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Pesticides and their Cytogenetic Effects on DNA: A Comprehensive Review

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Abstract: Although necessary for increased agricultural yield, pesticides present serious cytogenetic hazards to people. The impact of its extensive use on the integrity of DNA has raised concerns. In this review, the toxicokinetics, cytogenetic effects, exposure pathways, and classification of pesticides are reviewed. It is possible for pesticides to enter the body through the eyes, nose, lips, or through other routes. Absorption, distribution, metabolism, and excretion are all part of their toxicokinetic processes. SCEs, MN production, chromosomal abnormalities, and DNA strand breaks are indicators of cytogenetic effects. By directly interacting with DNA, oxidative stress, and inhibiting DNA repair, pesticides can cause such effects. Exposure to pesticides has been linked to a higher risk of cancer and issues with reproductive health, as per epidemiological research. Reducing the detrimental effects of pesticides can be accomplished through the use of mitigation measures such biopesticides, landscape mitigation, and biotechnological breakthroughs.

Keywords: Pesticides, Cytogenetic, Micronucleus, Sister chromatid exchange, Genotoxicity.

1. Introduction

Chemicals known as pesticides are used to eliminate weeds, fungi, insects, rodents, and other unwanted organisms (Ramchandar et al. 2025). Figure 1 represents the agricultural consumption of pesticides in leading countries during the year 2021 (Pesticide Consumption Worldwide 2021 | Statista, 2023). Despite their extensive use, their potentially harmful consequences must be assessed (Ahmad *et al.,* 2024). Only 1% of their yearly application which is estimated to be over 4 million tons reaches the intended pests (Al-Attar *et al.,* 2018). They continue to be the most

widely used and reliable method of protecting plants from pests, and their use has greatly increased crop yields and agricultural productivity (Sharma *et al.,* 2019). But because of their widespread use, there are now major concerns about possible negative effects on non-target organisms, such as humans. Pesticides' impacts on genetic material, especially their cytogenetic effects on DNA, are among the most serious health issues. Research indicates that agricultural laborers who are subjected to pesticide exposure show increased DNA damage, such as chromosomal translocations and abnormalities (Sherif et al*.,* 2024).

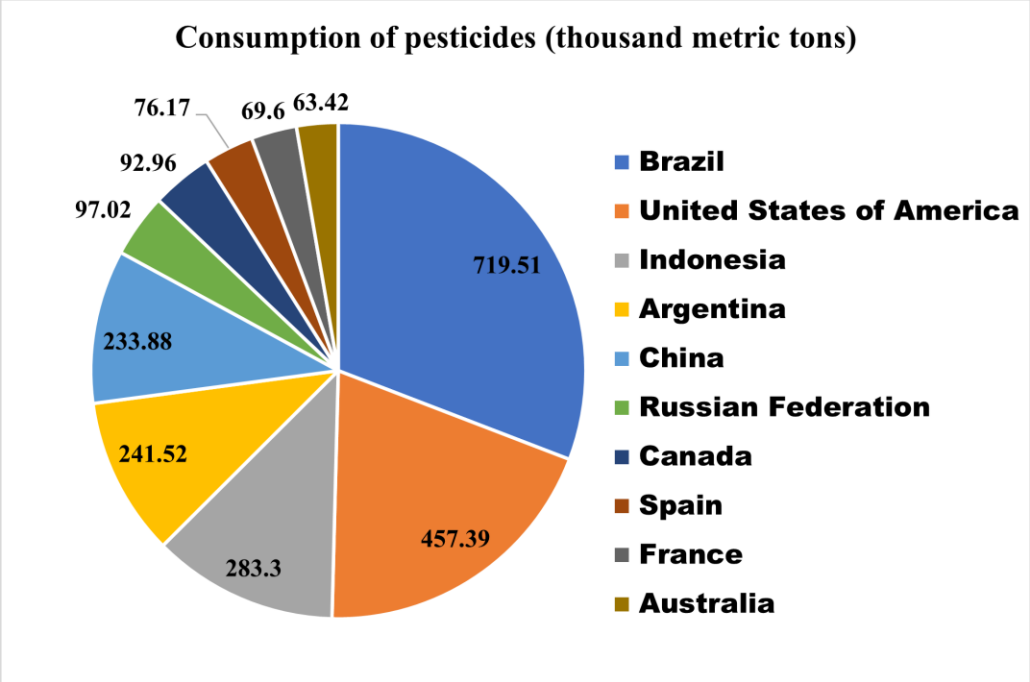

Figure. 1: Leading countries in agricultural consumption of pesticides worldwide in 2021

2. Classification of pesticides

Pesticides can be classified on the basis of target organism and their chemical structure.

