CHAPTER 1

The Fate of Pesticides in Wastewater: A Review

YUSUF OLORUNTOYIN AYIPO,[1,*] CHIEN FUNG CHONG,[2] AKEEM ADEBAYO JIMOH,[1] LUKMAN BOLA ABDULRA'UF,[1] and WAHAB ADESINA OSUNNIRAN[1]

[1]*Department of Chemistry and Industrial Chemistry, Kwara State University, Malete, Ilorin, Nigeria*

[2]*Bioprocess Technology Division, School of Industrial Technology, Universiti Sains Malaysia, Gelugor, Penang, Malaysia*

*Corresponding author

ABSTRACT

Pesticides are substances applied primarily in agricultural practices to improve the quality and yield of crops by protecting them against pests and diseases. The growing global demand for adequate and high-quality food necessitates an increased rate of pesticide application for food production. As a result, active pesticide ingredients, referred to as pesticide residues (PRs), accumulate in domestic and industrial wastewater through effluent discharge and runoff from agricultural or industrial settings. Consequently, these residues induce physicochemical (abiotic) processes and biochemical (biotic) transformations, degrading water quality and leading to acute or chronic toxicity in aquatic animals and humans, depending on their nature and concentration.

Long-term exposure to PRs can pose significant risks to public health through pesticide poisoning. The associated risks include ataxia, carcinogenicity, heart, liver, and kidney diseases, lung injury, skin irritation, paralysis, and death, depending on the pathways of cellular and molecular interactions within biological systems. Thus, the presence and fate of PRs in wastewater remain a global concern, requiring adequate scientific attention to mitigate their effects on humans and the environment.

This chapter provides an overview of some prominent PRs in wastewater from both agricultural and non-agricultural fields. These include organochlorines, organophosphorus compounds, S-metolachlor, polychlorinated biphenyls (PCBs), polychlorinated dibenzo-dioxins (PCDDs), atrazine, chlorfenapyr, chlorpyrifos, diazinon, difenoconazole, ethion, fipronil and its metabolites, malathion, dichlorvos, methyl parathion, profenofos, and thiacloprid. The review enumerates the processes leading to the deposition of pesticides in wastewater, the consequences of their accumulation, and approaches for their effective

Pesticide Removal Methods from Wastewater: Proactive Approaches and Future Trends. Pranav Deepak Pathak, Anuja Rajendra Jadhav, Sunil Kautikrao Deokar, and Sonali Patil (Eds.)
© 2026 Apple Academic Press, Inc. Co-published with CRC Press (Taylor & Francis)

removal. It also aids in developing promising strategies to minimize the adverse effects of pesticide toxicity on public health.

1.1 INTRODUCTION

Pesticides are chemicals primarily used in agricultural processes to improve the yield and quality of crops by protecting them from pests, disease attacks, and disease vectors [1]. They are employed to control the undesired competition between humans and other organisms for high-quality food and fiber. Thus, pesticides play a crucial role in economic growth through agricultural production and the preservation of various food types, such as cereals, fibers, forages, fruits, edible oils, tubers, vegetables, and livestock.

Pesticides are formulated as bactericides, disinfectants, fungicides, herbicides, insecticides, and other substances designed for controlling, destroying, or preventing pests, including vectors of animal (including human) and plant diseases [2]. Their applications are invaluable in the agricultural industries worldwide [3]. The toxicity of pesticides depends significantly on the physicochemical properties of their constituent residues.

Active ingredients of pesticides, also referred to as pesticide residues (PRs), include atrazine, aldicarb, diazinon, malathion, dichlorvos, methyl parathion, profenofos, chlorfenapyr, difenoconazole, thiacloprid, acephate, chlorpyrifos, ethion, and carbophenothion [1, 4–6]. Other notable PRs include organochlorines such as dieldrin, chlordane, heptachlor, dichlorodiphenyldichloroethylene (DDE), dichlorodiphenyltrichloroethane (DDT), and their metabolites [7].

The application of pesticides has significantly increased, rising by approximately 30% between 2000 and 2020 and reaching the highest global levels since 2017. Concurrently, the rate of pesticide application per cropland surged by about 25%, escalating from 1.5 kg/ha to 1.8 kg/ha between 2000 and 2010, with the primary contribution attributed to America [2]. Projections based on a linear trajectory of usage suggest an anticipated surge to over 4.19 million metric tons by 2024 [8].

Pesticides are also significant contributors to water pollution. For instance, organochlorines and a few persistent residues (PRs), such as dioxins and polychlorinated biphenyls (PCBs), are often classified as persistent organic pollutants (POPs) [7]. The fates of PRs in wastewater are influenced by unregulated reactions and processes, including biochemical degradation, disintegration, absorption, ingestion, and inhalation by plants and animals [9]. These processes can lead to pesticide poisoning when treated wastewater is used for various life activities.

The extensive application of pesticides on food crops and livestock production contributes to the direct and indirect accumulation of PRs, posing persistent risks to public health through pesticide poisoning. In biological systems, PR components disrupt normal cellular activities by inducing biochemical changes, resulting in acute or chronic toxicity depending on the nature, dose, and duration of exposure [4]. For example, organophosphorus-based PRs reportedly cause acute toxicity and Organophosphate-Induced Delayed Polyneuropathy (OPIDP) through the mechanistic inhibition of acetylcholinesterase (AChE) and other associated esterases with neuropathy, leading to the accumulation of acetylcholine

at nerve terminals. These adverse biochemical processes result in effects such as ataxia, paralysis, and, in severe cases, death [5].

Other health risks associated with PR exposure include carcinogenicity, hepatotoxicity, lung injury, heart disease, skin irritation, developmental and neurological disorders, organ damage, and infectious diseases [10, 11]. In 2020, approximately 385 million cases of Unintentional Acute Pesticide Poisoning (UAPP) were estimated to occur annually, resulting in about 11,000 fatalities worldwide. About 44% of these cases involved farmers, predominantly in Southern Asia and South-Eastern Asia, where agricultural practices are most extensive [10]. Asia is currently the second-highest consumer of pesticides after America, followed by Europe, Africa, and Oceania [2].

This alarming data underscores the dual nature of pesticides, which significantly contribute to economic growth while also posing serious risks, particularly when present in vital resources such as recyclable wastewater. Therefore, reviewing the fate of pesticides in wastewater is essential for understanding their occurrence, promoting safe and beneficial applications, and developing effective remediation strategies, as presented herein.

1.2 OCCURRENCE OF PESTICIDES IN WASTEWATER

Several factors influence the occurrence of pesticide residues (PRs) in wastewater, including the type of pesticide, its specific properties, and the methods of application [12]. The presence of PRs in wastewater is intricate and multifaceted, with various contributors. Activities such as agricultural practices, urban runoff, water overflow through farmlands, and improper discharge of PR-containing effluents or wastes significantly facilitate the infiltration of pesticides into wastewater. Among these, agricultural activities stand out as a prominent contributor, creating a complex dynamic that extends beyond local fields.

Environmental variables, including soil composition, topography, climate, proximity to water bodies, and inadequate erosion control measures, exacerbate pesticide contamination in wastewater [13, 14]. During rainfall or windfall, runoff containing PRs from application sites gains momentum and enters water bodies and soil [15, 16]. The subsequent leaching of PRs through soil profiles at these sites can pollute groundwater and wastewater systems [9].

Domestic pesticide use for pest control also contributes significantly to PR accumulation in wastewater. Residues from household products, such as insecticides and herbicides, are washed off surfaces into drains. Improper disposal practices, including discarding unused pesticides or empty containers, further introduce these chemicals into wastewater systems [17]. The cumulative impact of widespread domestic engagement in such activities amplifies the burden of PRs in domestic wastewater [18].

