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# Pesticides As Pollutants

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**ABSTRACT:** Pesticides are substances that are meant to control pests.[1] This includes herbicide, insecticide, nematicide, molluscicide, piscicide, avicide, rodenticide, bactericide, insect repellent, animal repellent, microbicide, fungicide, and lampricide.[2][3] The most common of these are herbicides, which account for approximately 50% of all pesticide use globally.[4] Most pesticides are intended to serve as plant protection products (also known as crop protection products), which in general, protect plants from weeds, fungi, or insects. As an example, the fungus *Alternaria solani* is used to combat the aquatic weed *Salvinia*.

In general, a pesticide is a chemical (such as carbamate) or biological agent (such as a virus, bacterium, or fungus) that deters, incapacitates, kills, or otherwise discourages pests. Target pests can include insects, plant pathogens, weeds, molluscs, birds, mammals, fish, nematodes (roundworms), and microbes that destroy property, cause nuisance, or spread disease, or are disease vectors. Along with these benefits, pesticides also have drawbacks, such as potential toxicity to humans and other species.

**KEYWORDS**-pesticides,pollutants,pests,crop,agriculture,toxicity,pathogens

## I.INTRODUCTION

Since before 2000 BC, humans have utilized pesticides to protect their crops. The first known pesticide was elemental sulfur dusting used in Sumer about 4,500 years ago in ancient Mesopotamia.[14] By the 15th century, toxic chemicals such as arsenic, mercury, and lead were being applied to crops to kill pests. In the 17th century, nicotine sulfate was extracted from tobacco leaves for use as an insecticide. The 19th century saw the introduction of two more natural pesticides, pyrethrum, which is derived from chrysanthemums, and rotenone, which is derived from the roots of tropical vegetables.[15] Until the 1950s, arsenic-based pesticides were dominant.[16] Paul Müller discovered that DDT was a very effective insecticide. Chlorinates such as DDT were dominant, but they were replaced in the U.S. by organophosphates and carbamates by 1975. Since then, pyrethrin compounds have become the dominant insecticide.[16] Herbicides became common in the 1960s, led by "triazine and other nitrogen-based compounds, carboxylic acids such as 2,4-dichlorophenoxyacetic acid, and glyphosate".[16]

The first legislation providing federal authority for regulating pesticides was enacted in 1910.[17] During the 1940s, manufacturers produced large amounts of synthetic pesticides and their use became widespread.[18] Before the first World War, Germany was the world's leading chemical industry and exported most of the dyes and other chemicals that were used in the United States. War implemented tariffs that stimulated the growth of the chemical industry in the U.S., which made chemistry a prestigious occupation as this industry expanded and became profitable. Money and ideas flowed back from Europe after the U.S. entered WWI, changing the way Americans interacted with themselves and nature, and the industrialization of war hastened the industrialization of pest control.[19] Some sources consider the 1940s and 1950s to have been the start of the "pesticide era."[20] Although the U.S. Environmental Protection Agency was established in 1970 and amendments to the pesticide law in 1972,[17] pesticide use has increased 50-fold since 1950 and 2.3 million tonnes (2.5 million short tons) of industrial pesticides are now used each year.[15] Seventy-five percent of all pesticides in the world are used in developed countries, but use in developing countries is increasing.[21] A study of USA pesticide use trends through 1997 was published in 2003 by the National Science Foundation's Center for Integrated Pest Management.[16][22]

In the 1960s, it was discovered that DDT was preventing many fish-eating birds from reproducing, which was a serious threat to biodiversity. Rachel Carson wrote the best-selling book *Silent Spring* about biological magnification. The agricultural use of DDT is now banned under the Stockholm Convention on Persistent Organic Pollutants, but it is still used in some developing nations to prevent malaria and other tropical diseases by spraying on interior walls to kill or repel mosquitoes.[23]

Pesticides may cause acute and delayed health effects in people who are exposed.[46] Pesticide exposure can cause a variety of adverse health effects, ranging from simple irritation of the skin and eyes to more severe effects such as affecting the nervous system, hearing,[47] mimicking hormones causing reproductive problems, and also causing cancer.[48] A 2007 systematic review found that "most studies on non-Hodgkin lymphoma and leukemia showed