2.1. Classification on the basis of the target organisms (Ali, 2023):

On the basis of the target organisms, pesticides may be classified as

- **Fungicides** are pesticides that kill or incapacitate fungus, protecting plants from fungal infections.
- **Herbicides** are employed to kill weeds. A weed is a plant that hinders crop plant growth.
- **Insecticides** are used to eradicate insects

- **Rodenticides** fight against rodents and rats,
- **Avicides** control unwanted bird populations.
- **Nematicides** are used to prevent nematodes.

2.2. Classification on the basis of chemical structure

Pesticides are categorized into four classes based on their chemical composition: organochlorines, organophosphorus, carbamates, and pyrethroids (Kaur et al. 2019).

I. Organophosphate

These are the esters of phosphoric acid. They have an effect on the neurological system by inhibiting the enzyme that controls acetylcholine, a neurotransmitter (Kaur *et al.,* 2019). Organophosphate insecticides include methyl parathion, malathion, phosphamidon, and chlorpyriphos. (Adeyinka *et al.,* 2023).

II. Carbamate pesticides

These are carbamic acid derivatives. Carbamate insecticides act in same fashion as organophosphate pesticides by interfering with nerve signal transmission, resulting in the pest's death by poisoning (Kaur *et al.,* 2019). Examples of carbamate insecticides include carbaryl, carbofuran, and carbosulfan (Mdeni *et al.,* 2022).

III. Organochlorine pesticides

Organochlorines represent a category of manmade organic compounds encompassing renowned pesticides including DDT, Chlordane, Endosulfan, and Lindane. These substances are distinguished by their chlorine constituents and have been extensively utilized in agricultural practices and pest management (Vasanth et al., 2024).

IV. Pyrethroid pesticides

Pyrethroids represent synthetic analogs of naturally occurring pyrethrin insecticides, which are extracted from the flowers of *Chrysanthemum cinerariaefolium*. The pyrethroids exert their impact on living cells by disrupting the proper operation of voltage-gated sodium channels. Notable examples of pyrethroid pesticides include deltamethrin, cypermethrin, and permethrin (Shukla, 2022).

3. Routes of pesticide's exposure

Pesticide toxicity is determined by its absorption into the body as well as the exposed individual's characteristics. Pesticides may penetrate the body via oral ingestion; through dermal exposure (skin contact); through respiratory inhalation; or through ocular exposure (eyes) (Tharp, 2019). Figure 2 shows the various route of pesticides exposure.