Industries involved in pesticide manufacturing and pharmaceutical production significantly contribute to the accumulation of PRs in wastewater. Wastewater from pesticide formulation processes often contains residues, further diversifying the chemical composition entering wastewater treatment facilities [19]. The multifaceted nature of industrial activities thus contributes to the complex web of pesticide contamination in aquatic environments [20].

Discharges from industrial processes, whether intentional or accidental, often introduce concentrated doses of pesticides into water systems. Untreated or insufficiently treated industrial wastewater is a prominent pathway for the infusion of PRs into local water bodies [21]. These interconnected pathways—domestic, agricultural, and industrial—highlight the challenges in mitigating pesticide contamination in wastewater [22].

Relevantly, some reported analyses have revealed the presence of various pesticide residues (PRs) in wastewater. Notably, these include organophosphates (OPs) [23, 24], carbamates (CMs) [25, 26], synthetic pyrethroids (PYs) [27, 28], s-triazines [24, 29], triazole pesticides [30], phenylpyrazoles [31], neonicotinoids [31, 32], and organochlorine pesticides (OCPs) [7, 33].

Once in wastewater, PRs disperse across biotic and abiotic environments through complex natural and anthropogenic phenomena such as diffusion, evaporation or volatilization, leaching, erosion, runoff, and uptake by plants and animals, potentiating their transformation into harmful products [9, 12, 21]. However, exposure to PRs through food consumption is more prevalent than other routes, such as air and drinking water [1].

Their accumulation in biological systems and environments can be indirectly triggered through their presence in treated wastewater used for domestic and industrial applications, including aquaculture, agriculture, engine cooling, and manufacturing processes, even in trace amounts. Another major concern regarding PRs in wastewater is the resultant water pollution, which can lead to water unavailability and eventual scarcity, presenting a significant global challenge [14].

Thus, efficient, cost-effective, timely, and environmentally friendly assessment and treatment strategies are essential to mitigate the consequential risks associated with polluted water. Prominent routes of pesticide infiltration into wastewater are illustrated in Figure 1.1.

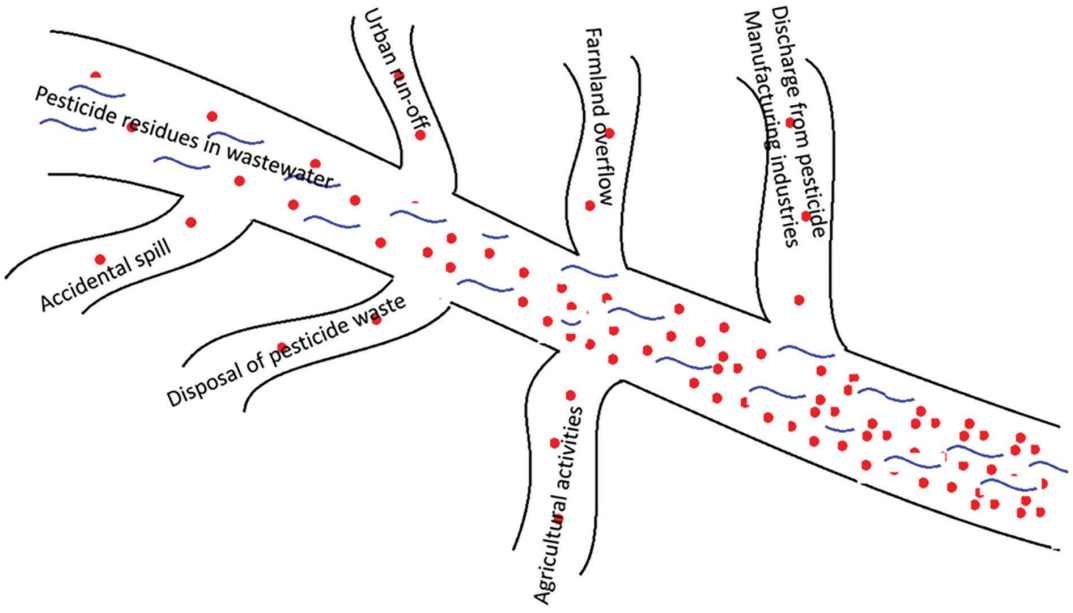


FIGURE 1.1 Common routes of infiltration of pesticides into wastewater.

1.3 RISK IMPACTS OF PESTICIDES IN WASTEWATER ON THE ENVIRONMENT AND HUMAN HEALTH

Undoubtedly, pesticides pose significant threats to both biotic and abiotic components of human environments when present in essential commodities such as water. A comprehensive understanding of these implications is crucial for instituting measures to mitigate their consequential effects on aquatic ecosystems and safeguard human health through water usage [34].

1.3.1 IMPLICATIONS OF PESTICIDES ON WATER ENVIRONMENT AND AQUATIC LIFE

The significant environmental ramifications of pesticide residues (PRs) in wastewater are evident. Within aquatic ecosystems, the persistent presence of these residues results in bioaccumulation, impacting the entire food chain. The discharge of treated wastewater into receiving water bodies introduces PRs into the environment, potentially jeopardizing water quality and posing risks to aquatic life [8, 35].

The process of bioaccumulation further amplifies ecological risks, affecting biodiversity and ecosystem services. Additionally, it adversely impacts the physiology and reproductive activities of aquatic organisms [8, 36].

1.3.2 HUMAN HEALTH CONCERNS

The underestimated presence of trace pesticides from runoff or leaching at dumping sites and in wastewater effluents is a significant concern, often accompanied by a lack of awareness regarding associated risks and necessary precautionary measures. The widespread contamination by these organic pollutants has emerged not only as a pressing environmental issue but also as a considerable threat to human well-being [34].

Despite the implementation of various safety measures aimed at mitigating the systemic impact of these chemical agents, their persistent toxicity—manifested in acute or chronic forms—remains a formidable challenge. Regrettably, a substantial knowledge gap exists among individuals regarding the potential harm posed by pesticide-contaminated wastewater, which contributes to the neglect of safety measures.

Therefore, it is crucial to emphasize and address the risks that pesticides in wastewater impose on humans.

In response to the ever-increasing demand for water in modern economies, wastewater has long been considered a primary alternative [37]. However, the retention of organic toxic pollutants remains a significant concern [31, 38–41]. The consequences extend far beyond immediate environmental impacts, as multiple pathways connect humans to pollutants present in wastewater. These exposure routes can be broadly categorized as:

1. **Direct Exposure:** This occurs through the ingestion of water contaminated with wastewater or dermal contact during recreational activities in wastewater-polluted water bodies [42–44].

2. **Indirect Exposure:** This arises from the consumption of aquaculture or agricultural products grown in areas irrigated with contaminated water [40, 45]. Additionally, individuals may be indirectly exposed through the inhalation of aerosols emitted by wastewater treatment plants [46].

Given these diverse exposure pathways, it is imperative to explore the potential health hazards associated with various pesticide classes.

1.3.2.1 ORGANOPHOSPHATES

Various organophosphorus compounds (OPs), including tributyl phosphate, malathion, glyphosate, and certain OP metabolites (e.g., aminomethylphosphonic acid, 3,5,6-trichloro-2-pyridinol, malathion dicarboxylic acid), have been found to exceed residue limits and guideline levels in wastewater [24, 47–50]. Acute OP toxicity typically presents with symptoms such as headache, tremors, drowsiness, and irritation in various body parts. General poisoning symptoms include muscle weakness, seizures, hallucinations, bradycardia, miosis, nausea, and respiratory issues [51, 52]. In severe cases, individuals may experience coma and potentially fatal shortness of breath caused by pulmonary edema at high exposure levels [53, 54].

