Review Article

Investigate the Effect of Pesticides on the Immune System Function

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Abstract

The use of pesticides in agriculture and pest control practices has led to environmental contamination, posing serious health threats to both humans and animals. This research aims to analyse the extent to which pesticides impose on the immune system function, focusing on their immunotoxic impacts. Pesticides have negative immunotoxic effects by hindering various stages of immune responses through immune suppression, allergic activity, autoimmune provocation and inflammatory stimulation. Furthermore, they may alter cytokine levels that increase the susceptibility to infections or chronic ailments. Besides these impacts, pesticide residues are found in food products of animal origin such as milk, meat and even internal organs, which raises concern beyond the direct immunotoxic effect. The chronic exposure to these substances may contribute to immune system deficits, endocrine disruption and possible cancer in animals and humans. In conclusion, pesticides are crucial for achieving increased productivity in farming, but endanger the health of humans and animals and weaken immune system function. As well as immunotoxic impacts due to prolonged exposure, it disables the activities of immune cells and inflicts non-functional toxicity through dependence on food containing animal products.

Keywords: Agricultural chemicals, animal health, immune system, immunotoxicity, pesticides

Introduction

Mammals possess an intricate and interrelated system of cells known as the immune system, which is divided into two types: antigen-non-specific (innate) and antigen-specific (adaptive). The role of the immune system is to protect other organ systems from invading pathogens, infectious agents and even cancer. It also aims at eliminating aged or modified cells like those in tumours, which are no longer fulfilling their roles(1).

Immune cells can be divided to a further level in depth, wherein lymphocytes encompass T ('T' refers to the thymus where T-cells mature and are differentiated into T lymphocytes), as well as B-cells and natural killer (NK) cells. T-cells or T lymphocytes have a major role in cellular immune response (2) and mature in the thymus gland. They can also be further divided into various subsets, such as helper, cytotoxic, memory and regulatory T-cells, based on their specific functions. Take T helper cells, for example, which can aid B-cells in forming plasma B-cells alongside and memory B-cells, as well as activating several B-cells, macrophages and cytotoxic T-cells (alternatively referred to as killer T-cells). These Cytotoxic T-cells (killer T-cells) have an important function of destroying infected and tumourigenic cells, which are synonymous to virus-infected cells. B-cells, also known as B lymphocytes, are produced in the liver and bone marrow. B-cells are remarkable for mediating humoral immunity as well, but primarily do so by producing antibodies after 'activation' (which can be T-dependent or T-independent)(3). NK cells form another subgroup of lymphocytes. These are phagocytic lymphocytes responsible for developing in the liver, lymph nodes and bone marrow. Through a swift immune response, NK cells have the ability to destroy infected or cancerous cells through numerous diverse functions aimed at those cells(4). Macrophages are mononuclear phagocytic cells that are created through the differentiation of, in this case, bone

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marrow monocytes. These versatile cells reside in nearly all tissues where they play a crucial role in phagocytosis during immune responses(5).

Pesticides are utilised to eliminate insects, rodents, fungi and unwelcomed plants using specific chemical compounds(6). This group includes insecticides, herbicides, nematicides, fungicides, molluscicides, rodenticides and even plant growth regulators(7). These chemicals are mainly used to contain diseases caused by vectors and are crucial in safeguarding crops as well as preserving food(8). They hold value in many industrial fields including aquaculture, agriculture and food processing and storage(9). Any organism that is harmful and dangerous to human and animal life whether in plant or animal form, is labelled as a pest. Pesticides aim at the reduction or total extermination of pests. It also affects animals on various levels, which include: the multi-system, the reproductive system, the nervous system and the immune system(10). The purpose of this review is to analyse the effects of pesticides on the immune system.

Sources of Animal Pesticide Residues

Food contamination, the use of insecticides and disinfectants during production, the use of insecticides for pest control in living organisms and *in vitro*, and the use of herbicides to control weeds in breeding environments are some of the factors that lead to the presence of pesticide residues in animal-derived food products. Because pesticides are used so extensively in agriculture, they can be carried and cycled through a variety of environmental media, including water, soil and air(11). During breeding, these residues have the potential to enter animal bodies, where they may disperse throughout various tissues and undergo metabolic changes. In the end, this procedure leads to the identification of these residues in animal-based foods(12). Yes, some major differences between animal and human immune systems can affect how pesticides affect immunity. These differences must be taken into account very carefully when extrapolating from animal tests to human health risk assessment. Below are some points to note, with references:

Differences between Animal and Human Immune Systems and Their Impact on Pesticide Studies

Species-specific immune responses

Vertebrates possess a heterogeneous immune system architecture, immune cell composition and cytokine profile. For example, mice utilised in the majority of pesticide studies possess more lymphocytes compared to humans, who possess higher levels of circulating neutrophils. These differences result in heterogeneous inflammatory or immunosuppressive responses to the same pesticide exposure(13).