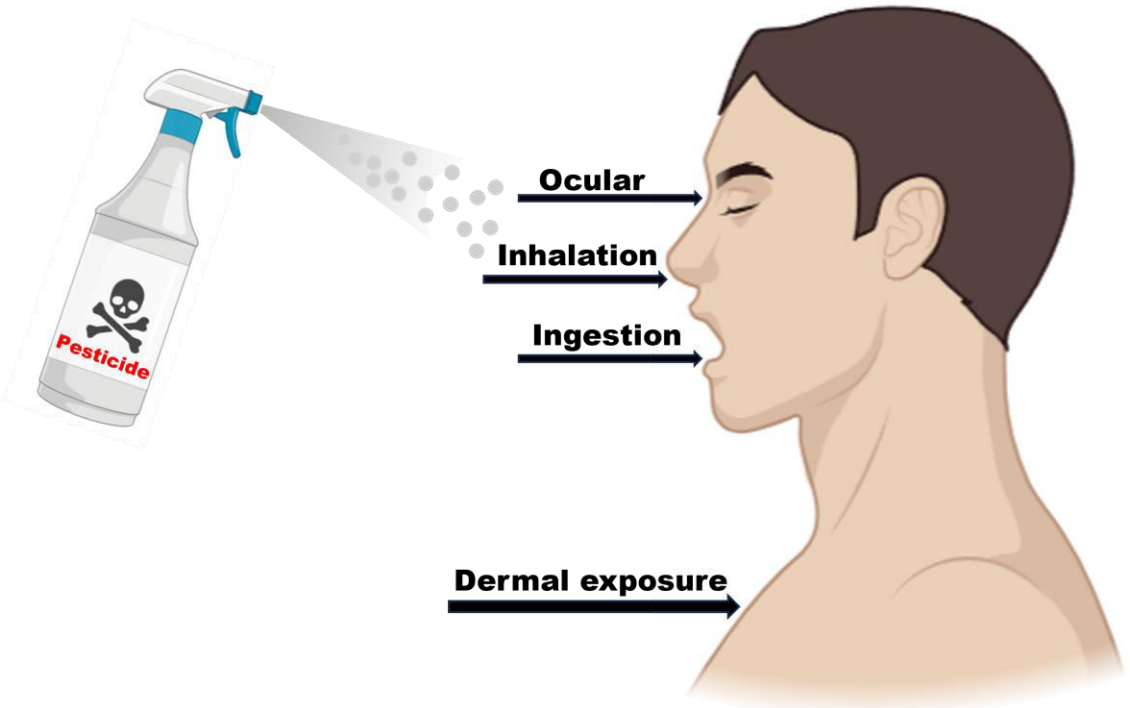

Figure 2: Different routes of pesticide's exposure

- **Ingestion:** Pesticide ingestion is common among children, although it can also occur due to negligence.
- **Dermal exposure:** Approximately 90 percent of pesticide exposure is attributed to the dermal route. The degree of absorption through the skin is contingent upon various factors, including the specific anatomical site of pesticide application, the extent of the contaminated area, the duration of contact with the skin, and the quantity of pesticide present on the dermal surface. For example, Table 1 illustrates the absorption rates of parathion via the dermal route across various anatomical regions of the body:

Table 1: Parathion absorption rates through the skin on body regions over a 24-hour period. (Tharp, 2019).

Body Region	Percent Relative Absorption
Forearm	8.6
Palm of Hand	11.8
Ball of Foot	13.5
Abdomen	18.4
Scalp	32.1
Forehead	36.3
Ear Canal	46.5
Genitalia	100.0

- **Inhalation exposure:** Inhalation exposure is often caused by airborne pesticides such as dusts and fumigants. Pesticides easily enter the bloodstream after being inhaled, causing poisoning symptoms.
- **Ocular exposure:** This particular form of exposure induces permanent harm to the ocular structures. Ocular exposure is frequently a consequence of splashing incidents that occur during the processes of mixing and cleaning, particularly in the absence of appropriate protective eyewear.

4. Sources of pesticide's exposure

Pesticide exposure occurs from a variety of sources, having a substantial influence on both human health and the ecosystem. The exposure to pesticides can be classified into two primary categories, namely occupational exposure and non-occupational exposure.

4.1. Occupational exposure: Occupational exposure to pesticides refers to the exposure that results from an individual's profession. Occupational exposure to pesticides occurs predominantly within agricultural environments wherein farmers and agricultural laborers engage with deleterious chemicals via inhalation, dermal absorption, or inadvertent ingestion. Empirical research highlights that these risks entail both physiological and psychological health consequences. For example, a research investigation conducted in Morocco examines the endocrine and epigenetic modifications in agricultural laborers, emphasizing that safety measures are frequently ignored, leading to variations

in hormone concentrations and alterations in DNA methylation due to pesticide exposure (Chbihi *et al.,* 2024).

Agricultural workers encounter elevated levels of exposure to pesticides, such as organophosphates (OPs) and pyrethroids, owing to direct interaction with these substances during their application, frequently without sufficient protective gear. These workers had higher amounts of pesticide metabolites in their bodies than non-farmworkers, indicating an elevated risk of acute and chronic health problems (Bravo *et al.,* 2022).

In addition to the agricultural sector, professions involved in pesticide manufacturing and formulation expose employees during the production phase, particularly in settings where chemicals are blended or stored, as evidenced in industries that process pest control substances (Carles *et al.,* 2018). Public health professionals engaged in activities such as disinfection and rodent eradication frequently encounter pesticides, especially in metropolitan regions, thereby presenting chronic health hazards (Colosio & Chandrasekar, 2024).

4.2. Non-occupational exposure: Non-occupational exposure to pesticides denotes exposure that does not arise from an individual's profession. This form of exposure may occur through the application of pesticides in domestic settings, consumption of contaminated food products, and proximity to agricultural endeavors (Ottenbros *et al.,* 2023). Individuals living in rural locales encounter pesticides through both particulate and gaseous states in indoor and outdoor settings, with research indicating comparable levels of exposure between bystanders and those employed in farming (Mus *et al.,* 2024). Another major contributor to pesticide exposure is the pollution of water, where agricultural runoff contaminates both surface and groundwater with pesticide residues. These tainted water supplies become a route for pesticide ingestion, particularly in rural communities (Chen *et al.,* 2022). Children, especially in developing areas, face heightened risks of exposure in their homes and schools, where pesticides are deployed for pest management, leading to severe acute and chronic health consequences (Karr & Rauh, 2024).

5. Toxicokinetics of pesticides

The study of a pesticide's absorption, distribution, metabolism, and excretion over time is known as its toxicokinetics. This information is essential for determining the dangers of pesticide exposure and for creating practical safety precautions. Toxicokinetic analysis of different pesticide classes sheds light on the effects of these

chemicals on people, particularly with regard to their absorption, distribution, metabolism, and excretion (ADME) processes.