Prolonged exposure to OPs is also associated with bronchitis, manifesting as cough and phlegm. Central respiratory depression, caused by bronchorrhea and bronchospasm, is a major cause of mortality in reported cases of OP toxicity [52]. Despite their limited ability to penetrate the brain, OPs exert profound neurotoxicity. Symptom onset varies, ranging from a few minutes to resolution within several weeks [52].

This neurotoxicity is attributable to OPs' ability to peripherally inhibit acetylcholinesterase (AChE) through the irreversible phosphorylation of the serine hydroxyl group [55]. Upon entering the body, OPs lead to the excessive accumulation of the neurotransmitter acetylcholine within synaptic junctions. This overstimulation of cholinergic pathways, involving both nicotinic and muscarinic receptors, ultimately results in delayed neurotoxicity or cholinergic toxidrome [56].

In a pilot study by Muniz and collaborators, pesticides were observed to potentially cause detrimental effects in humans via oxidative stress [57]. This stress is characterized by a reduction in glutathione levels and an amplification of lipid peroxidation products [58–60]. This phenomenon manifests consistently across various contexts (*in vitro*, *in vivo*, and real-world scenarios) following contact with organophosphates (OPs) [61].

In examining the influence of chronic exposure to organophosphorus compounds on reproductive systems, researchers found that these compounds mediate oxidative and antioxidant imbalance. This imbalance contributes to a defective hypothalamic and/or pituitary-endocrine axis, which has been associated with diminished spermatogenesis and impaired gonadal functions [62].

While OPs are generally not classified as oncogenic, certain studies have established links between these compounds and specific cancers. According to a systematic review, among all OPs detected as residues in wastewater, malathion and chlorpyrifos

exhibit a positive association with human breast cancer. Proposed mechanisms include elevated oxidative stress, estrogenic potential, and decreased apoptotic action [63]. A prospective study further supports this association, documenting links between OPs and hormonally mediated cancers, such as those affecting the thyroid, ovary, and breast. This evidence suggests that the estrogenic potential of OPs may act as a mechanism for cancer induction [64].

In an *in vivo* study involving chronic exposure to malathion at low doses, spatial memory and discrimination were impaired, likely due to a decrease in mitochondrial complex I activity within the hippocampus [65]. Extrapolating these findings to humans exposed to malathion residues in wastewater raises concerns about potential neurobehavioral and cognitive defects [65].

The mechanisms underlying Parkinson's disease (PD), characterized by oxidative stress and interference with neurotransmission, are becoming increasingly evident among populations frequently exposed to pesticides [66]. An intriguing case involves an individual who, after a four-year recovery from consuming a substantial amount of glyphosate, developed Parkinsonism, suggesting a link between organophosphates (OPs) and young-onset atypical PD [67, 68].

Rajak et al. proposed a potential immunotoxic effect of OPs in humans, rendering them more susceptible to opportunistic infectious diseases [69]. This effect extends to various aspects of mammalian immunity, including neutrophil-mediated immunity, T- and B-lymphocytes, the complement system, cytokine signaling, and basophilic cells [70].

Epidemiological studies further indicate that exposure to pesticides, particularly OPs, may serve as a risk factor for diabetes [71–73]. OPs have been observed to disrupt glucose and fat homeostasis, leading to impaired insulin secretion *in vivo*. One proposed mechanism involves the adenylyl cyclase/cyclic AMP pathway [74]. Additionally, OPs may damage pancreatic beta cells, contributing to excessive hepatic gluconeogenesis and insulin resistance [75].

Expanding beyond these health hazards, OP exposure has demonstrated strong correlations with other conditions, including Alzheimer's disease [76, 77], coronary artery disease [66], emotional disorders, and neurobehavioral diseases [78, 79].

1.3.2.2 CARBAMATE

Carbamates (CMs) exhibit relatively low persistence in the environment due to their rapid hydrolysis. Among them, oxamyl and methomyl have been identified in wastewater, albeit at low concentrations (in $\mu g\ L^{-1}$). Despite these low levels, CM residues can impact non-target organisms through environmental contamination [26, 80].

Acute exposure to CMs presents with both parasympathetic and sympathetic symptoms, closely resembling those of organophosphates (OPs). These symptoms include severe headaches, nausea, vomiting, abdominal pain, and salivation [81, 82]. Additional reported effects include body weakness, twitching, blurred vision, cramping, lethargy, ataxia, and sweating [83].

Mechanistically, carbamates (CMs) function similarly to organophosphate (OP) pesticides; however, instead of irreversible binding, they reversibly bind to acetylcholinesterase (AChE), resulting in a toxicological presentation with high similarity but a relatively short resolution period (generally less than 24 hours) [84, 85]. The bond formed between CMs and AChE is less stable, allowing spontaneous decarbamylation to occur shortly thereafter, leading to enzyme reactivation [85].

Because CMs act reversibly, their subchronic and chronic exposure is notably less toxic compared to acute exposure [83]. Studies on chronic exposure to CMs in mice have demonstrated significant differences in behavioral and neurochemical changes [86].

At sublethal concentrations, certain carbamates (CMs), such as oxamyl, exhibit a low tendency to cause genotoxicity in humans [87]. In contrast, others, such as methomyl, may display genotoxic properties, causing damage to cellular DNA and triggering apoptosis, which is considered a biomarker for carcinogenic risk [88, 89].

Additionally, CMs function as endocrine-disrupting chemicals, exerting deleterious effects on the endocrine system and male reproductive systems by perturbing the hypothalamic-pituitary-testicular (neuroendocrine-reproductive) axis. This disruption alters hormonal profiles, ultimately leading to reduced spermatogenesis, abnormal sperm quality, and/or diminished male fertilizing capability [90–92].

Interestingly, CMs possess a mitochondria-targeting ability, potentially augmenting developmental defects [93]. Also, oxidative stress action induced by CMs has been implicated in the adverse effects on the heart [94–96], brain [97], kidneys [91, 97], and liver [97, 98]. Nonetheless, repeated exposure may negatively impact emotions and lead to conditions such as depression, anxiety, or irritability [82].

While studies thus far have not established a correlation between the two CMs that were found as residues in wastewater and immunotoxicity or carcinogenicity [83, 87], other research suggests that most CMs can provoke immune alterations. Not only that, but they may also induce mutations in immunoregulatory genes and modify immune tolerance, thereby exacerbating pathological immune responses as seen in autoimmunity, hypersensitivity reactions, or cancer [99]. Studies have identified CMs-induced immune dysregulation owing to oxidative stress and inhibitory properties on esterases. In some instances, CMs, by suppressing immune functions and showing up in the tumor microenvironment, were thought to play a role in early tumor formation [99]. On an alternative note, CMs have been associated with elevated risks in specific cancers such as those affecting bones, bladder, uterus, liver, kidney, thyroid, and adrenal gland [100].

1.3.2.3 SYNTHETIC PYRETHROIDS

Multiple synthetic PY residues, including type-I (permethrin) and type-II (cypermethrin, cyhalothrin, cyfluthrin, deltamethrin), were significantly observed in wastewater [27, 28], prompting a critical examination of their relevance to human hazard. Publications on pyrethroid intoxication highlight common acute poisoning symptoms, including dizziness, nausea, skin itching/burning, and vomiting, with severe cases potentially leading to coma, seizures, and hemorrhage [101]. In comparison to previously discussed OPs and CMs,

pyrethroids display different acute toxicity profiles, generally resulting in milder toxicological outcomes. In most cases, they tend to rapidly metabolize into non-toxic metabolites, contributing to their minimal selectivity and knock-down effects on humans [102]. Concerning pyrethroid toxicity in mammals, two distinct toxidromes (neurotoxicity) are present depending on the structural subgroups (type-I and type-II) of PYs. Both subgroups mainly disrupt channels or proteins [102, 103]. Given that they act on voltage-gated Na^+, Cl^-, and Ca^{2+} channels [102], it is agreeable that excitable tissues are the primary sites of synthetic PYs.

