiveness of DDT spraying; estimates may not be good predictors of economic efficiency in current programs. [150] However, a study in Thailand found that the cost of a single case of malaria prevented the spraying of DDT (US$1.87), which is 21% more than the cost per case that prevented lambda-cygalotrin-processed mesh (US$1.54), questioning the assumption that DDT was the most cost-effective measure. Mexico's director of malaria control found similar results, announcing that it was 25% cheaper for Mexico to spray a house with synthetic pyretroids than with DDT. [149] However, another study in South Africa found typically lower costs for spraying DDT than on soaked nets. [152] A more comprehensive approach to measuring the effectiveness of costs or the effectiveness of malaria control not only measures the cost in dollars, but also the number of people rescued, but will also take into account environmental harm and negative effects on human health. One previous study found that it is likely that harm to human health is approaching or exceeding favorable reductions in malaria cases, except perhaps epidemics. This is similar to a previous study on estimated theoretical infant mortality caused by DDT and is subject to criticism, also mentioned earlier. [153] A study in the Solomon Islands found that although impregnated bed nets cannot completely replace DDT spraying without a significant increase in incidence, their use allows for reducing DDT spraying. [154] Comparing four successful anti-malaria programs in Brazil, India, Eritrea and Vietnam disapprove of any strategy, but instead argues: Common success factors included favourable country conditions, a targeted technical approach using a package of effective tools, data-driven decision-making, active leadership at all levels of government, community engagement, decentralized implementation and finance control, skilled technical and management capabilities at national and subnational levels. , practical technical and software support with partner agencies, as well as sufficient and flexible funding. [155] DDT DDT mosquitoes may be susceptible to pyretroids in some countries. However, pyretidist resistance in Anopheles mosquitoes is on the rise with persistent mosquitoes found in several countries. [156] See also DDT in New Zealand's Mickey Slim, an alleged cocktail that combined gin with a pinch of DDT. 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Australian Broadcasting Commission. 31-May-2008 DDT in popular culture Examples of DDT advertising in the US. 2-Nov-2010 In 2008, #230 1000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000000 Retrieved .php 989676661 Page 2 3-MCPD Names IUPAC Name 3-Chloropropan-1,2-diol Other names 3-Monochloroppan-1,2-diol Other names 3-Monochloroppan-1,2-diol; α-chlorohydrin; Glycerin α-monochlorohydrine; Chlorodeoxyglycerin; 3-Chloro-1,2-propanediol Identifiers CAS Number 96-24-2 Y 3D model (JSmol) Interactive image ChEBI CHEBI:18721 Y ChemSpider 7018 Y ECHA InfoCard 100.002.267 EC Number 202-492-4 KEGG C18676 N PubChem CID 7290 UNII QGS78A3T6P Y CompTox Dashboard (EPA) DTXSID4020664 InChI InChI=1S/C3H7ClO2/c4-1-3(6)2-5/h3,5-6H,1-2H2 YKey: SSZWWUDQMAHNAQ-UHFFFAOYSA-N YInChI=1/C3H7ClO2/c4-1-3(6)2-5/h3,5-6H,1-2H2Key: SSZWWUDQMAHNAQ-UHFFFAOYAR SMILES ClCC(O)CO Properties Chemical formula $C_3H_7ClO_2$ Molar mass 110.54 g·mol−1 Appearance Viscous, colorless liquid Density 1.32 g·cm−3 Melting point −40 °C (−40 °F; 233 K) Boiling point 213 °C (415 °F; 486 K) Hazards Safety data sheet External MSDS R-phrases (outdated) R26/27/28-R36/37/38 S-phrases (outdated) S24-S45 Except where otherwise noted, data are given for materials in their standard state (at 25 °C [77 °F] , 100 kPa). N check (what is YN ?) Infobox link 3-MCPD (3-monochloropropan-1,2-diol or 3-chloropropan-1,2-diol) is an organic chemical compound with the formula HOCH2CH (OH)CH2Cl. This is a versatile multifunctional building block. [1] The compound attracted attention as the most common member of chemical food pollutants known as chloropropanolys. It is suspected to be carcinogenic in humans. It is produced in foods treated at high hydrochloric acid temperatures to accelerate protein hydrolysis. As a byproduct of this process, chloride can react with glycerin spine lipids to produce 3-MCPD. 3-MCPD can also occur in foods that have been in contact with materials containing moisture strength based on epichlorohydrine used in the production of some tea bags and sausage coats. In 2009, 3-MCPD was found in some East Asian and Southeast Asian sauces such as oyster sauce, Hoisin sauce and soy sauce. Hydrochloric acid use is much faster than traditional slow fermentation. A 2013 report by the European Food Safety Authority lists margarine, vegetable oils (excluding walnut oil), stored meat, bread and fine baked goods as the main sources in Europe. [5] 3-MCPD can also be found in many paper products treated with polyamidoamine-epichlorohydrine of moisture-strong resins. [6] Absorption and toxicity by the International Agency for Research on Cancer classifies 3-MCPD as