5.1. Absorption and Distribution

Pesticides infiltrate the human body through dermal, inhalation, or oral pathways. Their distribution is heavily influenced by their chemical characteristics, such as lipophilicity, which facilitates accumulation in fatty tissues. Organophosphates (OPs), including chlorpyrifos, disrupt the nervous system by blocking acetylcholinesterase (AChE), leading to neurotoxicity. In instances of acute poisoning, chlorpyrifos displays the highest concentrations in the bloodstream, with rapid elimination phases noted in monocrotophos and dimethoate (Sinha, 2021). Likewise, in insects like Spodoptera littoralis, the uptake of insecticides is shaped by their feeding habits, with hydrophobic compounds demonstrating greater bioaccumulation potential (Römer *et al.,* 2024).

5.2. Metabolism of Pesticides: Role of Cytochrome P450

The metabolism of pesticides is profoundly influenced by cytochrome P450 (CYP) enzymes, which drive the transformation of these compounds into far less harmful forms. This process is vital for eliminating environmental pollutants, including pesticides, and accelerating their removal from the organism. For example, CYP3A4 and CYP2C19 are instrumental in the bioactivation of organophosphates and carbamate pesticides, amplifying their toxic potential. Investigations using human and animal in vitro hepatic models revealed that CYP enzymes convert profenofos and carbosulfan into extremely toxic metabolites, like carbofuran, underscoring the enzyme's critical role in shaping the overall toxicological consequences (Abass, 2010).

5.3. Elimination and Excretion of Pesticides

The process of eliminating pesticides from the human body is significantly contingent upon their metabolic pathways and solubility characteristics. Hydrophilic metabolites are expelled principally through the urinary system, whereas lipophilic substances tend to be eliminated at a more gradual pace via fecal matter. The half-lives of various pesticides exhibit considerable variability, thus affecting their duration of presence within the organism and their long-term toxicological implications. Certain OPs, like chlorpyrifos, are more stable and may cause longer exposure and chronic toxicity, but monocrotophos and dimethoate have shorter elimination phases, decreasing their persistence (Sinha, 2021). Furthermore, research indicates that organochlorine pesticides are predominantly excreted via

urine, sweat, and blood, with sweat displaying elevated detection rates for specific compounds (Genuis *et al.,* 2016).

6. Toxicity of Pesticides

The toxicity of a pesticide refers to its inherent capacity or potential to induce harm or illness. The toxicity associated with pesticides constitutes a significant concern, given their prospective detrimental effects on humans, animals, and ecological systems. The toxicity level of a specific pesticide is ascertained through the administration of diverse dosages of the active ingredient to test animals, alongside assessments of each of its formulated products. The active ingredient represents the chemical compound within the pesticide formulation that exerts control over the targeted pest species. The two types of toxicity are categorized as acute and chronic.

I. Acute toxicity

Acute toxicity is defined as the immediate detrimental effects resulting from a single or brief exposure to pesticides, with symptoms generally presenting within a 24-hour period post-exposure. Organophosphorus (OP) pesticides, which are extensively utilized in agricultural practices, are a significant contributor to instances of acute poisoning. These compounds irreversibly bind to acetylcholinesterase (AChE), leading to disruptions in neurotransmission and the onset of neurotoxic consequences (Van Melis *et al.,* 2022). The degree of acute toxicity is not uniform across pesticides, as exemplified by dichlorvos, which demonstrates a higher toxicity level in comparison to substances such as lindane. In a research study conducted on African catfish, dichlorvos was identified as being 50 times more toxic than lindane, thereby emphasizing the variability in toxicity profiles among different pesticide agents (Oribhabor & Ikeogu, 2016). The assessment of acute toxicity predominantly employs the median lethal dose (LD50), a metric that quantifies the concentration of a toxicant necessary to result in the mortality of 50% of a test population. The LD50 value serves as a fundamental parameter in acute toxicity evaluations, indicating the dosage at which half of the test subjects (typically animal models) perish as a result of the toxicant (Li *et al.,* 2024). This quantification is essential in toxicological assessments, facilitating safety evaluations for a range of chemical substances.

II. Chronic toxicity

Chronic toxicity pertains to the detrimental effects that arise from sustained or recurrent exposure to pesticides over an extended duration, frequently at sublethal doses. This category of toxicity bears considerable long-term consequences for both

human health and ecological systems. Prolonged exposure to pesticides has been associated with a range of serious health complications, including neurodegenerative disorders such as Alzheimer's disease and Parkinson's disease, as these substances commonly disrupt the functionalities of the nervous system (Arab & Mostafalou, 2021). Furthermore, bioinformatics investigations suggest that long-term exposure to pesticides modifies gene expression, particularly influencing pathways pertinent to neurodevelopment and the regulation of movement, as evidenced in agricultural workers subjected to these chemicals (Jiang *et al.,* 2022).