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positive associations with pesticide exposure" and thus concluded that cosmetic use of pesticides should be decreased.[49] There is substantial evidence of associations between organophosphate insecticide exposures and neurobehavioral alterations.[50][51][52][53] Limited evidence also exists for other negative outcomes from pesticide exposure including neurological, birth defects, and fetal death.[54]

The American Academy of Pediatrics recommends limiting exposure of children to pesticides and using safer alternatives:[55]

Owing to inadequate regulation and safety precautions, 99% of pesticide-related deaths occur in developing countries that account for only 25% of pesticide usage.[56]

One study found pesticide self-poisoning the method of choice in one third of suicides worldwide, and recommended, among other things, more restrictions on the types of pesticides that are most harmful to humans.[57]

A 2014 epidemiological review found associations between autism and exposure to certain pesticides, but noted that the available evidence was insufficient to conclude that the relationship was causal.[58]

Occupational exposure among agricultural workers[edit]

The World Health Organization and the UN Environment Programme estimate that 3 million agricultural workers in the developing world experience severe poisoning from pesticides each year, resulting in 18,000 deaths.[59] According to one study, as many as 25 million workers in developing countries may suffer mild pesticide poisoning yearly.[60] Other occupational exposures besides agricultural workers, including pet groomers, groundskeepers, and fumigators, may also put individuals at risk of health effects from pesticides.[61]

Pesticide use is widespread in Latin America, as around US$3 billion are spent each year in the region. Records indicate an increase in the frequency of pesticide poisonings over the past two decades. The most common incidents of pesticide poisoning is thought to result from exposure to organophosphate and carbamate insecticides.[62] At-home pesticide use, use of unregulated products, and the role of undocumented workers within the agricultural industry makes characterizing true pesticide exposure a challenge. It is estimated that 50–80% of pesticide poisoning cases are unreported.

Underreporting of pesticide poisoning is especially common in areas where agricultural workers are less likely to seek care from a healthcare facility that may be monitoring or tracking the incidence of acute poisoning. The extent of unintentional pesticide poisoning may be much greater than available data suggest, particularly among developing countries. Globally, agriculture and food production remain one of the largest industries. In East Africa, the agricultural industry represents one of the largest sectors of the economy, with nearly 80% of its population relying on agriculture for income.[63] Farmers in these communities rely on pesticide products to maintain high crop yields.

Some East Africa governments are shifting to corporate farming, and opportunities for foreign conglomerates to operate commercial farms have led to more accessible research on pesticide use and exposure among workers. In other areas where large proportions of the population rely on subsistence, small-scale farming, estimating pesticide use and exposure is more difficult.

## II.DISCUSSION

Pesticides may exhibit toxic effects on humans and other non-target species, the severity of which depends on the frequency and magnitude of exposure. Toxicity also depends on the rate of absorption, distribution within the body, metabolism, and elimination of compounds from the body. Commonly used pesticides like organophosphates and carbamates act by inhibiting acetylcholinesterase activity, which prevents the breakdown of acetylcholine at the neural synapse. Excess acetylcholine can lead to symptoms like muscle cramps or tremors, confusion, dizziness and nausea. Studies show that farm workers in Ethiopia, Kenya, and Zimbabwe have decreased concentrations of plasma acetylcholinesterase, the enzyme responsible for breaking down acetylcholine acting on synapses throughout the nervous system.[64][65][66] Other studies in Ethiopia have observed reduced respiratory function among farm workers who spray crops with pesticides.[67] Numerous exposure pathways for farm workers increase the risk of pesticide poisoning, including dermal absorption walking through fields and applying products, as well as inhalation exposure.

Measuring exposure to pesticides

There are multiple approaches to measuring a person's exposure to pesticides, each of which provides an estimate of an individual's internal dose. Two broad approaches include measuring biomarkers and markers of biological effect.[68] The former involves taking direct measurement of the parent compound or its metabolites in various types of media: urine, blood, serum. Biomarkers may include a direct measurement of the compound in the body before it's been

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biotransformed during metabolism. Other suitable biomarkers may include the metabolites of the parent compound after they've been biotransformed during metabolism.[68] Toxicokinetic data can provide more detailed information on how quickly the compound is metabolized and eliminated from the body, and provide insights into the timing of exposure.