Research has determined that PYs stimulate changes in the kinetic transition characteristics of voltage-gated Na^+ channels, prolonging the gate opening. This stimulation leads to extended sodium influx and repetitive firing, causing protracted neuronal excitation [104]. The duration of poisoning usually corresponds to the amplitude of the current, determined by the dose of PYs exposed and the amount of affected channel. In addition, various PYs potentiate varying durations for the state of excitability [104], with type II promoting longer durations compared to type-I PYs [105]. At high doses of exposure, a "conduction block" occurs, where sodium influx is no longer capable of generating action potentials. This contributes to the clinical presentation of paraesthesia and proarrhythmogenic effects [106, 107]. The common clinical feature (paraesthesia), along with relentless fine tremor and marked reflex hyperexcitability, contributes to the typical type-I syndrome induced by type-I PYs.

In the context of type-II PYs' action on voltage-gated Ca^{2+} channels, which control cell excitability in muscles, the brain, nerves, and salivary glands, they reduce the state of opening and lower chloride currents. This is expected to manifest symptoms such as salivation and myotonia, as well as synergize with Na^+ channel-mediated intoxication [102, 108]. Notably, these actions occur at low concentrations [108], emphasizing the significance of residues to human health. With direct effects on those targeted bodily parts, type-II PYs contribute to type-II syndromes featuring salivation, coarse tremor, moderate reflex hyperexcitability, and choreoathetosis.

While PYs present fewer chronic consequences in general, some literature points out their significance in affecting neurocognitive performance. One study concluded an association with deterioration in neurocognitive performance, though not affecting neuromotor performance [109]. Besides, a study by Hansen et al. revealed a higher occurrence of hyperglycemia, signifying a trend between cumulative PY exposure and dysregulated glucose control in the body [110]. PYs, similar to OPs and CMs, not only disrupt endocrine function, cause genotoxic effects, and exert oxidative stress but also bind to DNA to induce epigenetic modifications [111]. These mechanisms represent the basis for carcinogenicity. This aligns with a study that determined a correlation between PY exposure and childhood hematological cancer [112].

1.3.2.4 S-TRIAZINES

Wastewater contamination by triazine residues, such as chloro-s-triazine (atrazine) and thiomethyl-s-triazines (simetryn, prometryn), along with some degraded products, has

been documented [24, 29]. The persistence of these compounds, which are resistant to biological and chemical degradation, raises concerns [113]. While triazines have specific mechanisms affecting plants, such as interference with the photosynthetic process, they also pose toxicity to non-target species through common mechanisms shared by various pesticides, including neurotoxicity, endocrine-disrupting effects, and oxidative stress [114, 115].

Acute intoxication with triazines commonly presents symptoms such as vomiting, dermatitis, abdominal pain, diarrhea, and mild mucosal irritation. Some chloro-s-triazines act as substrates for xenobiotic-metabolizing enzymes. For instance, at high doses, atrazine or simazine interacts with rat cytochrome P450 (CYP) [116, 117]. The health impacts of atrazine are further verified using *in vitro* human CYP1A2, 2A6, 2B6, and 3A4, where repression or induction is observed [118, 119]. In terms of chronic intoxication, murine models subjected to high-dose treatment suffered from kidney damage, likely due to the effects of metabolites and precipitation in the kidneys [120].

Studies on reproductive and developmental intoxication are clear and observable, particularly regarding the effect on females [120]. Atrazine, as a hormone disrupter, inhibits the surge of preovulatory luteinizing hormone in female rats, leading to a series of adverse effects such as an extended oestrus period (adult) and delayed development (developing) at the maximum tolerated dose [121]. A similar study also revealed early-onset mammary and pituitary tumorigenesis, likely owing to disruption of neuroendocrine balance. Yet, this effect may not be extrapolated to humans due to strain-specific effects. Other researchers noted that atrazine alters pituitary hormone release, estrogen to androgen ratio, and up-regulates aromatase activity, all of which impact reproductive functions *in vivo* [120]. Interestingly, studies on whether chloro-s-triazines are considered teratogenic, carcinogenic, mutagenic, or genotoxic have consistently yielded negative results [122]. Multiple pieces of epidemiologic evidence have yet to establish a consistent causal relationship between triazine and human cancers [123, 124].

1.3.2.5 TRIAZOLE

In wastewater, triazole residues, exemplified by propiconazole and myclobutanil, are frequently detected due to their long half-life and low biodegradability, posing a threat to non-fungal organisms [30]. Accidental contact exposure to triazoles can result in acute toxicity, presenting symptoms such as headaches, chest pain, skin, and eye irritations, or irreversible injury, whereas inhalation may lead to mild respiratory irritation [125, 126].

In fungi, triazoles act by directly binding to heme iron of CYP51, hindering the enzyme's action in the fungal biosynthesis of ergosterol. While higher species, including humans, also possess a similar enzyme (sterol 14-demethylase) in various tissues for sterol biosynthesis, this could be a possible mechanism for triazole intoxication [127]. Apart from CYP enzymes, triazoles could interact with other CYP enzyme families to initiate mammalian toxicity [128]. Using laboratory animal models, common acute effects are related to development [127]. They generally exert embryotoxic properties with features such as low fetal weight, prolonged ossification, and malformed craniofacial and/or axial

structures [128]. The authors suggest that in addition to the expected mechanisms (CYP51 inhibition), some sensitive pathways involving retinoic acid metabolism are implicated. Furthermore, cases of triazine perinatal exposure, but not adult exposure, causing hippocampal deficits strongly suggest a developmental toxicant effect [129].

Moreover, triazoles have the potential to alter the activity of steroidogenic CYPs (i.e., CYP19A1 or aromatase), as evidenced in experimental (*in vitro* and *in vivo*) investigations [130, 131]. This property elicits unpleasant effects on the reproductive system, including infertility, lowered survivability of new litter, and greater perinatal mortality [127]. One study showed that rats treated with triazoles directly inhibit aromatase in rat Leydig cells, altering the conversion of testosterone into estrogen [131]. Another examination in female rats displayed virilization [132]. Following a longer exposure, testosterone levels increased in rat testes and the blood, plausibly due to the action of triazoles on the hypothalamic/pituitary/gonadal axis [132].

Long-term triazole exposure can also affect the liver [133]. The liver injury manifests in various patterns, including the undesired induction of enzymes, oxidative stress, hypertrophy, hyperplasia, and inflammatory events [127, 134]. Consequently, certain triazoles exhibit carcinogenic properties by stimulating tumor growth in the liver and thyroid follicular cells. Since most triazoles are negative in genotoxicity, the cancer-inducing effect occurs via non-genotoxic mechanisms [127, 135].

1.3.2.6 PHENYLPYRAZOLES

Phenylpyrazoles, prominently represented by fipronil and its metabolites, have recently been identified as major residues ubiquitously present in wastewater at toxicologically relevant levels [31]. Concerns have escalated due to reported greater toxicological profiles of metabolites compared to the parent compound [136, 137], and as such, understanding the undelivered effects in humans remains crucial. Acute toxicity symptoms range from mild outcomes, such as headache, abdominal pain, nausea, sweating, vomiting, dizziness, and agitation, to severe cases with Central Nervous System (CNS) toxicity, recurrent seizures, and, in certain instances, fatal outcomes [138, 139].

