

# Pesticide and its Environmental and Health Hazard.

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## 1. Introduction

Pesticides a heterogeneous category of chemicals are specifically designed to control pests that are responsible for plant diseases thus increasing the crop yields. The term insecticide is used to represent agents that are designed to kill only insects, but the term pesticide has a broader meaning and also includes herbicides, rodenticides, fumigants, nematocides, algacides, ascaricides, molluscicides, disinfectants, defoliant and fungicides (Erdman, 2004). A total of approximately 890 active ingredients are registered as pesticides in USA and they are currently marketed as approximately 20,700 pesticide products (US EPA, 1996; Bolognesi, 2003). The first known pesticide was elemental sulphur dust used in Sumeria about 4500 years ago. In the 19th century, pyrethrum derived from chrysanthemums, and rotenone derived from the roots of tropical vegetables was introduced. After its discovery in 1939 by Paul Muller, dichlorodiphenyltrichloroethane (DDT) found widespread use. However, with the recognition that it was a threat to biodiversity, the use of DDT has declined considerably. In India, the use of organic pesticides began in 1948 with the introduction of DDT for the control of malaria and benzene hexachloride (BHC) for locusts. Production of these substances in India started in 1952

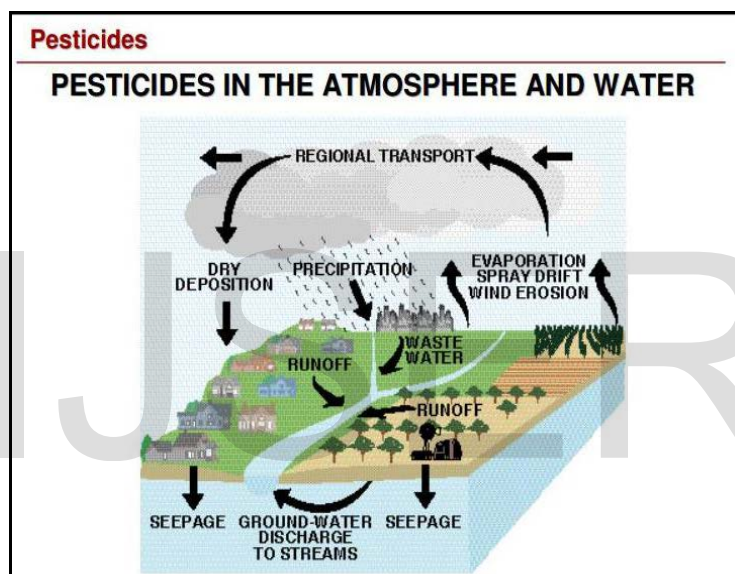
(Gupta, 2004). The primary benefit of the pesticides includes the improved productivity, protection of crop losses/yield reduction, vector disease control and quality of food. Table. I summarize the classification of chemical pesticides which are common in use.

**TABLE I. Classification of Pesticides**

<b>Types of Insecticides</b>	<b>Examples</b>
<b><i>Insecticides</i></b>	Acetylcholinesterase inhibitors: Organophosphates such as <b>Malathion</b> , carbamates Organochlorines Pyrethrins and pyrethroids
<b><i>Herbicides</i></b>	Dipyridyl pesticides: <b>Paraquat</b> and diquat Chlorphenoxyacetate weed killers: Bromoxynil, 2,4-D
<b><i>Fungicides</i></b>	Substituted benzene: Chloroneb, chlorothalonil Thiocarbamates Organomercurials: Methylmercury, phenylmercuric acetate
<b><i>Molluscicides</i></b>	Metaldehyde
<b><i>Rodenticides</i></b>	Aluminium phosphide Zinc phosphide Warfarin and superwarfarin compounds Heavy metal: Thallium-containing pesticides Yellow phosphorus
<b><i>Insect repellants</i></b>	Diethyl toluamide (DEET)
<b><i>Miscellaneous</i></b>	Anilides Avermectins

If the pros of pesticide include enhanced economic potential in terms of increased production of food, then their cons have resulted in serious health implications to man and environment. No segment of the population is completely protected against exposure to pesticides and the