group 2B, possibly carcinogenic to humans. [7] 3-MCPD is carcinogenic in rodents due to nongenotoxic mechanism. [8] It is capable of crossing the blood-yayetra barrier and brain barrier. [9] Oral LD50 with 3-chloro-1,2-propandiol is 152 mg/kg of body weight in rats. [10] 3-MCPD also has male birth control effects [10][11] and can be used as a rat chemosterylant. [12] Legal restrictions Joint Food Standards Australia New Zealand (FSANZ) set a limit of 3-MCPD in soy sauce of 0.02 mg/kg in accordance with European Commission standards, which came into force in the EU in April 2002. History In 2000, a survey of soy sauces and similar products available in the UK was conducted by the Joint Ministry of Agriculture, Fisheries and Food/Health Of the Food and Standards Safety Group (JFSSG) and reported that more than half of the samples collected from outlets contained different levels of 3-MCPD. [13] In 2001, the United Kingdom Food Standards Agency (FSA) found in tests various oyster sauces and soy sauces that 22% of the samples contained 3-MCPD at levels significantly higher than those considered safe by the European Union. About two-thirds of these samples also contained a second chloropropanol called 1.3-dichloroppan-2-ol (1.3-DCP), which experts advise should not attend any levels in food. Both chemicals have the potential to cause cancer, and the agency recommended that the affected products be taken off the shelves and avoided. [14] In 2001, FSA and Food Standards Australia New Zealand (FSANZ) carved out brands and products imported from Thailand, China, Hong Kong and Taiwan. Brands named in the British warning include Golden Mountain, King Imperial, Pearl River Bridge, Golden Mark, Kimlan, Golden Swan, Sinsin, Tung Chun and Wanjasham Soy Sauce. Also involved were Knorr soy sauce, as well as Uni-President Enterprises Corporation creamy soy sauce from Taiwan, Silver Swan soy sauce from the Philippines, Ta Tun soy bean sauce from Taiwan, Tau Wee yau seasoning sauce and sauce from beans from Vietnam, soy sauce Zu Miao Fo Shan and mushroom soy sauce from China and Golden Mountain and chicken marinade Li Kum Ki. [16] [17] Between 2002 and 2004, a relatively high level of and other chloropropanils have been found in soy sauce and other foods in China. In 2007, in Vietnam, 3-MCPD was found in toxic levels. In 2004, the City Institute of Hygiene and Public Health HCM found 33 of 41 samples of soy sauce with high rates of 3-MCPD, including six samples from up to 11,000 to 18,000 times over 3-GDC, Thanh Nien Daily newspaper commented: Health agencies knew that Vietnamese soy sauce, the country's second most popular sauce after fish sauce, has been full of cancer agents since at least 2001. In March 2008, in Australia, carcinogens were found in soy sauces, and Australians were advised to avoid soy sauce. In November 2008, the UK Food Standards Agency reported a wide range of household food items from sliced bread to crackers, beef and cheese from 3-MCPD above safe limits. Relatively high levels of the chemical have been found in popular brands such as Mother's Pride, Jacobs crackers, John West, Kraft Dairylea and McVitie's Kracwheatka. The same study also found relatively high levels at a number of the supermarket's own brands, including Tesco-char grill butchers, Sainsbury's Hot 'n Spicy Chicken Drumsticks and digestive biscuits from Asda. The highest level of 3-MCPD found in the product of non-soy sauce, crackers, was 134 micrograms per kg. The highest level of 3-MCPD found in soy sauce was 93,000 micrograms per kg, 700 times higher. The legal limit for 3-MCPD coming in next year[when?] will be 20 micrograms per kg, but the safety guidelines for daily consumption are 120 micrograms for 60kg of a person a day. [citation required] In 2016, 3-MCPD was reported in selected paper products (coffee filters, tea bags, disposable paper cups for hot drinks, milk cards, paper towels) sold on the Canadian and German market, and the transfer of 3-MCPD from these products to beverages was investigated. Exposure to 3-MCPD from packaging material is likely to account for only a small percentage of the overall dietary exposure compared to the consumption of processed oils/fats containing the equivalent of 3-MCPD (in the form of fatty acid esters), which are often present at levels around 0.2-2 micrograms/g. Reference ^ Fernandez-Megia, Eduardo; Correa, Huang; Rodriguez-Meisozo, Irene; The year was 2006. The 2000s were the first to be played† in the 2000s. 39 (6): 2113–2120. 2006MaMol.. 39.2113F. doi:10.1021/ma052448w. Howard, Philip H.; Muir, Derek K. G. (2010). In 2008, 2008, 2007. Technology. 44 (7): 2277–2285. Bibkod:2010EnST... 44.2277H. doi:10.1021/es903383a. 20163179. Retrieved 2016-10-12. in soy sauce and related products - Q&As. Archived from the original for 2014-02-22. ^ ^ Food Safety Administration (2013). Analysis of the emergence of 3-monochloropane-1,2-diolu (3-ICD) in Europe in 2009-2011 and preliminary impact assessment. EFSA log. 11 (9): 3381. 10.2903/j.efsa.2013.3381. 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