Mechanistically, this chemical family inhibits inhibitory receptors, specifically the γ-aminobutyric acid (GABA) gated chloride channel in the brain or $GABA_a$ receptor, with significant selectivity for insect isoforms over vertebrate counterparts [140, 141]. Researchers have noticed that phenylpyrazoles can also bind to the mammalian GABA receptors [142]. Fipronil, in particular, inhibits human glycine receptors (subtypes of α1, α1β, α2, α3), suggesting plausible toxicity through this targeting of human glycine receptors [143]. Consequently, these mechanisms could induce neurotoxicity by causing an excess Cl^-, leading to neuron overstimulation and hyperexcitation. Additionally, phenylpyrazoles can elicit cell death in neuronal cells through apoptotic events and rapid depletion of ATP via mitochondrial uncoupling, with fipronil acting as an oxidative phosphorylation uncoupler even at low doses, signifying potent cytotoxic properties towards the CNS [144]. Apart from neurotoxicity, high-dose fipronil treatment in animal studies altered normal exploratory behaviour, emotionality, and fear in rats [145].

Sub-chronic exposure to phenylpyrazoles revealed pronounced effects on the kidneys and liver, inducing lipid peroxidation and generating free radicals (oxidative stress mechanisms) [146], with signs of liver injuries, such as hepatomegaly, necrosis, and steatosis [147]. Moreover, they exhibited strong genotoxicity [148, 149]. The male reproductive system was affected after chronic exposure to high doses of fipronil, proposed to cause the effect through oxidative stress-induced DNA injury, resulting in markedly reduced sperm quality and viability [150]. A recent study using a mouse model revealed an association of lung injuries (e.g., airway inflammation) secondary to fipronil chronically exposed at low doses [151], indicating a potential concern for fipronil residues in wastewater.

1.3.2.7 NEONICOTINOID

Residues such as imidacloprid, clothianidin, acetamiprid, and its metabolite, all falling under the group of neonicotinoids, have been detected in wastewater, elevating the risk of exposure and poisoning [152]. Despite presumed lower non-target toxicity attributed to physiological differences between insects and vertebrates [152, 153], recent research challenges this assumption, revealing certain human health concerns associated with neonicotinoids [154]. Upon acute exposure, neonicotinoids lead to symptoms highly similar to OPs, presenting with muscarinic clinical features [155]. Severe cases may result in complications such as ventricular fibrillation, respiratory failure, or death, although the mortality rate thus far is < 3% [154]. Notably, clinical features for acute or chronic exposure to neonicotinoids may not always be similar due to the intricate interactions of receptor sites by neonicotinoids [156].

Neonicotinoids act as irreversible nicotinic agonists to neuronal receptors, specifically nicotinic acetylcholine receptors (nAChRs). Following binding to the receptors, Na^+/K^+ channels are blocked, hindering the transmission of neuronal influx. Neonicotinoids show a stronger affinity for insect subtypes of α4β2 than mammalian nAChRs, with lower blood-brain barrier (BBB) penetration; however, recent research indicates modulation activity on mammalian nAChRs (subtypes of α4β2 and α7) in the CNS [152], and certain neonicotinoids (i.e., imidacloprid, acetamiprid) can substantially cross the BBB [156]. Long-term exposure could stimulate the constant opening of nAChRs, in particular homopentamers α7, resulting in high Ca^{2+} permeability that interferes with multiple signaling pathways in neurons and glial cells [152]. These properties label neonicotinoids as neurotoxicants because they can promote neurotoxicity and neuromorphological defects in neuronal cells owing to impaired neurotransmission, neuroinflammation, and oxidative stress induction, all of which promote cell death [156].

Regardless of the diverse distribution of nAChRs in vertebrate brain regions, the hippocampus, among all, is found to be most vulnerable to the neurotoxic effect [152]. Development neurotoxicity of neonicotinoids is evidenced in multiple animal reports, with maternal exposure to acetamiprid increasing early post-natal mortality and causing defective auditory startle response in off-springs [156]. In murine models, transient in-utero exposure to a non-lethal dose of neonicotinoid (imidacloprid) induces neurobehavioral deficit [157]. In addition to that, even at very low doses, adults experience various neurobehavioral

impairments [158], which can be linked to modifications in neuropathology and neurochemical profiles [156, 159].

Studies by Şenyildiz and colleagues documented that certain neonicotinoids possess potent genotoxicity, inducing DNA damage in tested human neuroblastoma and liver cells [160]. A few other studies also observed their impacts on the liver, immune system, kidneys, and reproductive systems. Chronic exposure to imidacloprid in rat models showed signs of chronic inflammation accompanied by elevated pro-inflammatory cytokines not only in the brain but also in the liver, attributed to oxidative stress mechanisms [161, 162]. Another chronic study noted moderate pathological changes in the kidneys as well, indicating multi-organ toxicity induced by imidacloprid [163]. Again, chronic exposure to neonicotinoids can adversely influence the immune system, with a significant drop in lymphoproliferation and macrophage functions following sub-chronic treatment with acetamiprid [164, 165]. Interestingly, this effect is present in the absence of lymphoid organ toxicity. Regarding reproductive intoxication, some research activities have shown that persistent exposure to low concentrations of imidacloprid may exert oxidative injuries to the reproductive organs (testes) of developing and adult murine models, leading to lower testosterone levels and eventually poor sperm quality [166, 167].

1.3.2.8 ORGANOCHLORINE PESTICIDE

Among all classes of pesticides, OCPs have been regarded as the most dangerous. Even after being banned in most countries, their residues persist in wastewater and may pose a hazard. These include cyclodienes (dieldrin, endrin, endosulfan), cyclohexane (hexachlorocyclohexane, HCH), diphenyl aliphatics, DDT, and DDT metabolites [7, 33]. While the levels in wastewater are considered safe, the presence of other toxic compounds alongside OCPs can synergize and lead to greater adverse effects [168]. OCPs primarily exert their lethal adverse effects through synaptic dysregulation, involving voltage-gated channels, transporters, and receptors in the CNS. Similar to PYs, OCPs target the Na^+ voltage-gated channel and disrupt the balance of neural communication.

Besides, intoxication from OCPs can occur through a concept called "toxicant-induced loss of tolerance" [169], where these chemicals (i.e., OCPs) compromise the body's resilience and tolerance, leading to hypersensitivity in those individuals. Hence, subsequent exposure to low levels of unrelated antigens initiates and triggers immune responses, unfolding sensitivity-related illness [168, 169]. Additionally, OCPs interfere with human metabolic pathways via oxidative stress induction [170], endocrine-disrupting effects [171], mitochondrial dysfunction [172], and epigenetic modifications [173]. Even subtle, long-term exposures to OCPs can rewrite the metabolic pathways, significantly altering carbohydrate metabolism [174]. A temporal analysis over 12 years substantiates the metabolic disturbance effect, portraying that an OCP exposure may directly/indirectly provoke metabolic dysregulation [175]. The study also identified certain OCPs that may interfere with the homeostasis of uric acid and creatinine [175], adding complexity to their metabolic impact.

Other than metabolism-related illnesses, these mechanisms are known to contribute to a broad spectrum of diseases, for example, neurological disorders (neurodevelopmental, neurodegenerative, neurophysiological, neurobehavioral) [176–178], cancer [179–181], estrogen-dependent disease (endometriosis) [182], immune system dysregulation (autoimmunity) [183, 184], and reproductive system issues [185]. Digging deeper, it could be predicted that these persistent pollutants may pose a high risk to human health [186–189]. Their presence unfolds a complex tale, affecting various aspects of our well-being over time. A summary of routes of pesticide contamination in wastewater, impacts on human health, manifested symptoms; implicated cells, organs, and systems, as well as their mechanisms of toxicity, are enumerated in Figure 1.2 and Table 1.1.