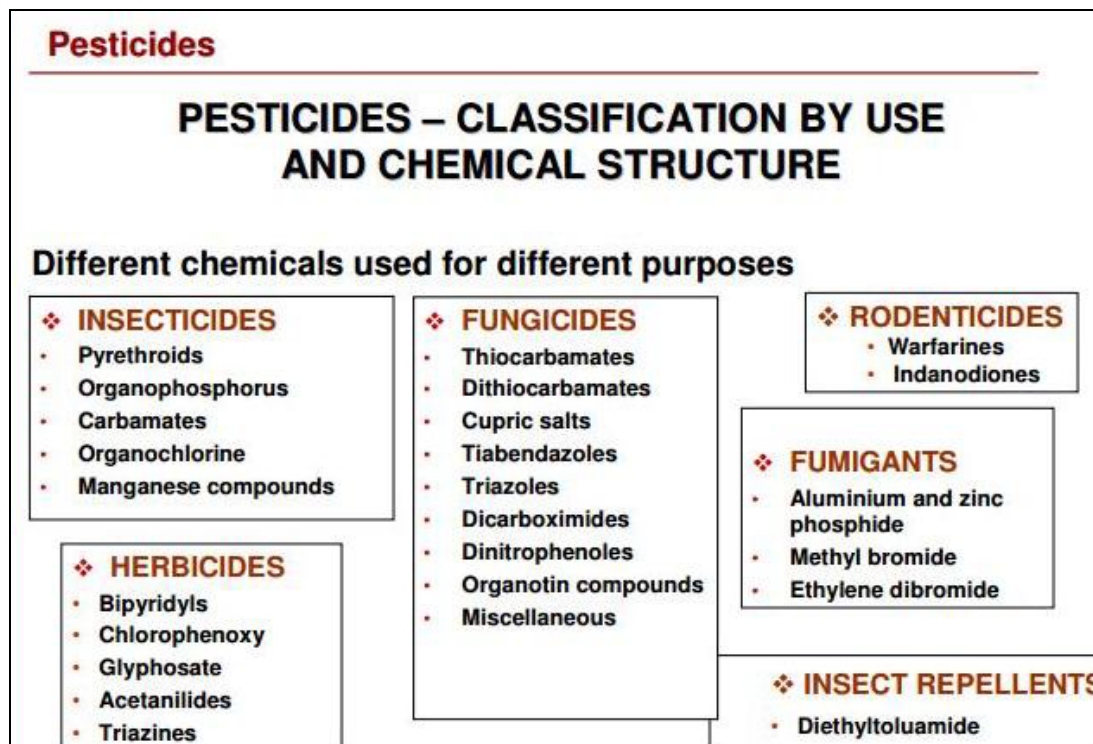
potentially serious health effects, though a disproportionate burden, is shouldered by the people of developing countries and by high risk groups in each country (WHO, 1990). According to the study made by the US Geological Survey (USGS), 90% of streams and 50% of wells tested ground and surface water were positive for at least one pesticide monitors among 76 pesticides and seven pesticide breakdown products. There are many groups of chemicals used as pesticides. A great potential for adverse effects of pesticides is through contamination of the hydrological system, which supports human life, aquatic life and related food-chains.



Courtesy: U.S. Geological Survey, Fact Sheet FS-152-95

Fig.1: Pathways of pesticide movement in the hydrologic cycle.

The contamination of the hydrologic system results in the adverse effect of pesticides. Water is one of the primary means by which pesticides are transported from their application areas to other parts of the environment. Thus, there is potential for movement of pesticides into and through all components of the hydrologic cycle which supports not only human life, but aquatic life and related food chains as well.

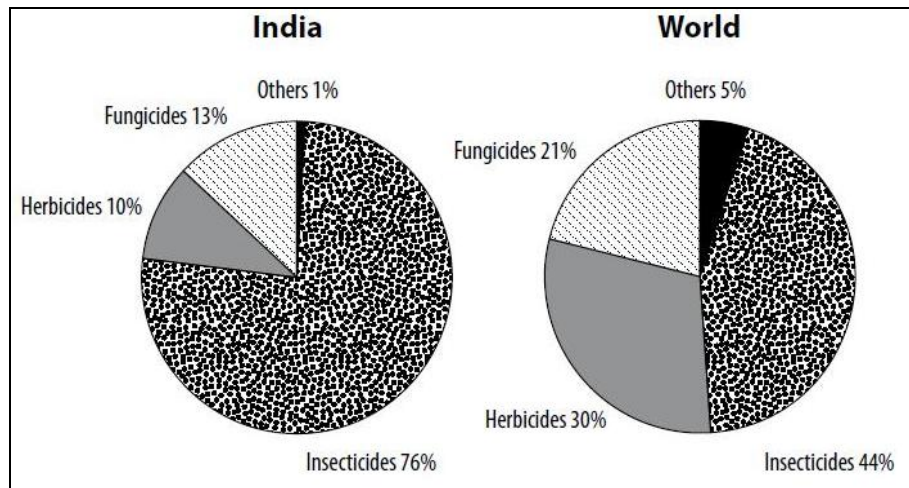


Courtesy:

