EFFECTS OF DDT ON ENVIRONMENT AND HUMAN HEALTH

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ABSTRACT

The study aims to discuss the source of DDT in the environment and critical review impacts of this insecticide in the environment and human health. Current monitoring and pollution prevention strategy for DDT also be assessed in a certain extent of the article. The methodology is based on review of literature and information from journals, published documents and the Internet. The study found that DDT has great deal of negative impacts on the environment and human health. Moreover, although DDT was not popular used in recent time, it still impacts on environment and human health due to long residual efficacy and accumulation through food chain. This was disseminated widely to warn population because there are still lots of countries manufacture and use DDT for many purposes, especially developing countries.

Key words: *DDT, environment, human health, impact*

Introduction

DDT is the abbreviation of *dichlorodiphenyltrichloroethane*. It was first synthesized by Zeidler in 1874; after that, in 1939 Paul Muller discovered its insecticidal properties (Mischke *et al*, 1985). DDT was first manufactured in 1943 (Pretty and Hine, 2005). This chemical agent was designed to destroy insects, weed, rodents, fungi and other human annoyance trouble (UNEP, 2004). In 1940s DDT was used as the first modern synthetic insecticide to control insect in agriculture, housing, institutes and to combat insect-borne human diseases (Environmental Protection Agency – EPA, 2012). In the period of World War II, DDT became a very popular and wonder-chemical for every extent of pest problem that "*if there is a single pesticide almost everyone can name, it's DDT*" (Pesticide Action Network, 2012). However, along with the widespread use of DDT is the escalation of more toxic material was found (Carson, 1962). DDT was realized as a reproductive toxic with increasing evidences of its adverse effects (Longnecker *et al*, 2005). Therefore, since 1970s DDT was banned in most of the Western countries (Biscoe, 2004). This particular pesticide was also considered as one of 12 restricted persistent organic pollutants in the world from the international agreement Stockholm Convention 2001 (WHO, 2011). Nevertheless, until present many developing nations still produce DDT for agriculture purpose and malaria vector combat (Biscoe *et al*, 2004). Moreover, WHO (2011) stated that "*there will be a continued role for DDT in malaria control*" which leads to reintroduce of this chemical agent in a number of countries and makes its usage become a controversial matter.

This study aims to discuss the source of DDT in the environment and critical review impacts of this insecticide in the environment and human health. Current monitoring and pollution prevention strategy for DDT also be assessed in a certain extent.

Methodology

In order to fulfill the study's onus, the research was based on review of literature and information from academic journals, the Internet and published documents. Collected information will be classified into three different groups:
- Background data to initiate and give essential perception about DDT, review the source and current status of this particular pesticide in the world,
- Relevant case studies from various researches to critical review effects of DDT in the wildlife and human health,
- Related documents to discuss strategy to prevent DDT pollution and suggest current monitoring and assessment methods for DDT.

The aim of the study will be addressed by critical reviewing, generalizing and comparing all information so that conclusion will be drawn refer to objectives.

Finding

Review of the source of DDT in the environment

There are many source of DDT in the environment. First of all, DDT can come from pesticide companies. In 1996, DDT residues in breast milk and environment were analyzed in Kafr El-Zayat, close to one of the biggest pesticide manufactures in Egypt. The result showed that high level of DDT was detected in food of animal origin, vegetable, fruit, soil and water (include groundwater, Nile river and tap water) (Dogheim *et al*, 1996).

Secondly, the prevalent use of insecticide in agriculture and industry has lead to widespread of DDT contamination in the environment (Hernandez *et al*, 1993). In 1940s DDT was accepted as *"a nearly universal insecticide for agriculture as well as residential, commercial and public health application*" (Mischke *et al*, 1985). In Ebro River basin of Spain where in the past pesticide extensively used in agriculture, the soil samples demonstrated a prevalence of 4,4'DDT and 4,4'DDE were detected (Hildebrabdt *et al*, 2008). In recent years, there is concern about the use of DDT in developing countries not only in agriculture but also in sanitary purpose (Al-targi *et al*, 2011).