Markers of biological effect provide an estimation of exposure based on cellular activities related to the mechanism of action. For example, many studies investigating exposure to pesticides often involve the quantification of the acetylcholinesterase enzyme at the neural synapse to determine the magnitude of the inhibitory effect of organophosphate and carbamate pesticides.[68][64][65][66]

Another method of quantifying exposure involves measuring, at the molecular level, the amount of pesticide interacting with the site of action. These methods are more commonly used for occupational exposures where the mechanism of action is better understood, as described by WHO guidelines published in "Biological Monitoring of Chemical Exposure in the Workplace".[69] Better understanding of how pesticides elicit their toxic effects is needed before this method of exposure assessment can be applied to occupational exposure of agricultural workers.

Alternative methods to assess exposure include questionnaires to discern from participants whether they are experiencing symptoms associated with pesticide poisoning. Self-reported symptoms may include headaches, dizziness, nausea, joint pain, or respiratory symptoms.[65]

Challenges in assessing pesticide exposure

Multiple challenges exist in assessing exposure to pesticides in the general population, and many others that are specific to occupational exposures of agricultural workers. Beyond farm workers, estimating exposure to family members and children presents additional challenges, and may occur through "take-home" exposure from pesticide residues collected on clothing or equipment belonging to parent farm workers and inadvertently brought into the home. Children may also be exposed to pesticides prenatally from mothers who are exposed to pesticides during pregnancy.[70] Characterizing children's exposure resulting from drift of airborne and spray application of pesticides is similarly challenging, yet well documented in developing countries.[71] Because of critical development periods of the fetus and newborn children, these non-working populations are more vulnerable to the effects of pesticides, and may be at increased risk of developing neurocognitive effects and impaired development.[72][70]

While measuring biomarkers or markers of biological effects may provide more accurate estimates of exposure, collecting these data in the field is often impractical and many methods are not sensitive enough to detect low-level concentrations. Rapid cholinesterase test kits exist to collect blood samples in the field. Conducting large scale assessments of agricultural workers in remote regions of developing countries makes the implementation of these kits a challenge.[68] The cholinesterase assay is a useful clinical tool to assess individual exposure and acute toxicity. Considerable variability in baseline enzyme activity among individuals makes it difficult to compare field measurements of cholinesterase activity to a reference dose to determine health risk associated with exposure.[68] Another challenge researchers face in deriving a reference dose is identifying health endpoints that are relevant to exposure. More epidemiological research is needed to identify critical health endpoints, particularly among populations who are occupationally exposed.

## III.RESULTS

Pesticide use raises a number of environmental concerns. Over 98% of sprayed insecticides and 95% of herbicides reach a destination other than their target species, including non-target species, air, water and soil.[21] Pesticide drift occurs when pesticides suspended in the air as particles are carried by wind to other areas, potentially contaminating them. Pesticides are one of the causes of water pollution, and some pesticides are persistent organic pollutants and contribute to soil and flower (pollen, nectar) contamination.[73] Furthermore, pesticide use can adversely impact neighboring agricultural activity, as pests themselves drift to and harm nearby crops that have no pesticide used on them.[74]

In addition, pesticide use reduces biodiversity, contributes to pollinator decline,[75][76][77] destroys habitat (especially for birds),[78] and threatens endangered species.[21] Pests can develop a resistance to the pesticide (pesticide resistance), necessitating a new pesticide. Alternatively a greater dose of the pesticide can be used to counteract the resistance, although this will cause a worsening of the ambient pollution problem.

The Stockholm Convention on Persistent Organic Pollutants, listed 9 of the 12 most dangerous and persistent organic chemicals that were (now mostly obsolete) organochlorine pesticides.[6][79] Since chlorinated hydrocarbon pesticides dissolve in fats and are not excreted, organisms tend to retain them almost indefinitely. Biological

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magnification is the process whereby these chlorinated hydrocarbons (pesticides) are more concentrated at each level of the food chain. Among marine animals, pesticide concentrations are higher in carnivorous fishes, and even more so in the fish-eating birds and mammals at the top of the ecological pyramid.[80] Global distillation is the process whereby pesticides are transported from warmer to colder regions of the Earth, in particular the Poles and mountain tops. Pesticides that evaporate into the atmosphere at relatively high temperature can be carried considerable distances (thousands of kilometers) by the wind to an area of lower temperature, where they condense and are carried back to the ground in rain or snow.[81]