U.S. Geological Survey, Fact Sheet FS-152-95

Fig. 2: Pesticides Classification

In India, the production of pesticides started in 1952 with the establishment of a plant for the production of BHC near Calcutta. Since then, its production and usage increased with a greater pace as a result of which in 1996–97 the demand for pesticides in terms of value was estimated to be around Rs. 22 billion (USD 0.5 billion), which is about 2% of the total world market. The pattern of pesticide usage in India is different from that for the world in general.



Courtesy: Aktar *et al.*, 2009

Fig. 3: Consumption of Pesticides in India

## 2. PESTICIDES EXPOSURE:

Different groups of population are exposed to pesticides in different ways and in different degrees. Individuals are frequently exposed to many different pesticides or mixtures of pesticides, either simultaneously or serially. These exposures are often highly correlated, particularly within functional or chemical groups, making it difficult to identify effects of particular agents. Exposure is acute if exposed to a large amount of pesticide once. It's usually easy to identify acute exposure. Exposure is chronic when having a low-level exposure over and over. Chronic exposure may be hard to tell. Either kind of exposure is dangerous. There are four ways a person can be exposed to pesticides:

- **Oral exposure** — swallowing pesticide
- **Dermal exposure** — getting pesticide on your skin, the most common type
- **Inhalation exposure** — breathing in pesticide
- **Ocular exposure** — getting pesticide in your eye.

There are different causes of each type of exposure as summarized in Table 2.

Type of Exposure	Cause of Exposure
<i>Oral exposure</i>	<ul style="list-style-type: none"> <li>• Not washing hands before eating, drinking, using tobacco.</li> <li>• Eating or drinking a pesticide by mistake.</li> <li>• Getting pesticide on food.</li> <li>• Splashing pesticide into the mouth.</li> <li>• Blowing out plugged nozzles with the mouth.</li> </ul>
<i>Dermal exposure</i>	<ul style="list-style-type: none"> <li>• Getting pesticides on bare skin.</li> <li>• Applying pesticides in windy weather.</li> <li>• Wearing inadequate PPE.</li> </ul>
<i>Inhalation exposure</i>	<ul style="list-style-type: none"> <li>• Prolonged contact in poorly ventilated areas.</li> <li>• Not using proper PPE.</li> <li>• Breathing vapors after application.</li> <li>• Using the wrong respirator.</li> <li>• Using an improperly fitted respirator.</li> <li>• Using tainted filters, cartridges, or canisters.</li> </ul>
<i>Ocular exposure</i>	<ul style="list-style-type: none"> <li>• Getting pesticides in the eyes.</li> <li>• Not using proper eye cover when:                             <ul style="list-style-type: none"> <li>• Spraying pesticide</li> <li>• Handling pesticide</li> </ul> </li> <li>• Rubbing the eye with tainted gloves or hands.</li> </ul>

### 3. PROS AND CONS OF PESTICIDES

Crops are affected by different pests and by competition from weeds. Several insects and other arthropods, fungi, mollusks and bacteria attack crops and result in quantitative and qualitative losses and the degree of damage varies greatly in different climatic and agricultural regions. During last three decades, chemical control of pests and weeds aimed at minimizing losses has been introduced throughout the world. Pesticides have been an integral part of the process by reducing losses from weeds, diseases and insect pests that can markedly reduce the amount of harvestable produce (Aktar *et al.*, 2009).