DDT was recognized as a very strong insecticide and was extensively used in the World War II. After that, "*DDT became the global insecticide of choice"* to control malaria and other insect-borne diseases (Eskenazi *et al*, 2009). In 2006, WHO and the U.S. Agency for International Development approved of indoor DDT spraying to combat malaria (Eskenari et al, 2009). Currently, DDT is one of 12 insecticides recommended for Indoor residual spraying (IRS) by WHO (WHO, 2011). DDT use for IRS is considered as viable malaria control option (Biscoe *et al*, 2004). Nonetheless, spraying DDT to combat malaria causes to accumulation of this agent in the wildlife through food chains and in tissues of exposed organisms, as well as residents who live in sprayed houses (WHO, 2011).

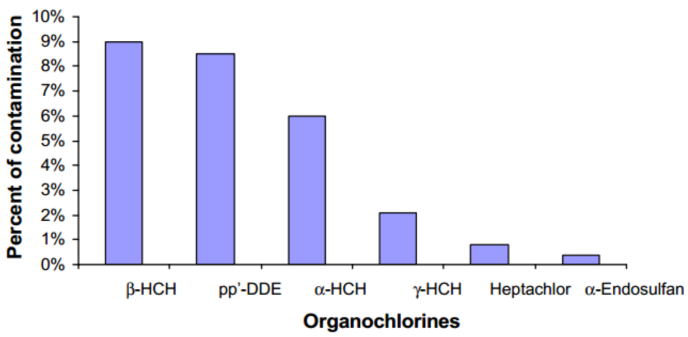
Effects of DDT on the environment and human health

DDT is recommended for malaria vector control because of its characteristic of the longest residual efficacy (WHO, 2011). When this agent is sprayed on walls or ceilings it can stay for 6 – 12 months. Until present, there is no substitute for DDT in terms of comparable efficiency and operational possibility on vector-borne diseases control (WHO, 2011). However, if DDT is not improperly used or stored, it can damage environment (UNEP, 2004).

DDT was classified into class II (moderate toxic) in the WHO classification system for pesticides (Pretty and Hine, 2005). In the environment, DDT has been found in rainfall and can travel a very long distance (Conway and Pretty, 1991). Its residue in the environment is very persistent and has not entirely disappeared for a long time (Pretty and Hine, 2005). This leads to disruption in the wildlife with the first recognized case was effect of DDE (the stable breakdown product of DDT) on eggshell thickness of herring gull in 1971 (Cade *et al*, 1971). After that, discoveries of Peakall *et al* (1976) and Hickley (1988) showed similar relationship with peregrines, falcons and hawks. Another noticeable case that DDT disrupted the wild population was in Lake Apopka in 1980. A major spill of a mixture including dicofol and DDT led to raised level of endocrine disrupting chemicals in the alligators' tissue, a range of developmental abnormalities phenomenon and a decrease of 50% of juvenile alligator numbers (Vos *et al*, 2000). Jensen and Jansson (1976) reported that high body concentration of DDT and its metabolites were clearly resulted to the disease syndrome in grey and ringed seals in Baltic which caused to decline of seal population. DDT is a neuro-developmental toxicant because it "*exerts the insecticidal effects by disrupting the nervous system*" (Eskenazi et al, 2009). For example, there were evidences in changes of behavior and neurochemistry into adulthood of mice which exposure to DDT in stage of pre-natal and neonatal nervous system development (Johansson *et al*, 1996).

Contamination of organochlorine pesticides (OCPs) include DDT in daily production of food is a worldwide phenomenon (Salem *et al*, 2009). In study by Salem *et al* (2009) in Jordan, 20 samples (8.5%) out of 233 daily product samples were contaminated with pp'-DDE (figure 1 and 2).

Figure 1: Percent of contaminated samples with organochlorine pesticides in Jordan (Salem *et al*, 2009)



Negative impacts of DDT on human health include "*acute and persistent injury to the nervous system, lung damage, injury to the reproductive organs, dysfunction of the immune and endocrine systems, birth defects, and cancer*" (Mansour, 2009). DDT is a possible long-term toxicity and remains for a long time in the environment (WHO, 2011). Consequently, by consuming food and contact with soil, water, air, people are potential exposed to excessive level of pesticide (UNEP, 2004). Table 1 below summarizes the expose level of DDT from the expose assessment of WHO expert consultation (2009) in areas where IRS is used extensively.