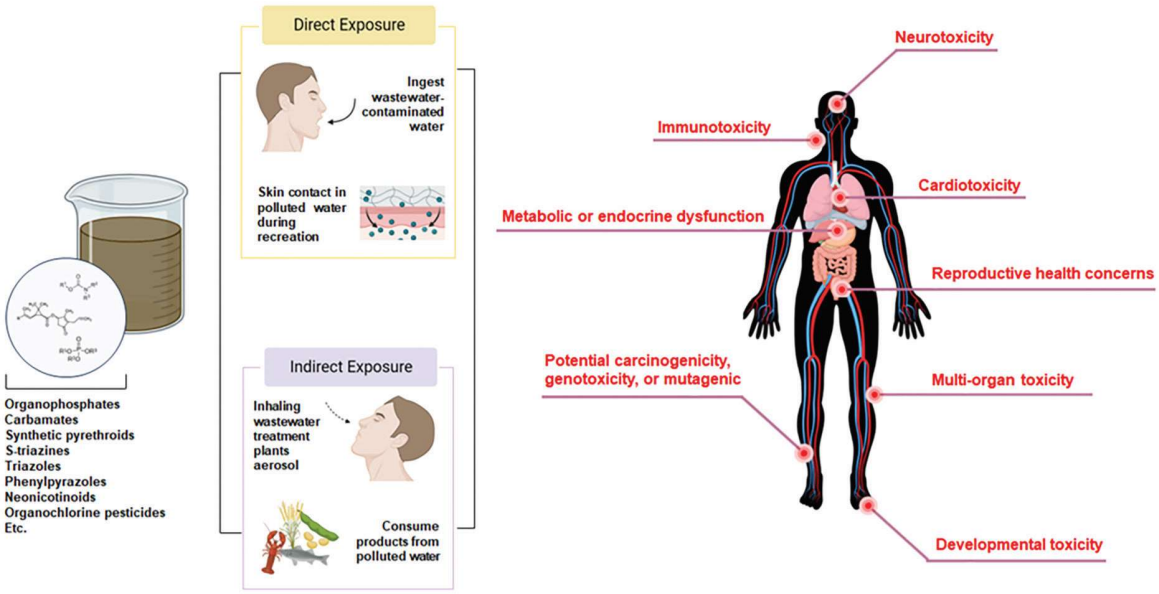


FIGURE 1.2 Pathways of exposure to pesticide residues in wastewater and potential implications on human health.

TABLE 1.1 Potential Health Risks from Pesticide-Polluted Wastewater

Chemical Class	Detected Contaminants	Health Hazards	References
Organophosphate	Tributyl phosphate, malathion, glyphosate, glyphosate metabolite (aminomethylphosphonic acid), chlorpyrifos.	• Potent neurotoxic effects. • Reproductive issues. • Endocrine disruption. • Potential carcinogenicity (hormonally-mediated cancers). • Neurological disorder (impact behaviors, emotions, cognitive functions). • Impaired immune function. • Disrupted metabolic pathways. • Adverse cardiovascular effects.	[24, 47–50, 56, 62, 64–66, 69, 73, 78]

TABLE 1.1 *(Continued)*

Chemical Class	Detected Contaminants	Health Hazards	References
Carbamate	Oxamyl, methomyl	• Neurotoxicity. • Potential genotoxicity. • Endocrine disruption. • Reproductive issues. • Developmental defects. • Negative impact on emotions; linked to depression, anxiety, irritability. • Potential immunotoxicity; linked to autoimmunity, hypersensitivity reactions. • Potential carcinogenicity; risks of development of specific cancers. • Multi-organ toxicity.	[26, 80, 82, 88, 92, 93, 97, 99, 100]
Synthetic pyrethroid	Permethrin, cypermethrin, cyhalothrin, cyfluthrin, deltamethrin.	• Neurotoxicity; associated with type-I and type-II syndromes. • Neurological disorders (impact neurological functions and cognition). • Impaired metabolic pathways. • Epigenetic modification. • Endocrine disruption. • Genotoxicity. • Potential carcinogenicity.	[27, 28, 109–112]
S-triazine	Atrazine, simetryn, prometryn	• Neurotoxicity. • Endocrine disruption. • Reproductive issues. • Developmental toxicity. • Influence on CYP enzymes (CYP1A2, 2A6, 2B6, 3A4). • Risk of kidney injury.	[24, 29, 114, 115, 118, 120, 121]
Triazole	Triticonazole, myclobutanil	• Neurotoxicity. • Affecting neurodevelopment. • Embryotoxicity. • Impaired retinoic acid metabolism. • Endocrine disruption. • Reproductive issues. • Carcinogenicity; linked to the development of specific cancers.	[30, 127–129, 134, 135]
Phenylpyrazole	Fipronil, fipronil metabolites (fipronil sulfone, fipronil sulfide)	• Neurotoxicity. • Influence on emotion. • Endocrine disruption. • Potent genotoxicity. • Reproductive issues. • Liver, kidney, and lung injuries.	[31, 144–148, 150, 151]

TABLE 1.1 *(Continued)*

Chemical Class	Detected Contaminants	Health Hazards	References
Neonicotinoid	Imidacloprid, acetamiprid, acetamiprid metabolite (acetamiprid-*N*-desmethyl), clothianidin.	• Neurotoxicity. • Neurological disorders (encompassing neuromorphological, neurodevelopmental, neurobehavioral). • Endocrine disruption. • Potential genotoxicity. • Immunotoxicity. • Reproductive issues. • Multi-organ toxicity.	[152, 156–158, 160, 163, 165, 166]
Organochlorine pesticide	Dieldrin, endrin, endosulfan, hexachlorocyclohexane, dichlorodiphenyltrichloroethane.	• Potent neurotoxicity. • Impaired metabolic pathways. • Neurological disorders (encompassing neurodevelopmental, neurodegenerative, neurophysiological, neurobehavioral). • Carcinogenicity. • Epigenetic modifications. • Sensitivity-related illness. • Risk of endometriosis. • Immunotoxicity; linked to autoimmunity. • Reproductive issues. • Risk of vitamin D insufficiency. • Risk of gallstone disease.	[7, 33, 170, 175–179, 182, 183, 185–187, 189]

1.4 METHODS FOR ASSESSING AND MONITORING PESTICIDE RESIDUE IN WASTEWATER

Monitoring PRs in wastewater is paramount for understanding their presence, assessing environmental risks, and developing effective management strategies. There are several methods employed for assessing and monitoring PRs in wastewater, emphasizing their importance in maintaining environmental vigilance and safeguarding water quality. Some of these methods are discussed in subsections.

1.4.1 ANALYTICAL TECHNIQUES

Chromatographic methods are relevant in analyzing pesticides in wastewater, providing precise and efficient means of separating and quantifying individual pesticide compounds. High-Performance Liquid Chromatography (HPLC) is widely employed for its exceptional precision. It remains highly relevant for the identification, isolation, and quantification of diverse pesticide compounds. It excels in the separation and quantification of diverse pesticide compounds. When coupled with detectors like UV-Vis or mass

spectrometry, HPLC enhances its analytical capabilities, offering high sensitivity and selectivity [190–193]. This makes it particularly advantageous in detecting trace levels of pesticides in complex matrices, ensuring accurate and reliable results. On the other hand, Gas Chromatography (GC) is highly effective for volatile and semi-volatile pesticides. GC separates compounds based on their vaporization characteristics, making it suitable for analyzing pesticides with varying volatility profiles. When coupled with mass spectrometry, GC further enhances its analytical power, allowing for the identification of individual pesticides with high accuracy [194–196].