7. Cytogenetic effects of pesticides on DNA

Through the study of chromosomal structures and their corresponding dynamics, cytogenetics furnishes a vital insight into the effects that pesticides enact on DNA. A variety of cytogenetic anomalies, including Sister Chromatid Exchange (SCE), the formation of micronucleus (MN), chromosomal aberrations (CA), and breaks in DNA strands, may be induced by exposure to pesticides. These cytogenetic abnormalities not only serve as indicators of genetic damage but are also linked to various pathologies, including cancer and reproductive health issues (Bolognesi, 2003).

7.1. Induction of SCE

The reciprocal exchange of DNA segments between sister chromatids during the mitotic phase is referred to as SCE. SCE occurs during the process of DNA replication, wherein segments of genetic material are exchanged between sister chromatids, frequently visualized through methodologies such as fluorescence plus Giemsa (FPG) staining or bromodeoxyuridine (BrdU) labeling (Rampias & Klinakis 2023) (Russell 2022). Increased frequencies of SCE have been associated with a variety of pathological conditions, including type II diabetes mellitus and breast carcinoma, thereby indicating a potential relationship between SCE and genomic instability (Ghali 2022).

SCE frequency serves as a critical biomarker for assessing the genotoxicity of pesticides, as it effectively reflects genetic instability and inaccuracies in DNA repair mechanisms (Alarcón, *et al.,* 2021). Elevated SCE levels have been consistently associated with exposure to pesticides. Empirical evidence suggests that glyphosate markedly increases SCE levels in human lymphocytes, especially at elevated concentrations (200 μmol/L) (Tarboush *et al.,* 2022). Furthermore, Organophosphate pesticides, which have been extensively researched in Latin America, exhibit correlations with genotoxicity, thereby indicating their potential to induce SCE (Zúñiga-Venegas *et al.,* 2022). An investigation into tetrachlorvinphos demonstrated a dose-responsive escalation in SCE values, particularly at higher concentrations (50

μM), which underscores its genotoxic potential (Akgün & Çobanoğlu 2024). A meta-analysis indicated that the average SCE frequency among individuals exposed to pesticides was significantly greater (7.88) than that observed in control groups (6.05), thereby implying that SCE frequency may serve as an early biomarker for genetic damage within exposed populations (Yang *et al.,* 2014).

7.2. Induction of CA

The cytogenetic consequences of pesticides that have been extensively studied include chromosomal anomalies, which consist of structural modifications such as deletions, duplications, inversions, and translocations. These pesticides have the potential to instigate such anomalies either directly via interactions with DNA or indirectly through the generation of reactive oxygen species (ROS) (Ahmad *et al.,* 2024). Empirical evidence suggests that pesticides can provoke chromosomal instability and influence the expression of genes associated with DNA repair, culminating in irreversible chromosomal damage (Costa *et al.,* 2021). In Colombia, agricultural workers subjected to pesticide exposure exhibited elevated frequencies of chromosomal instability and alterations, particularly among individuals possessing certain genetic polymorphisms, signifying an amplified risk of genomic impairment (Aldana-Salazar *et al.,* 2024).

A comprehensive systematic review of investigations conducted in Mexico has documented elevated frequencies of CAs, SCEs, and MN in populations subjected to pesticide exposure, thereby indicating pervasive DNA damage (Sánchez-Alarcón *et al.,* 2021). An empirical study conducted in Punjab, India, illustrated that agricultural laborers exhibited significantly higher occurrences of CAs compared to non-exposed counterparts, with alcohol consumption further intensifying the associated risk (Ahluwalia & Kaur 2021). A systematic review focusing on agricultural workers within Arab nations disclosed an augmented prevalence of chromosomal translocations and aberrations among individuals exposed to pesticides, thereby emphasizing the public health ramifications of pesticide utilization in these areas (Sherif *et al.,* 2023).

7.3. MN formation

The formation of MN serves as a noteworthy parameter for assessing cytogenetic effect. MN originate from chromosomal segments or entire chromosomes that do not integrate into the daughter nuclei during the process of cell division, thereby signifying genomic instability (Adams *et al.,* 2024). The MN assay is extensively employed to identify genetic harm induced by various chemicals, such as pesticides, rendering it an essential parameter for evaluating cytogenetic damage across diverse populations, including those within occupational

settings (Reddy *et al.,* 2023). Experimental studies reveal that individuals subjected to pesticide exposure demonstrate a significant increase in the frequency of MN.