Figure 2: Percent of daily products contaminated with organochlorine pesticides in Jordan (Salem *et al*, 2009)

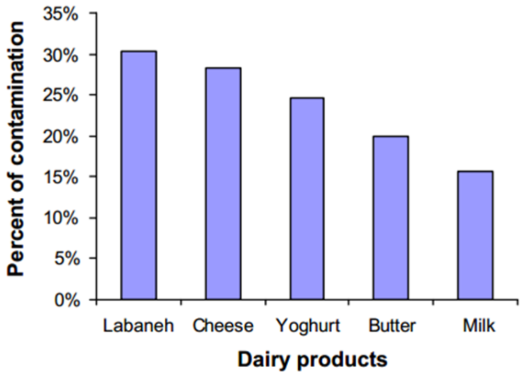


Table 1 illustrates that people in IRS areas were facing with the risk of high DDT expose level in which the IRS spray operator had the highest level. DDT is an acute poisoning hazard to children which can cause to convulsion and death if it is ingested at high dose (WHO, 2011). In fact, DDT causes acute poisoning cases and hospitalization to millions every year (UNEP, 2004).

Table 1: Exposures of different populations, measured as total DDT or DDE concentrations

Tissue	Occupational exposure (IRS spray operators)		Residents in IRS-treated homes[b]		General population[c]	
	Total DDT	DDE	Total DDT	DDE	Total DDT	DDE
Blood serum (lipid adjusted)	Mean: 77.8 (8.7–241.1) µg/g lipid	Mean: 41.8 (7.1–131.8) µg/g lipid	Mean: 9.8 (1.09–21.8) µg/g lipid	Mean: 19.7 (0.8–77.9) µg/g lipid	Mean: 5.0 (0.38–26.1) µg/g lipid	Mean: 1.0 (0.2–3.18) µg/g lipid
	Median: 58.6 µg/g lipid	Median: 25.6 µg/g lipid	Median: 5.18 µg/g lipid	Median: 9.7 µg/g lipid	Median: 0.93 µg/g lipid	Median: 0.77 µg/g lipid
Umbilical cord blood	—	—	Mean: 29.9 µg/l	Mean: 60.3 µg/l	Mean: 15.0 µg/l	Mean: 3.1 µg/l
	—	—	Mean: 4.6 µg/g lipid	Mean: 9.3 µg/g lipid	Mean: 0.44 µg/g lipid	Mean: 0.36 µg/g lipid

(Source: WHO, 2011)

The International Agency for Research on Cancer (IARC, 2006) classified compounds of DDT as carcinogenic to humans that leads to many kinds of cancer. Reports of Falck *et al* (1992) and Wolff *et al* (1993) linked exposure to p,p'-DDT and its persistent metabolites with the augmentative risk of breast cancer in women, probably due to p,p'-DDT's known estrogenic properties (Schecter et a,l 1997). Research of McGlynn et al (2006) showed that liver cancer risk was considerably raised in Chinese men who have highest levels of DDT in blood in comparison with men have lower levels of DDT. "*DDT could play a role in pancreatic cancer by modulating activation of the oncogene K-ras"* (Porta *et al*, 1999).

DDT impact on pregnant women was proved by research of Cohn *et al* (2003). This study assessed DDT in preserved maternal serum samples in 1960 and 1963; 28–31 years later, 289 eldest pregnant daughters were examined. The research found an unexplained decrease in fecund ability associated with prenatal DDT of these daughters of 32% per 10 µg/L DDT in maternal serum (Cohn *et al*, 2003). The study of Longnecker et al (2001) found that women with high levels of DDE in serum during pregnancy have 3.1 times higher odds of preterm delivery. DDE existence in breast milk also lead to a shorter lactation duration (Rogan *et al*, 1987).

In 1950 report of Laug *et al* about the present of OCP in human milk was recorded at the first time (Jani *et al*, 1988). Since then, human milk was contaminated by pesticides DDT became a concern that promoted numerous studies around the world (Smith, 1999) and was reported from various nations during 1970 - 1980 (Hernandez *et al*, 1993) (See figure 3). Recently, collected data of Al-targi *et al* (2011) about OPC residues in human milk is illustrated in table 2 below:

Table 2: Concentration of organochlorine pesticide residues (µg/g lipid) in human milk in various parts of the world