Metabolism and detoxification

Pesticide metabolism is widely different between species due to variations in the level of activity of liver enzymes (e.g., cytochrome P450 enzymes), which can alter the pesticides' immune-toxic capability. Rodents can metabolise certain pesticides more rapidly or more slowly than human beings, influencing the immune response indirectly through the generation of systemic toxicity(14).

Dose and exposure differences

Laboratory animals are typically exposed to higher or more controlled doses than typical human environmental exposures, which might exacerbate or suppress immune effects(15).

Immunological development and lifespan

The animal immune system develops in a different way and at an accelerated rate compared to humans. This can influence developmental immunotoxicity assessments and cannot fully predict long-term effects in humans(14).

Mechanisms of Toxicology of Pesticides in Immunology

Immunosuppressive effects

Pesticides exhibit an immunosuppressive effect on the immune system, as indicated in [Table 1].

Allergic responses (hypersensitivity)

Exposure to pesticides has increasingly been defined as leading to allergic responses and respiratory complications. Organophosphates have been found to cause induction and exacerbation of asthma through autonomic dysregulation, contact dermatitis among those exposed, delayed hypersensitivity, and inhibition of acetylcholinesterase (AChE) activity (22-23) as showed in Table 2. Pyrethrins have been found to induce immunoglobulin (Ig) E-mediated allergy, like skin contact allergy, asthma and allergic rhinitis, presumably through haptenisation and mast cell degranulation; elevated serum IgE has been reported in occupationally exposed workers(23,24). Chemicals such as heptachlor, parathion and ethylene dibromide are implicated in asthma pathogenesis and atopic reaction, also through AChE inhibition(25). Exposure to pesticides by chemicals such as glyphosate, diazinon, chlorpyrifos and carbaryl has been linked to elevated rates of rhinitis symptoms among farm workers(26) [Figure 1]. Similarly, chemicals such as paraquat, dipyridyl, dithiocarbamate and CB-carbamates are implicated in respiratory symptoms through mechanisms that involve AChE inhibition(27). Malathion and its metabolites cause IgE-mediated immune responses, with both the presence of specific IgE antibodies in model animals and rare human instances(28). In addition, exposure to residual oil fly ash and carbaryl increases IgE responses to common allergens like house dust mites, illustrating an exaggerated humoral immune response(29). Finally, prenatal exposure to pesticides has also been associated with increased vulnerability to asthma and allergy symptoms during childhood, which emphasises the transgenerational impact of pesticide-evoked immunotoxicity(30-33) as mentioned in Table 2. Collectively, the previous studies indicate the broad spectrum of allergic

Table 1: Immunotoxic effects of pesticides and their mechanisms of action on the immune system

Pesticide	Immunotoxic effects on the immune system	Mechanism of action	Practical uses	Reference
Atrazine (ATR)	Inhibits CD4+ T-cell proliferation Reduces CD3+ T-cells in the spleen Lowered IgM production, altered Th1/Th2 balance Inhibits B-cell proliferation, reduces B-blasts, lowers antibody production Decreased lytic granule release, impaired cytotoxicity	Interferes with cell viability and development through oxidative stress, ROS production and mitochondrial dysfunction Causes DNA damage and genotoxic effects in lymphocytes Endocrine disruption Interferes with granule exocytosis	Used as a herbicide to control broadleaf and grassy weeds in crops such as corn, sorghum and sugarcane	(16-18)
Carbamates	Induces apoptosis in T-cells Inhibits IL-2 production Inhibits cell proliferation Reduced spleen weight, thymus involution, decreased antibody titres Reduced perforin and granzyme levels	Inhibits acetylcholinesterase Interferes with lymphocyte-specific kinases Reduces cytokine signalling Bone marrow suppression, oxidative stress Suppressed cytotoxic machinery	Used as insecticides in agriculture and public health to control pests on crops, livestock and in homes	(19)
Organophosphates	Suppresses CTL activity Reduced macrophage function, suppressed NK cell activity Induces plasma cell apoptosis, depresses humoral response Inhibited lytic function without cell death Decreases T-cell populations in a dose-dependent fashion Decreased T-cell proliferation, decreased IL-2 and IFN-γ levels	Affects perforin/granzyme and Fas/FasL mechanisms Lipophilic accumulation and mitochondrial impairment Mitochondrial dysfunction, ER stress Decreased ATP, perforin, granzyme, granulysin General immunomodulation (possibly via oxidative stress and AChE inhibition) Affects CD4+ Th1 response	Commonly used insecticides for pest control in agriculture, households and vector-borne disease control	(20,21)