A comprehensive investigation conducted among Brazilian agricultural farmers revealed increased MN frequencies and chromosomal abnormalities, thereby underscoring the genetic hazards associated with the handling of pesticides (Lucio *et al.,* 2023). In a parallel study conducted in Iraq, it was observed that agricultural workers subjected to pesticide exposure demonstrated a noteworthy increase in MN frequency along with various nuclear anomalies, signifying cytotoxic and genotoxic effects (Alhamadany *et al.,* 2023). In another study, farm laborers exposed to pesticides exhibited elevated levels of MN, which serve as indicators of genomic instability (Santos *et al.,* 2022). Similarly, one more study conducted by Nagy *et al.,* in 2021 demonstrate that Glyphosate and glyphosate-containing herbicides induced the formation of MN in human leukocytes, thereby underscoring the genotoxic consequences of these agrochemicals on human health.

7.4. Causing DNA strand breaks

Pesticides have been demonstrated to cause DNA strand breakage, thereby presenting considerable genotoxic hazards to both humans and non-target organisms. The comet assay is a highly sensitive method utilized for the detection of DNA strand breaks. This comet assay has been extensively utilized to evaluate the genotoxic implications of pesticides. Investigations have shown that organophosphate and carbamate pesticides, including chlorpyrifos-methyl and azinphos ethyl, are capable of inducing DNA damage in human peripheral blood lymphocytes. The comet assay indicated a dose- and time-dependent escalation in DNA damage, highlighting the potential health hazards related to these pesticides (Karsli *et al.,* 2022).

Another investigation concerning agricultural laborers in Iraq indicated elevated DNA damage within lymphocytes and buccal cells attributable to pesticide exposure, with statistically significant variances in comet tail length in comparison to control subjects (Alhamadany *et al.,* 2023). Additionally, a separate study evaluated the genotoxic ramifications of organophosphate and carbamate pesticides utilizing the comet assay, elucidating dose- and time-dependent DNA damage while underscoring the potential health hazards linked to pesticide exposure (Karsli *et al.,* 2022). Cayir et al. (2019) employed the comet assay to examine DNA damage among greenhouse workers subjected to pesticides, thereby revealing the potential genotoxic consequences of pesticide exposure on genetic material. Similarly, Martínez-Valenzuela et al. (2018) investigated the genotoxic effects of pesticides on pilots through the Comet Assay, evidencing substantial DNA damage relative to

control groups, thereby signifying the possible risks associated with pesticide exposure.

8. Mechanisms of cytogenetic damage induced by pesticides

Pesticides elicit cytogenetic damage via multiple pathways. These pathways include direct interactions with DNA, production of reactive oxygen species (ROS), interference with DNA repair processes, and disruption of the mitotic spindle apparatus (Figure 3). Pesticides are known to induce cytogenetic damage via direct interactions with DNA. Research indicates that various pesticides can induce significant genotoxic effects, leading to DNA damage in human cells. For instance, A study on the impacts of fungicides revealed that compounds such as thiram exhibited a strong binding affinity for DNA, resulting in significant cytotoxic effects and DNA damage observed at low concentrations (Barut *et al.,* 2023). In a related context, an additional study on pesticides encompassing α-cypermethrin, chlorpyrifos, and imidacloprid has indicated their potential to induce direct DNA damage in human lymphocytes and HepG2 cells, with α-cypermethrin and chlorpyrifos emerging as notably powerful agents (Želježić *et al.,* 2016).