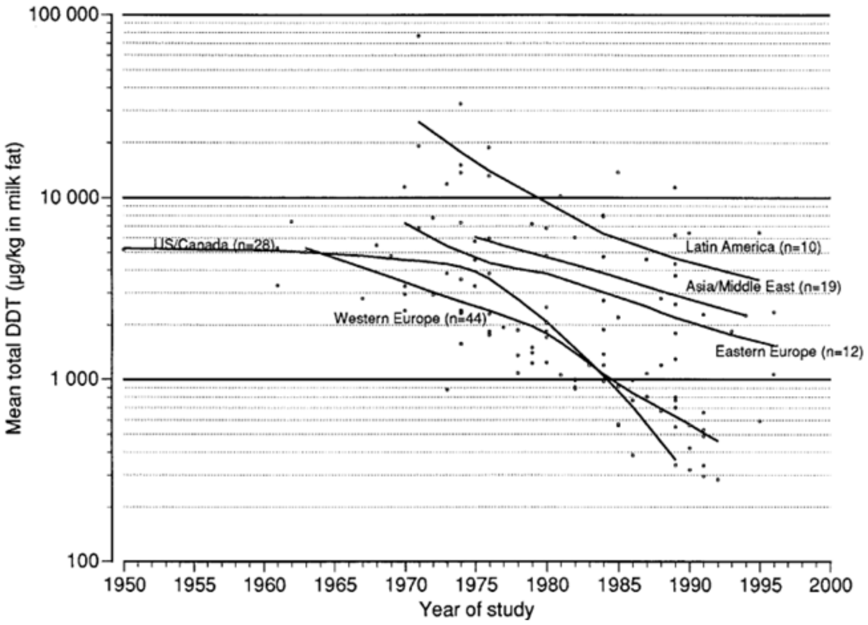
Countries	P,p'-DDE	p.p'-DDT	ΣDDT	DDE///DDT
China Beijing	1.72	0.24	1.96	7.17
Czech Republic	0.98	0.83	1.96	11.8
Central Taiwan	0.30	0.02	0.32	13.7
Egypt	0.14	0.01	0.15	14

India –Agra	0.06	0.05	0.17	1.2
Indo-nesia	0.28	0.06	0.34	46.6
Japan	0.27	0.02	0.29	13.5
Mexico	3.99	0.65	4.64	5.07
New Zeland	0.63	0.03	0.66	2.1
Russia	0.97	0.15	1.12	6.6
France	2.18	0.08	2.26	27.3
Spain	0.60	0.01	0.61	60
Libya	0.07	0.15	0.22	0.45

(*Source:* Al-targi *et al*, 2011)

Human milk is a "*contamination source for the breast-feeding baby*" (Campot *et al*, 2001). In investigation of Siddiqui *et al* (1981) noticeable level of DDT was detected in all samples of milk, blood, and neonatal blood of mothers. After the significant correlations of these compounds were observed, the study concluded that the existence DDT in the neonatal blood specified its passage from mother to unborn babies across the placenta, and warn about a great deal of daily intake of toxic food that neonates consume by feeding contaminated breast milk (Siddiqui *et al*, 1981). This chemical agent was also the most common of OCP compounds in women breast milk in India (Devanathan *et al*, 2009). OCP compounds expose can leads to low birth weight, neurodevelopment delay and disturbance of thyroid hormone status in newborns (Al-targi *et al*, 2011).

Figure 3: Mean total DDT compounds in breast milk reported by region and year of study (Smith, 1999)



In term of lasting impact of DDT on mental capacities, study proved that this agent is a "*neuroendocrine disrupter*" and a "*functional teratogen*" which cause to "*harmful in later life"* (Dorner and Plagemann, 2002 *effects on brain development and mental capacities*). The study of Eskenazi *et al* (2006) furthermore concluded that children who were highly exposed to DDT had very low ability in copy and immediate recall test; they recalled less information on the memory score. Report of Ribas-Fito et al (2003) researched on 92 newborn babies of a population located next to an electrochemical company in Spain proved that prenatal exposure to p,p'DDE was related to an impediment in the mental and psychomotor development of 1year infants. Children who had high concentrations of DDE in prenatal period were notably shorter at 1, 4, and 7 years of age (Ribas-Fito *et al*, 2006).