In the realm of Mass Spectrometry (MS), two prominent techniques stand out: Liquid Chromatography-Mass Spectrometry (LC-MS) is an important analytical method that combines the separation capabilities of chromatography with the specificity of mass spectrometry. LC-MS excels in providing accurate identification and quantification of PRs. The chromatographic separation allows for the isolation of individual compounds, and the mass spectrometer precisely identifies these compounds based on their mass-to-charge ratio. This technique is particularly valuable for complex samples, offering high sensitivity and selectivity [197–199]. Additionally, Gas Chromatography-Mass Spectrometry (GC-MS) is specialized for volatile compounds. The gas chromatograph separates volatile pesticides, and the mass spectrometer provides detailed information about the chemical structure of these compounds. GC–MS enhances sensitivity, making it a valuable tool for the identification of volatile PRs in environmental and agricultural samples [200].

Collectively, these chromatographic and mass spectrometric techniques form a robust toolkit for comprehensive PR analysis. Their precision, sensitivity, and versatility make them essential for the accurate assessment of the quality and safety of agricultural products and environmental samples. By employing these sophisticated analytical methods, researchers, and regulatory authorities can effectively monitor and eventually control PRs, contributing to the overall safety and sustainability of food production and environmental management [201–203].

1.4.2 BIOSENSOR TECHNOLOGIES

Two key methods for PR analysis are Enzyme-Linked Immunosorbent Assay (ELISA) and Biosensor Platforms. ELISA is a rapid and cost-effective technique relying on antigen-antibody interactions. It furnishes both qualitative and quantitative assessments of PRs, rendering it particularly suitable for large-scale monitoring programs. On the other front, Biosensor Platforms encompass diverse technologies, including electrochemical and optical biosensors, providing real-time monitoring capabilities. These biosensors leverage enzymes or antibodies for the selective detection of specific PRs, offering a dynamic and versatile approach to residue analysis [204, 205].

ELISA, characterized by its speed and cost-effectiveness, hinges on the interactions between antigens and antibodies. It facilitates both qualitative and quantitative evaluations of PRs, making it ideal for extensive monitoring initiatives. Biosensor Platforms, embracing various technologies like electrochemical and optical biosensors, contribute real-time monitoring capabilities to PR analysis [206, 207]. These platforms commonly employ

enzymes or antibodies for the selective detection of specific residues, presenting a flexible and responsive approach to the dynamic landscape of residue monitoring. Together, these methods form a comprehensive toolkit for efficient and adaptable PR analysis, catering to diverse monitoring needs in agriculture and environmental management [208–211].

1.4.3 EMERGING TECHNOLOGIES

Two cutting-edge techniques for PR detection are nanotechnology and metabolomics. Nanoparticle-based sensors, a facet of nanotechnology, exhibit great potential for detecting minute amounts of PRs. Leveraging nanomaterials, these sensors significantly enhance sensitivity and selectivity, providing innovative solutions for real-time monitoring. The application of nanotechnology in PR analysis opens avenues for precise and efficient detection, addressing the challenges associated with trace-level contamination in various environments [212–214].

Metabolomics, as an approach in pesticide analysis, involves studying the comprehensive metabolic profile of organisms exposed to PRs. This holistic perspective offers valuable insights into the systemic impact of pesticides on organisms within aquatic ecosystems. By examining the entire spectrum of metabolic changes, metabolomics provides a nuanced understanding of how pesticides influence the overall health and functioning of organisms. Integrating metabolomics into pesticide analysis enriches our ability to assess ecological impacts, contributing to a more comprehensive and informed approach to environmental monitoring [216–219]. Together, nanotechnology, and metabolomics represent sophisticated methodologies that enhance the precision and depth of PR analysis, addressing the evolving challenges in environmental and agricultural contexts.

1.4.4 SAMPLING STRATEGIES, REMOTE SENSING, DATA INTEGRATION, AND MODELLING

Two innovative methodologies for environmental monitoring are passive sampling and automated sampling systems. Passive samplers, exemplified by solid-phase microextraction (SPME) fibers, are strategically deployed in water bodies to accumulate PRs over time. These samplers offer an integrated snapshot of pesticide concentrations, providing a cost-effective and efficient means of monitoring environmental contamination. On the other hand, automated sampling systems revolutionize data collection by enabling continuous or programmed sampling. This adaptability ensures representative data collection over varying time intervals, making these systems particularly beneficial in dynamic environments with fluctuating pesticide concentrations. The combination of passive sampling and automated sampling systems enhances the precision and reliability of pesticide monitoring, catering to the evolving demands of environmental assessment [216, 220, 221].

In the realm of Remote Sensing, satellite-based approaches and Data Integration and Modeling strategies contribute significantly to environmental monitoring efforts. Satellite Imaging provides a valuable tool for observing land use patterns and identifying areas

with high pesticide usage. While indirect, this approach furnishes essential information for targeting and optimizing monitoring efforts. Data Integration, facilitated by Geographic Information Systems (GIS), harmonizes spatial and temporal data. This integration enables a better understanding of the circulation and movement of PRs in wastewater, offering insights crucial for effective monitoring and management. Additionally, Modelling approaches, such as fate and transport models, enhance predictive capabilities. These mathematical models consider hydrodynamics, sedimentation, and degradation kinetics to forecast the dispersion of PRs in water bodies [222–224]. Remote Sensing and Data Integration and Modeling methodologies empower environmental scientists with valuable tools for proactive and informed decision-making in PR management. The summary of some currently available methods/strategies for assessing and monitoring PRs in wastewater is presented in Table 1.2.

1.5 TREATMENT OF PESTICIDES IN WASTEWATER

Pesticide residues can be treated in wastewaters through several methods, including chemical treatment such as electro-oxidation, Fenton oxidation, hydrolysis, rotavapor distillation, ozone/ultraviolet application, and photoremediation; physical treatment such as the application of organic/inorganic absorbents (e.g., activated carbon); aerobic/anaerobic biological treatment such as bioaugmentation, bioremediation, composting, and phytoremediation; and incineration treatment, which involves open burning and incinerator usage [3, 14].

Organophosphorus pesticides such as monocrotophos and pirimiphos-methyl groups can be removed from wastewater efficiently via the application of polyethyleneimine-modified fabrics [225].

The cumulative impact of industrial activities on pesticide accumulation in wastewater underscores the importance of stringent regulations and responsible industrial practices. Efforts to minimize discharges, enhance spill prevention and response measures, and promote proper disposal practices are essential components of mitigating the industrial contribution to PR build-up [226].

Treatment of PRs in wastewater is a critical step in mitigating environmental contamination and safeguarding water quality. This exploration delves into the diverse array of treatment methods, encompassing biological, chemical, and physical approaches (Table 1.2).