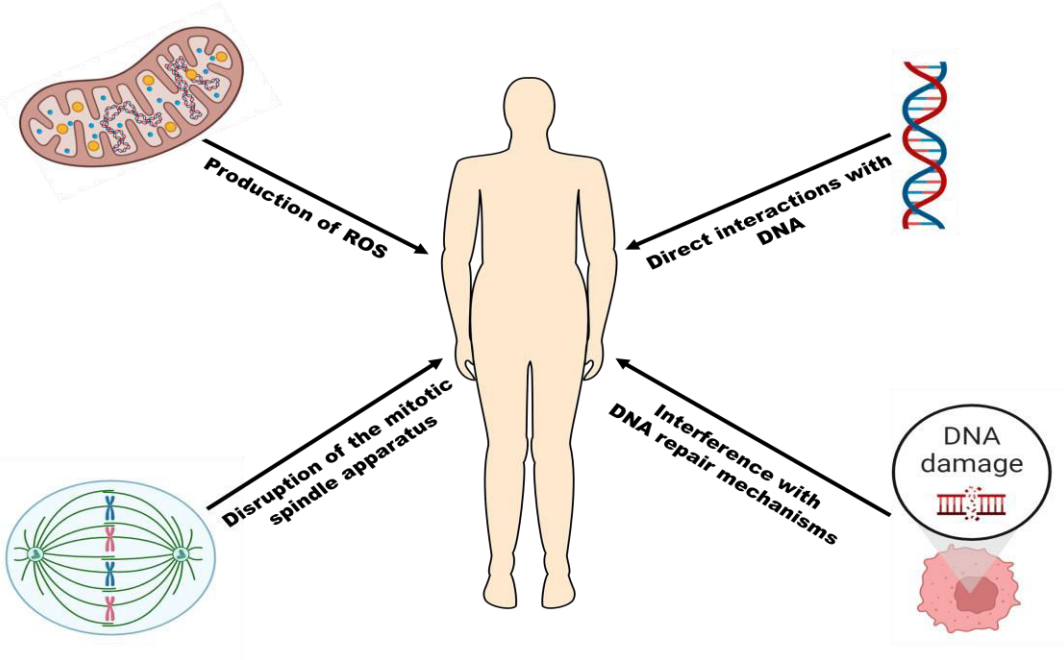

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Figure 3: Different mechanisms of cytogenetic damage induced by pesticides

8.1. Direct interactions with DNA

8.2. Production of Reactive Oxygen Species (ROS)

One of the fundamental pathways through which pesticides elicit cytogenetic injury is by production the formation of reactive oxygen species (ROS). ROS are exceedingly reactive molecules capable of inflicting damage on DNA, proteins, and lipids, thereby precipitating oxidative stress and impairing cellular functionality (Saleh et al., 2023). Certain pesticides, including bifenthrin, have been demonstrated to provoke oxidative stress, resulting in mitochondrial dysfunction and programmed cell death in human cells (Bouaziz *et al.,* 2020). Research has indicated that pesticides enhance the production of ROS, which subsequently leads to oxidative injury to DNA and various cellular constituents (Sule *et al.,* 2022).

8.3. Interference with DNA repair mechanisms

Pesticides have been demonstrated to interfere with key DNA repair enzymes, resulting in substantial genetic damage and possible health hazards. Nevertheless, numerous pesticides have been evidenced to impede the function of essential DNA repair enzymes, culminating in the accumulation of genetic mutations. For instance, research has indicated that exposure to pesticides such as atrazine markedly diminishes the activity of DNA methyltransferases, consequently leading to a reduction in global DNA methylation levels (Wirbisky-Hershberger *et al.,* 2017). Additionally, a study conducted by Kaur *et al.,* (2018) illustrates that occupational exposure to a combination of pesticides resulted in a significant decline in DNA repair capacity.

8.4. Disruption of the mitotic spindle apparatus

The mitotic spindle is a crucial for the precise segregation of chromosomes during the process of cell division. A multitude of investigations have revealed that various categories of pesticides can precipitate abnormalities within the spindle apparatus, consequently leading to significant cellular ramifications. For instance, Dichlorvos (a type of insecticide) induces the formation of monopolar spindles by causing the delocalization of the kinesin Kif2a from the centrosomes, which culminates in hyper-condensed chromosomes and the potential for aneuploidy (Fiore *et al.,* 2013). Furthermore, research concerning exposure to Chlorpyrifos has demonstrated instances of spindle disorganization and misalignment of chromosomes in porcine oocytes, thereby emphasizing its harmful effects on meiotic processes (Jiang *et al.,* 2021).

9. Epidemiological studies and human health implications

Epidemiological research delivers fundamental understanding pertaining to the cytogenetic ramifications of pesticide exposure among human population. Such research has consistently demonstrated that individuals subjected to pesticide exposure, especially those engaged in agricultural professions, exhibit elevated occurrences of chromosomal anomalies, MN, SCEs (SCEs), and DNA strand breaks relative to populations that are not exposed.

9.1. Pesticides and Cancer Risk

The correlation between pesticide exposure, CAs, and the risk of cancer is increasingly substantiated by scholarly research demonstrating pronounced genotoxic effects among agricultural personnel. Investigations indicate that exposure to pesticides is associated with chromosomal instability and modifications, which may serve as potential precursors to cancer. A research study conducted by Aldana-Salazar *et al.,* in 2024, revealed that farmers subjected to pesticide exposure exhibit an increased frequency of chromosomal alterations and instability in comparison to their unexposed counterparts, thereby suggesting a direct genotoxic influence of these chemicals.