Negative impacts of DDT on human health were summarized by Eskenazi et al (2009) which included important findings such as association of DDT exposure with an increase cases of diabetes; pregnancy loss; changes in gestational length, birth weight and time to conception; shorter duration of lactation; urogenital birth defect; earlier age at menarche; change in male and female fertility; depress triiodothyronine and effect on immune system (Eskenazi *et al*, 2009).

Discussion

Current monitoring and assessment methods of DDT in the environment

Common methods to assess DDT in laboratory are using chemical and physicochemical indicators. However, such methods are usually both labor and time consuming, requiring highly-trained workers and obtained data is often limited and routine (Wolska *et al*, 2007). In recent years, bio-analytical and eco-toxicological techniques have been developed as methods using live organisms indicators. Bio-tests identify the existence of toxic materials in the environment and find out their toxicity by quantitatively estimating their harmful effects on live organisms. In eco-toxicological tests environmental records and information related to impacts of pollutants on live organisms have been examined by both chemical techniques and biotests (Wolska *et al*, 2007).

Another widely used assessment method and monitoring of DDT in the environment is a biomarker. Biomarkers are employed to identify the effects of toxic agents before they cause to harmful impacts on human health (Anwar, 1997). Biomarkers help to recognize causal connection and make quantitative estimation of those connections better at appropriate exposure level (WHO, 1993). Biomarkers can detect the chemical agents in the environment or their metabolites in blood, urine, genetic material, and even cell death (Imran and Dilshad, 2011). This assessment measure can also help identify people who are vulnerable to the exposure risk to certain types of agents in environment and at work, especially with the development of molecular genetics (Vainio, 2001).

In a range of recent studies, residual content of DDT and its metabolites were assessed by gas chromatography - electron capture detector (GC-ECD) and gas chromatography–mass spectrometry (GC-MS) which involve the use of efficient extraction and sample purification processes and detection by gas chromatography (Margariti *et al*, 2007). The process comprise *"denaturation of serum proteins, off-line SPE (C18), and elution withn-hexane/dichloromethane. The extracts were purified with a florisil–silica gel column and analyzed by GC with micro-electron capture detection (mECD). The method was validated by using PCB-174 as internal standard (IS), and the LODs were 1.5– 5.0 pg/mL for organochlorine pesticide"s* (Conka et al, 2005). Assessment of DDT was especially improved with the development of the oasis lypophily balanced matrix (HLB) sorbent cartridges for extraction of DDT from serum because it helps progress extraction recoveries and save time spend on lipid clean-up and sample-preparation (Margariti *et al*, 2007).

Strategy to prevent environmental pollution from DDT

Although there are many evidences about persistent residues and adverse effects of DDT in the environment and human health, the fundamental mode of action of DDT compound is still not fully understood (Vos *et al*, 2000). Especially, in developing countries negative impacts of pesticide are not highlighted (Mansour, 2004). Education and propaganda therefore play a crucial role in prevention of pollution and acute poisoning of DDT. However, pesticide industry often base on "*safe use*" campaigns to encourage the awareness that training will decrease health problem related to the use of dangerous pesticide. This leads to underestimation of the risk and harmful impacts of pesticide use and limited or biased research into this area (Wulp and Pretty, 2005). Furthermore, pesticide is still a crucial factor that contribute into current level of production in many farming system. Thus, it is a responsibility of governments with a range of legislation and policy instruments to prevent potential risks from DDT. In fact, *"It took nearly half a century of scientific and policy effort to establish some degree of causality and to make important policy decisions to remove DDT products from use"* (Pretty and Hine, 2005).

Childhood is the most important period for primary preventive medicine and preventive pedagogics. Since various findings demonstrated source of DDT contamination in breast milk, a neuroendocrine prophylaxis in early life was recommended by Dorner and Plagemann (2002).

The abuse of insecticides and the lack of or weakness of national managing plans cause to adverse effects of DDT in numerous developing nations (Mansour, 2004). Therefore, it is necessary to severely restrict the usage of DDT by law and legislations in both international and national level. For instance, the US Government has established regulatory controls on the use of pesticide to diminish exposure of residents to residues of pesticide in food (National Resource Council, 1993).