CTL: Cytotoxic T lymphocyte, ROS: Reactive oxygen species, NK: Natural killer, AChE: Acetylcholinesterase, ER: Endoplasmic Reticulum, IL-2: Interleukin-2, IFN-γ: Interferon-gamma, IgM: Immunoglobulin M, ATP: Adenosine Triphosphate, ATR: Atrazine

Table 2: Type and mechanism of hypersensitivity and clinical manifestations

Type	Mechanism	Pesticide examples	Clinical manifestations	References
Type I (immediate/IgE-mediated)	Allergen exposure triggers IgE antibodies, leading to mast cell degranulation and histamine release	Pyrethrins, Malathion, Carbaryl	Asthma, allergic rhinitis, anaphylaxis, skin hives, IgE elevation	(31)
Type IV (delayed-type/cell-mediated)	T-cell-mediated immune response occurs hours or days after exposure	Organophosphates, Glyphosate, Paraquat	Contact dermatitis, eczema and delayed asthma exacerbation	(32)
Type II (cytotoxic) (less common)	Antibody-mediated cell destruction, usually via IgG or IgM	Rare in pesticide exposure, but can occur in chronic high-dose exposure scenarios	Autoimmune-like hemolysis or cytopenias (very rare)	(33)
Type III (immune complex-mediated) (rarely implicated)	Immune complexes deposit in tissues, triggering inflammation	Some reports with long-term organochlorine exposure	Vasculitis, serum sickness-like symptoms	(31)

Ig: Immunoglobulin

Table 3: Autoimmune effects and the mechanism of action induced by pesticides

Pesticide	Autoimmune effect	Mechanism action	References
Organochlorines (e.g., DDT)	Lupus-like symptoms, ANA positivity	B-cell hyperactivity occurs via disruption of immune regulation and oxidative stress mechanisms through ROS-mediated damage and Altered cytokine signalling	(38,39)
Triazine herbicides (e.g., atrazine)	Altered cytokine balance leading to SLE-like features	Estrogenic modulation of immune cells occurs by their endocrine-disrupting actions, which either mimic or block typical oestrogen signalling through binding to oestrogen receptors. Immune Cell imbalance and altered cytokine production	(38,39)
Glyphosate	Associated with autoimmune thyroid disease, IBD	Chronic inflammation and Treg suppression	(38,39)

ROS: Reactive oxygen species, SLE: Systemic lupus erythematosus, ANA: Antinuclear antibodies, DDT: Dichloro-diphenyl-trichloroethane, IBD: Inflammatory bowel disease

and respiratory endpoints elicited by pesticide exposure through mediation by immune dysregulation, cholinergic impairment and IgE-dependent hypersensitivity [Figure 1].

Autoimmune effects

Autoimmune rheumatic diseases (ARD) such as rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) are