The exposure to pesticides has been correlated with the initiation of MN formation, which are recognized as biomarkers indicative of genetic damage and potential carcinogenic risk. Research conducted by Di Bona & Bakhoum (2024) demonstrated that MN arise from chromosome mis-segregation and are notably prevalent among individuals who have been exposed to pesticides, especially within occupational settings. The detection of MN within buccal cells from pesticide-exposed workers indicates detrimental cellular responses, thereby support the association between pesticide exposure and the risk of cancer (RafiqKhan *et al.,* 2014).

In an investigation involving human lymphocytes subjected to tetrachlorvinphos, an organophosphate pesticide identified as a potential carcinogen, researchers observed a dose-dependent increase in SCEs across various concentrations. The observed increase in SCE values attained statistical significance at elevated concentrations, implying that pesticides such as tetrachlorvinphos possess genotoxic properties, which may play a role in the process of carcinogenesis (Akgün & Çobanoğlu, 2024).

9.2. Reproductive Health Implications

Pesticides exert significant influence on reproductive health, affecting both male and female systems through diverse biological and environmental exposures.

Empirical evidence suggests that pesticides can interfere with hormonal equilibrium, resulting in reproductive dysfunction and negative pregnancy outcomes. For example, Alawiyah *et al.,* (2024) identified associations between pesticide exposure and ovarian dysfunction, hormonal anomalies, and heightened risks of conditions such as endometriosis and stillbirths. In addition, an analysis targeting women before pregnancy unveiled substantial contamination with current pesticides, with notable health threats correlated to exposure, especially from organophosphate insecticides (Tian *et al.,* 2024). Additionally, pesticides can detrimentally affect male reproductive capabilities, particularly influencing sperm capacitation, which is essential for successful fertilization. This disruption may result in infertility (Uwamahoro *et al.,* 2024). Another scientific study analyzes the effects of occupational and environmental pesticide exposure, especially in relation to endocrine-disrupting chemicals, on fertility, sexual maturation, and pregnancy outcomes. It highlights that even low doses of these substances can profoundly affect reproductive health, resulting in reduced fertility and birth defects (Fucic *et al.,* 2021).

10. Mitigation strategies and future directions

Given the possible cytogenetic hazards linked to pesticide exposure, there is an urgent need for efficacious mitigation strategies. One such strategy entails the implementation of biopesticides. Biopesticides, which are derived from natural sources such as flora, fauna, microorganisms, and minerals, provide targeted pest management while maintaining a minimal environmental impact. Empirical studies indicate that biopesticides play a crucial role in the framework of Integrated Pest Management (IPM) strategies, thereby diminishing dependence on detrimental chemical pesticides. A pertinent illustration is that microbial biopesticides, which employ bacteria, fungi, and viruses, have demonstrated effectiveness in suppressing pest populations via mechanisms such as antibiosis and parasitism, exerting negligible effects on non-target species and the surrounding ecosystem (Haroon *et al.,* 2023).

Although exhibiting lower toxicity levels, biopesticides effectively function in minimal doses and are rapidly decomposable, consequently avoiding the environmental pollution that is typically connected to synthetic chemicals. Nevertheless, obstacles such as regulatory limitations and restricted market penetration remain prevalent (Soetopo & Alouw, 2023). A deliberate transition towards biopesticides is imperative for the promotion of sustainable agricultural practices, accompanied by recommendations for the amendment of pesticide regulations and the enhancement of public policy support (Kumari *et al.,* 2022).

An additional significant strategy encompasses landscape mitigation measures, such as the establishment of vegetative buffer strips, which have been

validated as effective in diminishing pesticide runoff in agricultural catchments; however, challenges such as climatic variability and baseflow persistence introduce complexities that may affect their efficacy (Quaglia *et al.,* 2024). Furthermore, biotechnological approaches, including CRISPR (clustered regularly interspaced short palindromic repeats) and RNA interference (RNAi), present viable solutions by facilitating the development of pest-resistant crops, thus indirectly reducing the necessity of chemical pesticides (Chaudhary *et al.,* 2024). Recent scholarly investigations also underscore the potential of nanotechnology-based methodologies for decreasing pesticide bioavailability and leaching into soil matrices, thereby mitigating adverse effects on non-target environments (Parthipan *et al.,* 2024). These multifaceted strategies, which integrate biotechnological advancements with landscape-oriented interventions, are pivotal for the progression of sustainable pest management while minimizing environmental degradation.

11. Conclusion

Pesticides, although indispensable for modern agricultural practices, present considerable cytogenetic hazards to both human health and the ecological environment. The findings from a plethora of investigations indicate that exposure to these chemical agents can induce various types of DNA damage, encompassing CA, formation of MN, SCEs, and breaks in DNA strands. Given the considerable health implications, particularly the increased risk of carcinogenesis and reproductive issues, it is essential to develop strategies focused on reducing pesticide exposure and to create safer alternatives.

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