In many areas where DDT is presently being used for malaria control there is the lack of understanding about human exposure and adverse effects of this agent; hence, research is recommended to deal with this gap and to increase safe and effective alternatives to DDT (Eskenazi *et al*, 2009). In order to completely restrict the use of DDT in agriculture it is necessary to encourage other effective measures to grow crop such as implementing integrated pest management (IPM) program. A study in Nicaraguan found that IPM is an effective way to reduce pesticide use and decrease incidences of acute pesticide poisoning (Kishi, 2005).

Conclusion

The study shows that DDT was the most popular and effective pesticide to help people combat unwanted organisms and gain dramatically improvement in agriculture. The wide spread use and manufacture of DDT after the World War II was main sources of DDT in the environment. However, since a number of adverse effects of this insecticide were reported, usage of DDT was banned international wide. Despite the severe restriction, DDT is still illegally used in many areas, especially in developing nations.

Negative impacts of DDT on the environment and human health were acknowledged and disseminated widely to warn population and prevent unexpected situations occur. Nevertheless, although DDT was not used in recent time, it still impacts on environment and human health due to long residual efficacy and accumulation through food chain. DDT leads to great deal of negative effects in the wildlife such as on eggshell thickness of herring gull, peregrines, falcons and hawks; led to raised level of endocrine disrupting chemicals in the alligators' tissue and a decrease of 50% of juvenile alligator numbers; resulted to the disease syndrome in grey and ringed seals in Baltic which caused to decline of seal population; DDT is also a neuro-developmental toxicant with a lot of evidences in changes of behavior and neurochemistry into adulthood of mice which exposure to DDT in stage of pre-natal and neonatal nervous system development. In term of human health, DDT is the cause of many kinds of cancer, acute and persistent injury to the nervous system, lung damage, injury to the reproductive organs, dysfunction of the immune and endocrine systems, birth defects.

There are great deal of efforts have been brought out worldwide to monitor DDT as using chemical and physicochemical indicators, biomarker, gas chromatography - electron capture detecto and gas chromatography–mass spectrometry; combine with education and propaganda, legislation and policy instruments, national managing plans or encourage other effective measures to control insect in agriculture. Nevertheless, how to diminish the harmful impacts of DDT still a permanent concern not only of scientists and environmentalists but also people facing with risk of organ chlorine compound exposure.

The study is not a full update on all incidences of effects of DDT on environment and human worldwide; it also did not provide the best measures to deal with DDT residues in the soil, water (including groundwater) environment which is being applied in the world. Therefore, it is recommended that future studies should focus on concrete measures to control the usage and harmful effects of DDT residues, especially in poor and developing countries in Africa and Asia.

References

Al-targi. H. M. Z., Abou El Ela. G. R., El-Dressi. A.Y. (2011) Organochlorine pesticide residues in human breast milk in El-Gabal Al-Akhdar, Libya. International Conference on Life Science and Technology IPCBEE vol.3. IACSIT Press, Singapore.

Anwar. A. W. (1997) Biomarker of human exposure to pesticide. Environment Health Perspect, 105, 801 – 806.

Hashmi, Imran and Khan A. Dilshad (2011). Adverse Health Effects of Pesticides Exposure in Agricultural and Industrial Workers of Developing Country. http://www.intechopen.com/books/pesticides-the-impacts-of-pesticides-exposure/adverse-health-effects-of-pesticides-exposure-in-agricultural-and-industrial-workers-of-developing-c (Cited: 7 February 2014).

Biscoe. L. M., Mutero. M. C., Kramer. A. R., (2004) Current policy and status of DDT use for malaria control in Ethiopia, Uganda, Kenya and South Africa. Working paper 95. International Water Management Institute.

Burse. V. W., Najam. A. R, Williams. C. C, (2000) Utilization of umbilical cords to assess in utero exposure to persistent pesticides and polychlorinated biphenyls. Expo Anal Environ Epidemiol, 10, 776–788.

Cade. T. J., Lincer. J. L., White. C. M., Roseneau. D. G., Swartz. L. G. (1971) DDE residues and eggshell thinning changes in Alaskan falcons and hawks. Science 172, 955 – 957.

Campoy. C., Jimenez. M., OleaS. M. F.,MorenoFrias. M., Canabate. F., Olea. N., Bayes. R., &Molina-Font. J. A.,(2001) Analysis of organochlorine pesticides in human milk: preliminary results. Early human development, 65, 183-190.

Carson. R., (1962) Silent Spring. Boston, New York: Houghton Mifflin.