If the credits of pesticides include enhanced economic potential in terms of increased production of food and amelioration of vector borne diseases, then their debits have resulted in serious

health implications to man and his environment. These chemicals produce a potential risk to humans and other life forms and unwanted side effects to the environment (Forget, 1993; Igbedioh, 1991). The world wide deaths and chronic diseases conditions due to pesticide exposure number about 1million per year (Environews Forum, 1999). The high risk groups exposed to pesticides include production workers, formulators, sprayers, mixers, loaders and agricultural farm workers. In industrial settings, workers are at increased risk since they handle various toxic chemicals including pesticides, raw materials, toxic solvents etc. Various pesticides in industrial settings of the unorganized sector revealed a high occurrence of generalized symptoms (headache, nausea, vomiting, fatigue, irritation of skin and eyes) besides psychological, neurological and gastrointestinal symptoms coupled with low plasma cholinesterase (ChE) activity (Gupta *et al.*, 1984). Many pesticides target the nervous system of insect pests. Because of the similarity of neurochemical processes, these compounds are also likely to be neurotoxic to humans. This concern is of particular relevance to the developing human brain, which is inherently much more vulnerable to injury caused by toxic agents than the brain of adults (Dobling, 1968). Most types of pesticides, including organophosphates (OPs), carbamate, and organochlorine insecticides as well as fungicides and fumigants, can be neurotoxic, but only OPs have been studied in detail (Keifer and Mahurin, 1997). The response to OPs can occur within minutes. Less severe cases of OP poisoning display symptoms including headache, dizziness, nausea, vomiting, pupillary constriction, and excessive sweating, tearing, and salivation. More severe cases develop muscle weakness and twitches, bronchospasm, and changes in heart rate and can progress to convulsions and coma. Studies have also shown that prenatal exposure to a OPs reduces child IQ (Bouchard *et al.* 2011), impairs mental development

(Engel *et al.*, 2011), and causes cognitive deficits (Eaton *et al.*, 2008; Bouchard *et al.*, 2010; Rauh *et al.*, 2011).

Individual response to pesticide exposure may be affected by polymorphisms in genes affecting pesticide metabolism. The best-known example is paraoxonase, an enzyme that hydrolyzes active metabolites of OPs (Costa *et al.*, 2003). In humans, paraoxonase polymorphisms affect the relationship of OP exposure to both erythrocyte acetylcholinesterase (AChE) inhibition and symptom prevalence (Lee *et al.*, 2003; Mackness *et al.*, 2003; Sozmen *et al.*, 2002). Many studies have found an association of Parkinson disease risk with living in rural areas, drinking well water, and farming as an occupation (Priyadarshi *et al.*, 2001). Several studies found increased risk associated with exposure to either insecticides or herbicides (Gorell *et al.*, 1998, Mehdi *et al.*, 2013, Mehdi *et al.*, 2017, Mehdi and Qamar, 2011), and one study indicated that risk was elevated by exposure to organochlorines, OPs, or carbamates (Seidler *et al.*, 1996). Information on pesticide exposure and other neurologic diseases is more limited.

This review covers the background evidence and the epidemiological indication, on exposures of people and different experimental animals to pesticide and its consequences. These problems would tend to cause an satire of the true extent of the risks. The general experimental and epidemiological proposal suggests that the substantial vulnerability of low concentrations of pesticides should lead to a wired emphasis on protection of workers and general people who handle the pesticides. For both toxicological and epidemiological reasons, it is essential that potential of low-level, chronic exposure to pesticides and pesticide mixtures be determined. The available data suggests there is a high possibility for subtle adverse health effects. A cautionary principle in regard to pesticide toxicity should be applied in occupational health. On the basis of the above articles reviewed and reported observations we can say that the clinical and



experimental reports had increased the interest that these environmental toxin appears to be a promising tool to study the neurochemical process which is neurotoxic to humans and animals.

**Conflict of Interest:** No

**References:**

Akhtar Wasim MD, Sengupta D, Chowdhary A (2009) Impact of pesticides use in agriculture: their benefits and hazards. *Interdisc. Toxicol.* 2(1): 1–12.

Bolognesi C. (2003) Genotoxicity of pesticides: a review of human biomonitoring studies. *Mut. Res.* 543:251-272.

Bouchard MF, Bellinger DC, Wright RO, Weisskopf MG (2010) Attention-deficit/hyperactivity disorder and urinary metabolites of organophosphate pesticides. *Pediatrics.* 125:1270–1277.

Bouchard MF, Chevrier J, Harley KG, Kogut K, Vedar M, Calderon N, Trujillo C, Johnson C, Bradman A, Barr DB, Eskenazi B (2011) Prenatal Exposure to Organophosphate Pesticides and IQ in 7- Year Old Children. *Environ Health Perspect.* 119:1189–1195.