In order to reduce negative impacts, it is desirable that pesticides be degradable or at least quickly deactivated in the environment. Such loss of activity or toxicity of pesticides is due to both innate chemical properties of the compounds and environmental processes or conditions.[82] For example, the presence of halogens within a chemical structure often slows down degradation in an aerobic environment.[83] Adsorption to soil may retard pesticide movement, but also may reduce bioavailability to microbial degraders.[84]

Pesticides are often referred to according to the type of pest they control. Pesticides can also be considered as either biodegradable pesticides, which will be broken down by microbes and other living beings into harmless compounds, or persistent pesticides, which may take months or years before they are broken down: it was the persistence of DDT, for example, which led to its accumulation in the food chain and its killing of birds of prey at the top of the food chain. Another way to think about pesticides is to consider those that are chemical pesticides are derived from a common source or production method.[94]

Insecticides

Neonicotinoids are a class of neuro-active insecticides chemically similar to nicotine. Imidacloprid, of the neonicotinoid family, is the most widely used insecticide in the world.[95] In the late 1990s neonicotinoids came under increasing scrutiny over their environmental impact and were linked in a range of studies to adverse ecological effects, including honey-bee colony collapse disorder (CCD) and loss of birds due to a reduction in insect populations. In 2013, the European Union and a few non EU countries restricted the use of certain neonicotinoids.[96][97][98][99][100][101][102]

Organophosphate and carbamate insecticides have a similar mode of action. They affect the nervous system of target pests (and non-target organisms) by disrupting acetylcholinesterase activity, the enzyme that regulates acetylcholine, at nerve synapses. This inhibition causes an increase in synaptic acetylcholine and overstimulation of the parasympathetic nervous system.[103] Many of these insecticides, first developed in the mid 20th century, are very poisonous. Although commonly used in the past, many older chemicals have been removed from the market due to their health and environmental effects (*e.g.* DDT, chlordane, and toxaphene).[104][105][106] Many organophosphates do not persist in the environment.

Pyrethroid insecticides were developed as a synthetic version of the naturally occurring pesticide pyrethrin, which is found in chrysanthemums. They have been modified to increase their stability in the environment. Some synthetic pyrethroids are toxic to the nervous system.[107]

Herbicides

A number of sulfonylureas have been commercialized for weed control, including: amidosulfuron, flazasulfuron, metsulfuron-methyl, rimsulfuron, sulfometuron-methyl, terbacil,[108] nicosulfuron,[109] and triflusulfuron-methyl.[110] These are broad-spectrum herbicides that kill plants weeds or pests by inhibiting the enzyme acetolactate synthase. In the 1960s, more than 1 kg/ha (0.89 lb/acre) crop protection chemical was typically applied, while sulfonylureates allow as little as 1% as much material to achieve the same effect.[111]

## IV.CONCLUSIONS

Pesticide residue refers to the pesticides that may remain on or in food after they are applied to food crops.[136] The maximum allowable levels of these residues in foods are often stipulated by regulatory bodies in many countries. Regulations such as pre-harvest intervals also often prevent harvest of crop or livestock products if recently treated in order to allow residue concentrations to decrease over time to safe levels before harvest. Exposure of the general population to these residues most commonly occurs through consumption of treated food sources, or being in close contact to areas treated with pesticides such as farms or lawns.[137]

Many of these chemical residues, especially derivatives of chlorinated pesticides, exhibit bioaccumulation which could build up to harmful levels in the body as well as in the environment.[138] The problem is most acute in China, the largest

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producer of chlorinated pesticides.[139] Persistent chemicals can be magnified through the food chain and have been detected in products ranging from meat, poultry, and fish, to vegetable oils, nuts, and various fruits and vegetables.[140]

Pesticide contamination in the environment can be monitored through bioindicators such as bee pollinators.[73]

There is an ongoing research focused on pesticide residues in farming system

Minimizing harmful exposure to pesticides can be achieved by proper use of personal protective equipment, adequate reentry times into recently sprayed areas, and effective product labeling for hazardous substances as per FIFRA regulations. Training high-risk populations, including agricultural workers, on the proper use and storage of pesticides, can reduce the incidence of acute pesticide poisoning and potential chronic health effects associated with exposure. Continued research into the human toxic health effects of pesticides serves as a basis for relevant policies and enforceable standards that are health protective to all populations.

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