Cohn. B. A, Cirillo. P. M., Wolff. M. S., Schwingl. P. J., Cohen. R. D., Sholtz. R. I., Ferrara .A, Christianson. R. E, van den Berg B. J. and Siiteri. P. K,.(2003), DDT and DDE exposure in mothers and time to pregnancy in daughters. Lancet. 2003 Jun 28;361(9376):2205-6. http://www.niehs.nih.gov/research/supported/sep/2003/ddt-dde/index.cfm. Cited: 10 February 2014.

Conka K, Drobna B, Kocan A, (2005) Simple solid-phase extraction method for determination of polychlorinated biphenyls and selected organochlorine pesticides in human serum. Chromatogr A, 1084, 33–38.

Conway. G. R. and Pretty. J. N. (1991) Unwelcome Harvet: Agriculture and Pollution. London: Earthscan.

Devanathan. G., Subramanian. A., Someya. M., Sudaryanto. A., Isobe. T., Takahashi. S., Chakraborty. P., and Tanabe. S., (2009) Persistent organochlorines in human breast milk from major metropolitan cities in India. 157, 148–154.

Dogheim, S. M., El-Zarka. M., Gad-Alla. S. A., El-Saied. S., Emel. S. Y., and Mohsen. A. M.,(1996) Monitoring of pesticide residues in human milk , soil, water, and food samples collected from Kafr El-Zayat Governorate. AOAC International, 79, 111-116.

Dorner. G., and Plagemann. A., (2002) DDT in human milk and mental capacities in children at school age: an additional view on PISA. Neuroendocrinol Lett, 23, 427-431.

Environmental Protection Agency (2012) DDT – A brief history and Status. http://www.epa.gov/pesticides/factsheets/chemicals/ddt-brief-history-status.htm. Cited: 15 February 2014

Eskenazi. B., Marks. A. R., Bradman. A., Fenster. L., Johnson. C., Barr. D.B., (2006.) In utero exposure to dichlorodiphenyltrichloroethane (DDT) and dichlor -odiphenyldichloroethylene (DDE) and neurodevelopment among young Mexican American children. Pediatrics, 118, 233–241.

Eskenazi. B., Chievrier. J., Rosas. G. L., Anderson. A. H., Bornman. S. M., Bouwman. H., Chen. A., Cohn. A. B., Jager. C., Henshel. S. D., Leipzig. F., Leipzig. S. J., Lorenz. C. E., Snedeker. M. S., Stapleton. D. (2009) The Pine River Statement: Human Health Consequences of DDT Use. Environment Health Perspect, 117, 1359–1367.

Hernandez. L. M., Fernandez. M. A., Hoyas. E., Gonzalez. M. I., and Garcia. G. F. (1993) Organochlorine insecticides and polychlorinated biphenyl residues in human breast milk in Madrid (Spain) Bull. Environ. Contam. Toxicol, 50, 308 – 315.

Hickley. J. (1988) Some recollections about eastern North America's peregrine falcon population crash. The Peregrine Fund, Boise, Idaho.

Hildebrandt. A., Lacorte. S., Barcelo. D. (2009) Occurrence and fate of orgachlorinated pesticide and PAH in agriculture soil from the Ebro River basin Arch Environ Contam Toxicol, 57, 247–255.

Hong. Z, Gunter. M, Randow. F. E. (2002) Meconium: a matrix reflecting potential fetal exposure to organochlorine pesticides and its metabolites. Ecotoxicol Environ Saf, 5, 60–64.

IARC, International Agency for Research on Cancer (2006) Overall Evaluations of Carcinogenicity to Humans. http://www.monographs.iarc.fr/ENG/Classification/crthallcas.php. Cited: 25 February 2014

Jani. J. P., Patel. J. S., Shah. M. P., Gupta. S. K., & Kashyap. S. K(1988) Levels of organochlorine pesticides in human milk in Ahmedabad, India. Int Arch Occup Environ Health, 60, 111-113.

Kishi. M. (2005) The health impacts of pesticides: What do we now know? London: Earthscan.

Lacassie E, Marquet P, Gaulier J, (2001) Sensitive and specific multiresidue methods for the determination of pesticides of various classes in clinical and forensic toxicology. Forensic Science Int, 121, 116–125.