Dobbing J (1968) Vulnerable periods in developing brain. In *Applied Neurochemistry* Edited by: Davison AN, Dobbing J. Philadelphia: Davis, 287-316.

Engel SM, Wetmur J, Chen J, Zhu C, Barr DB, Canfield RL, Wolff MS (2011) Prenatal Exposure to Organophosphates, Paraoxonase 1, and Cognitive Development in Childhood. *Environ Health Perspect.* 119:1182–1188.

Environews Forum (1999) Killer environment. *Environ. Health Perspect.* 107: A62,

Erdman AR (2004) Insecticides. In: Dart RC (ed). *Medical toxicology*. 3rd ed. Philadelphia, PA:Lippincott Williams and Wilkins 1475–96.

- Forget G (1993) Balancing the need for pesticides with the risk to human health. In: Impact of Pesticide Use on Health in Developing Countries. Eds. Forget G, Goodman T and de Villiers A IDRC, Ottawa: 2.
- Gorell JM, Johnson CC, Rybicki BA, Peterson EL, Richardson RJ (1998) The risk of Parkinson's disease with exposure to pesticides, farming, well water, and rural living. *Neurology* 50:1346–1350.
- Gupta PK (2004) Pesticide exposure-Indian scene. *Toxicology* 198:83–90.
- Gupta SK, Jani JP, Saiyed HN and Kashyap SK (1984) Health hazards in pesticide formulators exposed to a combination of pesticides. *Indian J. Med.Res.*79:666.
- Igbedioh SO (1991) Effects of agricultural pesticides on humans, animals and higher plants in developing countries. *Arch. Environ. Health.*46: 218.
- Keifer M, Mahurin R (1997) Chronic neurologic effects of pesticide overexposure. *Occup. Med.* 12:291–304.
- Lee BW, London L, Paulauskis J, Myers J, Christiani DC (2003) Association between human paraoxonase gene polymorphism and chronic symptoms in pesticide-exposed workers. *J. Occup. Environ. Med.* 45:118–122.
- Mackness B, Durrington P, Povey A, Thomson S, Dippnall M, Mackness M, et al. (2003) Paraoxonase and susceptibility to organophosphorus poisoning in farmers dipping sheep. *Pharmacogenetics* 13:81–88.
- Mehdi SH, Qamar A. (2011) Neuropathological studies of *Drosophila melanogaster* with the sub-lethal doses of Malathion. *Biol and Med.* 3 (2): 265-269.
- Mehdi SH, Qamar A (2013). Paraquat-induced ultrastructural changes and DNA damage in the nervous system is mediated via oxidative-stress induced cytotoxicity in *Drosophila*

*melanogaster*. Toxicol. Sci. 134 (2): 355-365.

Mehdi SH, Qamar A, Zafaryab Md, Nafees S, Rizvi MMA (2017) Malathion–Induced cell injury and cell death in the Nervous System via Oxidative–Stress–Induced Cytotoxicity in *Drosophila melanogaster*. Intern. J. Scient. Res. 6(12): 447-450.

Priyadarshi A, Khuder SA, Schaub EA, Priyadarshi SS (2001) Environmental risk factors and Parkinson’s disease: a metaanalysis. Environ. Res. 86:122–127.

Rauh V, Arunajadai S, Horton M, Perera F, Hoepner L, Barr DB, Whyatt R (2011) 7-Year Neurodevelopmental Scores and Prenatal Exposure to Chlorpyrifos, a Common Agricultural Pesticide. Environ. Health Perspect. 119:1196–1201.

Seidler A, Hellenbrand W, Robra BP, Vieregge P, Nischan P, Joerg J, et al. (1996) Possible environmental, occupational, and other etiologic factors for Parkinson’s disease: a case-control study in Germany. Neurology 46:1275–1284.

Seidler A, Hellenbrand W, Robra BP, Vieregge P, Nischan P, Joerg J, et al. (1996) Possible environmental, occupational, and other etiologic factors for Parkinson’s disease: a case-control study in Germany. Neurology 46:1275–1284.

US EPA Pesticide Industry Sales and Usage: 1996 and 1997 Market Estimates, <http://www.epa.gov/oppbead1/pestsales/97pestsales>

WHO. Public Health Impact of Pesticides Used in Agriculture. (1990). World Health Organization, Geneva: 88.