KILLING MOSQUITOS TO PREVENT MALARIA

DDT and Africa

The insecticide dichlorodiphenyltrichloroethane (DDT) was used widely throughout the world for agricultural pest control beginning in 1940 and for disease vector control beginning in 1944.(1) Vectors targeted with DDT included mosquitoes, lice, and fleas responsible for typhus, plague, yellow fever, and malaria. Its use increased after World War II to include protection of crops, and peaked in the United States in 1959.(2)

Bans on DDT began in Hungary in 1968.(3) By 1972, mostly environmental concerns over the use of DDT led to a restriction of its use as a general pesticide in the United States.(4) Bans were also implemented in Norway and Sweden in 1970, and throughout the developed world until 1984, when DDT was banned in the United Kingdom.(5) Bans in the rest of the world followed, and in 2001, the Stockholm Convention on Persistent Organic Pollutants restricted the use of DDT for disease vector control.(1)

As a result of these severe restrictions, much of the DDT originally present in the environment now exists as one of its metabolites, most commonly dichlorodiphenyldichloroethylene (DDE), which is formed when a HCl is eliminated from DDT. Where DDE:DDT ratios are less than one in the environment, such as those found recently in fish species in Côte d'Ivoire, recent DDT use can be inferred.(6) With regard to those environmental concerns, DDT's use in a more limited role as a vector control agent continues in many parts of the world.

Recent vector control efforts in Africa focus on indoor residual spraying (IRS) in areas where malaria is endemic. The cost-effectiveness of DDT reported by a 2007 study was cited as a measure of superiority for malarial vector control over other insecticides.(1) A variety of other organophosphate, carbamate, and pyrethroid insecticides are recommended by the WHO for indoor residual spraying, and varying resistance in mosquitoes to all insecticides affects insecticide choice by region.(1)

The WHO guidelines for indoor residual spraying include considerations for insecticide susceptibility and vector behavior, safety for humans and the environment, efficacy and cost-effectiveness, as well as sustained political support.(7)

> "In DDT therefore, we also possess an extremely valuable remedy in the fight against malaria, this the most widespread of all contagious diseases which yearly affects about 300,000,000 people and causes a yearly death rate of at least 3,000,000. In the cases of many other diseases spread by insects, diseases such as plague, murine typhus and yellow fever, significant results have been obtained."
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> ***Speech by Professor G Fischer, member of the Staff of Professors of the Royal Caroline Institute, as he presented to the King of Sweden the 1948 Nobel Laureate in Physiology or Medicine, chemist Dr Paul Mueller, who identified the antimalarial properties of DDT.***

In addition to its cost-effectiveness, there is a relative lack of health concerns in humans stemming from DDT's use in vector control. Possible concerns include neurological delays in breastfeeding babies, abbreviated lactation, higher incidence of preterm births, and impaired fertility, though hard evidence proving that these effects lead to increased mortality and morbidity is difficult to find.(1,8,9)

Most studies conclude there is no adverse effect in humans living in areas where widespread DDT use was once employed.(10,11) Vector control strategies, such as indoor residual spraying, also do not appear to cause harm to humans.(1) Liver function was unaffected not only in those benefiting from an indoor residual spraying program in South Africa, but also in those charged with applying DDT to households participating in an indoor residual spraying program.(12,13) A meta-analysis of studies attempting to correlate incidence of breast cancer with DDT exposure found no increased risk from

Road from Nairobi to Nakuru, in the malaria-endemic Rift Valley of Kenya. Photo WAW.

River in Edo State, Nigeria. Globally, Nigeria suffers the greatest burden of malaria. Photo courtesy of Jessie Edoro.

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DDT load.(14)

Even when adverse effects from DDT exposure are found, they are limited in their scope and severity. A 2006 study of neurodevelopment of Mexican-American children exposed to DDT found limited evidence of psychomotor and mental delays in children up to 2 years of age that correlated with exposure to DDT.(15) However, correlations between DDT and DDE and psychomotor and mental development were often insignificant at 95% confidence intervals, and were only 0.2 standard deviations or less when they were significant.

More recent environmental studies also support these assertions. Studies of Nile perch and tilapia from Lake Victoria in Uganda in 1998 found total DDT at 8% of the Australian maximum residue limits for DDT.(16) The Australian standard was the strictest cited. United States Food and Drug Administration action cocentrations were 5 times higher than the Australian standard.(16)

Studies of juvenile tilapia from the Tana River in Kenya from 1998 to 1999 measured concentrations of DDT that were below detection limits.(17) Concentrations of DDT in water samples from the Sabaki River in Kenya also had DDT levels that were below detection limits.(17) While DDE levels were considerably higher in fish samples from the Tana River, they were still only 14% of the Australian limit cited in the Ugandan study.(16,17) Concentrations of DDE in the Sabaki River were less than half that measured in the Tana River. In the Kenyan study, as well as most other DDT studies, analysis of other metabolites of DDT was included.

Even where DDT levels are considered high, the health risk is questionable. A 2006 study concluded that the Ouémé River catchment in Bénin "exceeded environmental quality standards".(18) However, a concomitant study concluded that the consumption of pesticides from fish in the Ouémé River did "not present an immediate risk".(19) A 2003 study of North American and African infant mortality concluded that the increased risk of death to infants from DDT application was comparable to the decrease in risk of infant death from malaria.(20)

Less obvious concerns, such as the effect of DDT on other drugs merit further study. A 2002 study of breastfeeding mothers using the non-steroidal anti-inflammatory drug paracetamol in Zimbabwe found that drug's half-life to be significantly reduced in those mothers as DDT concentrations in their milk increased.(20)

The mechanism of action of DDT has long been theorized to depend on the shape and size of the DDT molecule and the way it fits into voltage-gated sodium channels.(21) The molecule is believed to be of the appropriate size and shape to hold voltage-gated sodium channels of nerve cells in an open conformation, thereby slowing voltage gradient repolarization and encouraging repetitive discharges. Insects exposed to DDT exhibit hyperactivity and convulsions, consistent with this neurological hypothesis.(21)

Mutations in the DDT target receptor at key amino acids have been shown to confer DDT resistance. The threonine to isoleucine or valine mutation at The human health risks from use of DDT in IRS appear to be, at worst, a break-even proposal in infants, and include the possibility of mild neurodevelopmental delays. Data from non-infant populations suggest strongly that DDT poses a mild to insignificant health risk compared to risk of death and debilitation from malaria, though further study may be warranted to determine DDT's effects in the presence of other drugs or chemicals. Its high toxicity to Anopheles, low toxicity to humans, and superiority over other pesticides in combating malaria should encourage health authorities to pursue its use in indoor residual spraying, making alternative pesticide choices only when insect DDT resistance rises or particular human individuals or populations exhibit increased sensitivity to its use.

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