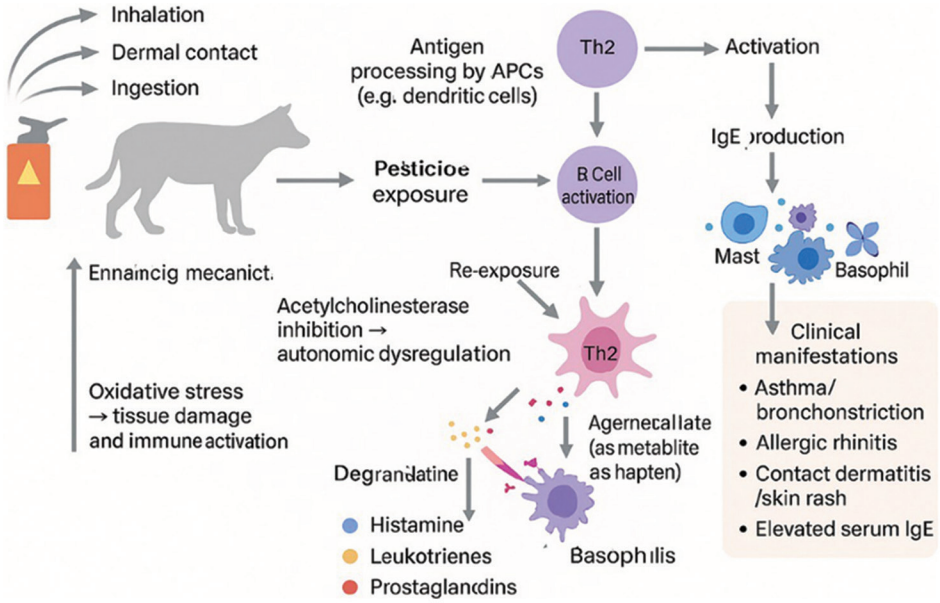

Figure 1: Allergic response to pesticides in animals

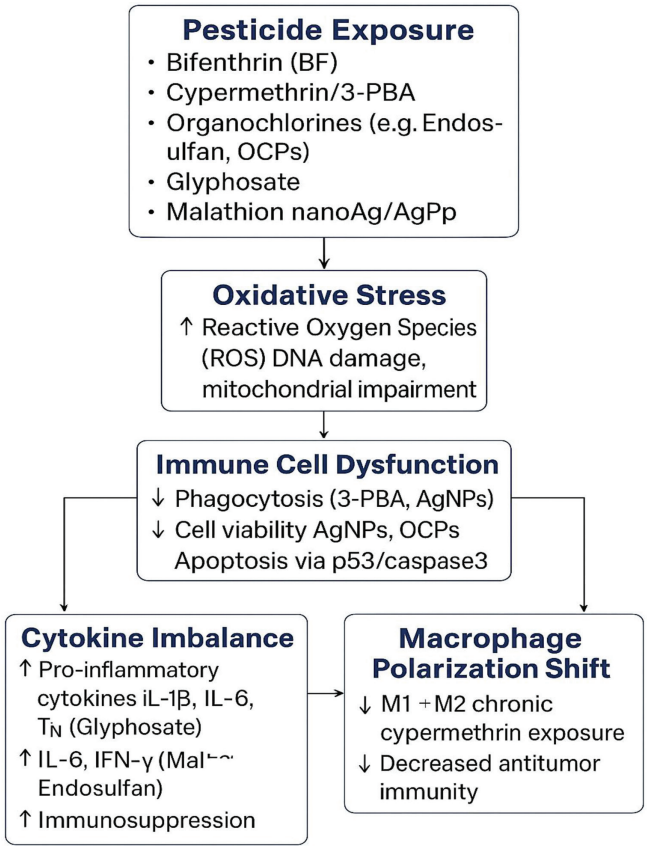

Figure 2: Inflammation response and cytokine modulation. IL-2: Interleukin-2, IFN-γ: Interferon-gamma

influenced by genetic and environmental factors, including family history, smoking and occupational exposure(34). Farming is particularly linked to ARD, with pesticide exposure identified as a risk factor, especially for men. Studies showed that high serum organochlorine pesticides (OCPs) correlate with self-reported RA, while pesticide mixing in farmers is associated with SLE. The previous research suggests that agricultural pesticides may either increase or decrease the risk of SLE/Sjogren's syndrome, indicating a complex interplay of factors, including childhood exposure(35,36). Animal models are informative regarding how pesticides may trigger autoimmune reactions: Dichloro-Diphenyl -Trichloroethane (DDT): Mouse experiments identify lupus-like characteristics, including the production of antinuclear antibodies. Atrazine: Rodents exhibit compromised cytokine balance and SLE-like presentation. Glyphosate: Associated with autoimmune thyroiditis by inflammation and suppression of Treg(37). The impact of pesticides on autoimmune diseases is detailed in Table 3.

Inflammatory responses and cytokine modulation

Macrophages, the leading cells of the innate immunity, play a crucial role in modulating pro- and anti-inflammatory responses. They recognise pathogens, phagocytize and produce cytokines, initiating adaptive immunity by presenting antigens to T-cells(40,41) [Figure 2]. Multiple pesticides disrupt immune function and cell viability via oxidative stress and inflammation. Bifenthrin: suppresses cell viability and pro-inflammatory cytokines (interferon-gamma [IFN-β], interleukin [IL]-1β, IL-6, tumour necrosis factor-alpha [TNF-α]) and promotes apoptosis via p53/caspase-3 pathways. Cypermethrin: increases reactive oxygen species, which cause DNA damage and mitochondrial impairment(42,43) [Figure 2]. Chronic exposure skewers macrophage polarisation to M2, reducing M1 and increasing cancer metastasis. 3-Phenoxybenzoic acid, cypermethrin's metabolite, disables immune function by suppressing phagocytosis. Silver nanoparticles and OCPs inhibit cell viability and phagocytic activity and increase nitric

oxide, resulting in synergistic immunosuppression(44,45). Glyphosate activates pro-inflammatory responses in macrophages by increasing IL-1 β, TNF-α and IL-6 through upregulation. Malathion augments inflammatory responses in lymphoid tissue by enhancing the levels of IL-6 and IFN-γ. Endosulfan disturbs cytokine homeostasis by decreasing IL-12 and increasing IL-10, exhibiting the immunomodulatory potential of pesticides(46,47).

Conclusions

Pesticides offer a range of advantages through the regulation of organisms that are regarded as harmful, leading to higher crop yield and quality, effective disease management and cost savings in terms of commodity protection against rot. They can, however, exert a significant impact on immunocompetence through a range of mechanisms: Immunosuppression or overactivation of immune cells, disruption of cytokine networks, triggering of allergic and autoimmune reactions, enhanced infection susceptibility and chronic disease. These effects are especially significant in workplace settings, among children in less developed nations, within individuals with weakened immune systems and across animal groups.

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Conflicts of interest

There are no conflicts of interest.

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