Longnecker. P. M., Klebanoff. A. M., Dunson. B. D., Guo. X., Chen. Z., Zhou. H., Brock. W. J., (2005) Maternal serum level of the DDT metabolite DDE in relation to fetal loss in previous pregnancies. Environmental Research, 97, 127 – 133.

Margariti. G. M, Tsakalof. K. A., and Tsatsakis. M. A. (2007) Analytical Methods of Biological Monitoring for Exposure to Pesticides: Recent Update. Ther drug Monit, 29, 150 – 163.

Mansour. S. A.,(2004) Pesticide exposure-Egyptian scene. Toxicology, 198, 91-115.

Mischke. T., Brunetti. K., Acosta. V., Weaver. D., Brown M. (1985) Agriculture source of DDT residues in California's environment. California, US: California Department of Food and Agriculture.

National Resource Council (1993) Pesticides in the diets of infants and children. National Academy Press, Washington, DC.

Nagayama. G., Okamura. K., Iida. T., Hirakawa. H., Matsuda. T., Tsuji. H., Hasegawa. M., Sato. K., Yanagawa. T., Igarashi. H., Fukushige. J and Watabane. T.,(1998) Oganochlorine pesticide residues in human breast milk in El-Gabal Al-Akhdar, Lybia. Chemosphere, 37, 1789-1793.

Peakall. D. B., Reynolds. L. M., French. M. C. (1976) DDE in eggs of the peregrine falcon. Bird Study, 23, 183 – 186.

Pesticide Action Network (2012) The DDT story. http://www.panna.org/issues/persistent-poisons/the-ddt-story. Cited: 27 February 2014

Pretty. J. and Hine. R. (2005) Pesticide Use and the Rnvironment. London: Earthscan.

Ribas-Fito. N., Cardo. E., Sala. M., Eulalia de Muga. M., Mazon. C., Verdu. A., Kogevinas. M., Grimalt. J. O., Sunyer. J.(2003) Breastfeeding exposure to organo- chlorine compounds and neurodevelopment in infants. Pediatrics, 111, 580–585.

Schecter. A., Toniolo. P., Dai. L. C., Thuy. L. T. B., Wolff. M. S. (1997) Blood Levels of DDT and Breast Cancer Risk Among Women Living in the North of Vietnam Arch. Environ. Contam. Toxicol, 33, 453–456.

Siddiqui. M. K. J., Saxena. M. C., Bhargava. A. K., Seth. T. D., Krishnamurthi. C. R., Kutty. C. R. (1981) Agrochemicals in the maternal blood, milk, and cord blood: a source of toxicants for prenates and neonates. Environ Res, 24, 24-32.

Smith. D. (1999) Worldwide trends in DDT levels in human milk. Int J Epidermal, 28, 179-188.

Sundberg. S. E, Ellington J. J, Evans J. J. (2006) A simple and fast extraction methodfor organochlorine pesticides and polychlorinated biphenyls in smallvolumes of avian serum. Chromatogr B, 83, 99–104.

Vianio, H. (2001).Use of biomarkers in risk assessment. I. J. Hyg. Environ. Health, 204, 91-102.

Vos. T. G. M. (2000) Vietnam Showcase. Training News, Biocontrol News & Information 21 (1)

WHO, World Health Organisation (2011), DDT in indoor residual spraying: Human heath aspects. Environmental health criteria 241.

WHO, World Health Organization (2013) Childhood pesticide poisoning: Information for advocacy and action. http://www.who.int/ceh/publications/pestipoison/en/. Cited: 20 February 2014

WHO, World Health Organization (2011) The use of DDT in malaria vector control. WHO Position Statement. Electronic facsimile downloaded as a PDF from www.google.co.uk

WHO, World Health Organization (1993) Biomarkers and risk assessment: Concepts and Principles. Geneva: World Health Organization.

Wolska. L., Sagaidakow. A., Kuczynska. A., Namiesnik. J. (2007) Application of ecotoxicological studies in integrated environmental monitoring: Possibilities and problems. TrAC Trends in Analytical Chemistry, 26, 332 – 344.

Yu. Z., Palkovicova. L., Drobna. B., Petrik. J., Kocan. A., Trnovec. T., and Picciotto. I. H., (2007) Comparison of organochlorine compound concentrations in colostrum and mature milk, 66, 1012-1018.