1.5.1 BIOLOGICAL TREATMENT

Bioremediation, a key biological treatment, utilizes microorganisms, notably bacteria and fungi, to enzymatically degrade PRs into less harmful compounds. Microbes are crucial for this degradation process, transforming pesticides through metabolic pathways and thereby reducing their toxicity [227–229]. Two effective strategies within bioremediation include biostimulation, which involves the provision of growth-promoting nutrients/factors that

TABLE 1.2 Overview of Assessment and Monitoring Methods for Pesticide Residues

Techniques	Procedures	Remarks	References
Analytical	• Liquid chromatography-coupled detector. • [LC-MS (ESI), LC-HRMS/MS, LC-MS/MS (EI-QqQ), LC-HRMS/MS (ESI-Q-TOF), LC-MS/MS (ESI-QqLIT), HPLC-MS, UPLC-MS/MS (EI-QqQ), UHPLC-MS/MS (EI-QqQ)]. • Gas-chromatography-coupled detector. • [GC-MS, GC-ECD, GC-MS (EI-Q), GC-NPD, GC-MS.MS (EI-QqQ)].	• High sensitivity and selectivity. • High precision and versatility.	[192, 197–200]
Biosensor	• Enzyme-linked immunosorbent assay. • Biosensor platforms [electrochemical, optical].	• Rapid. • Cost effective. • Large scale applicability. • Real time monitoring.	[204–207]
Emerging	• Nanoparticle-based sensors. • Metabolomic approaches.	• High sensitivity and selectivity. • Real time and high-precision monitoring. • Comprehensive assessment of ecological/environmental impacts.	[216–219]
Sampling	• Passive sampling [solid phase microextraction fibers]. • Automated sampling systems.	• Cost effective. • High performance. • Beneficial in dynamic environments.	[216, 220, 221]
Remote sensing	• Satellite imaging. • Data integration (facilitated by geographic information systems). • Modelling approaches [fate and transport models].	• Effective for land use analysis to identify patterns of pesticide usage. • Harmonization of spatial and temporal data. • Enhance predictive capabilities and support informed decision-making.	[222–224]

enhance microbial activity, and bioaugmentation, which introduces specialized microbial cultures. These techniques optimize conditions, accelerating the bioremediation process and facilitating efficient PR degradation [230, 231].

In microbial degradation, microorganisms, particularly bacteria and fungi, play a pivotal role in breaking down PRs into less harmful compounds through enzymatic transformations along metabolic pathways. This process significantly diminishes the toxicity of pesticides, contributing to environmental safety. Biostimulation and bioaugmentation are complementary techniques that enhance microbial activity within the bioremediation process. Biostimulation involves providing growth-promoting nutrients/factors to optimize conditions for microbial communities, while bioaugmentation introduces specialized microbial cultures, further accelerating the degradation of pesticides [232–234]. These

strategies collectively highlight the effectiveness of biological treatments, emphasizing the potential of harnessing microbial capabilities for sustainable PR removal from wastewater.

1.5.2 CHEMICAL TREATMENT

Chemical treatment methods for PR removal from wastewater encompass Advanced Oxidation Processes (AOPs), as well as adsorption and desorption. AOPs, including photodegradation, leverage reactive oxygen species generated by UV or sunlight exposure to effectively break down pesticide molecules. This process is efficient for degrading both parent compounds and their metabolites, converting them into less harmful byproducts. Ozone, a potent oxidizing agent, is applied in ozonation to break down PRs, forming reactive intermediates that react with and degrade pesticides, making it particularly effective for refractory compounds [235, 236]. Redox reactions encompass chemical reduction and Fenton/Photo-Fenton processes. In chemical reduction, reactive materials like zero-valent iron facilitate the transfer of electrons to transform pesticides into less toxic forms. This process shows promise in breaking down a variety of pesticide compounds. The Fenton reaction, involving hydrogen peroxide and iron, and its photo-enhanced counterpart (Photo-Fenton), creates hydroxyl radicals that oxidize and degrade PRs in wastewater [237–239].

Adsorption and desorption methods involve the application of organic/inorganic materials such as activated carbon and certain clay minerals for pesticide removal. Activated carbon, with its high surface area and adsorptive capacity, effectively captures pesticides from wastewater. Desorption processes can then be employed to regenerate activated carbon for reuse. Similarly, certain clay minerals, such as montmorillonite and kaolinite, exhibit adsorption capabilities for pesticide removal. The adsorbed pesticides can be subsequently desorbed or degraded, presenting a comprehensive treatment approach [240, 241].

These chemical treatment methods collectively provide effective and versatile approaches to address the complex challenge of PR removal, ensuring the safety of water resources and environmental well-being.

1.5.3 PHYSICAL TREATMENT

Physical treatment methods for PR removal from wastewater primarily involve membrane filtration and sorption/ion exchange techniques. Membrane filtration, a pivotal physical treatment, utilizes techniques like reverse osmosis and ultrafiltration to physically separate pesticide molecules based on their size and molecular weight. These methods are highly effective in removing both dissolved and suspended PRs from wastewater. Nanofiltration, a specialized form of membrane filtration, employs membranes with smaller pore sizes than ultrafiltration, allowing for the selective removal of pesticides while retaining essential components of the treated water [242, 243].

Sorption and ion exchange techniques further contribute to physical treatment approaches. Activated carbon sorption, building on its adsorption properties, surface functional groups, and high surface area, aids in sorption, where pesticides adhere to the

surface through non-covalent interactions [244]. Notably, this is the most efficient way and is commonly applied as it readily removes dissolved pesticides from water [244], but its widespread adoption is limited by cost considerations [245]. Lately, there has been a lot of focus on finding eco-friendly and economically viable sources. One promising option is using ash-based sorbents (plant biomass ashes), distinguished by their suitable particle size, large surface area, favorable organic carbon content, and lipophilicity [245, 246]. Other absorbents, such as clay and graphene, are suitable for use in water-based treatment methods [247].

Ion exchange resins, another effective method, selectively capture charged pesticide molecules, facilitating their removal from wastewater. The regeneration of these resins through desorption ensures sustained effectiveness, making ion exchange resins a valuable component of physical treatment strategies [248–250].

These methods not only physically separate and remove pesticide molecules but also demonstrate versatility in retaining essential components, contributing to the overall safety and quality of water resources. The various treatment strategies for PRs in wastewater systems, including the newly emerging technologies, are summarized in Table 1.3.

1.6 CHALLENGES IN WATER TREATMENT FOR DRINKING AND OTHER HUMAN APPLICATIONS

Ensuring the provision of safe and clean water is fundamental for public health, yet the water treatment process encounters various challenges that demand innovative solutions for sustainable water management. One major challenge is the presence of emerging contaminants, including some prominent pharmaceuticals and industrial chemical residues that conventional treatment methods cannot effectively remediate. Advanced treatment technologies such as AOPs and membrane filtration, coupled with continuous monitoring and research, offer solutions to enhance the removal of such contaminants [251–254].

Microbial contamination remains a persistent challenge, necessitating continuous improvement in disinfection technologies, regular water quality monitoring, and adherence to water treatment standards. Limited access to clean water, particularly in rural and underserved communities, calls for decentralized water treatment solutions, community engagement, and innovative financing models. Stringent water quality regulations and evolving standards challenge water treatment facilities to consistently meet compliance. Regular training, investment in monitoring systems, and proactive engagement with regulatory bodies are crucial for compliance maintenance [215, 255].

1.7 FUTURE PROSPECTS AND LIMITATIONS

Among the recently developed monitoring strategies, analytical techniques (HPLC, UV, GC-MS, and LC-MS), biosensor technique (ELISA), remote sensing, data modelling and integration and other emerging technologies including nanotechnologies have been effectively demonstrated. Efficient treatment strategies include biological (biostimulation and

TABLE 1.3 Pesticide Residue Treatment Strategies and Mechanisms

Treatment Properties	Methods	Mechanisms	References
Biological	• Biostimulation or bioaugmentation of microbes [bacteria, fungi].	• Contribution to the degradation process by converting PRs into less harmful metabolites.	[232–234]
Chemical	• Advanced oxidation processes [photo catalysis, Fenton based, ozonation, sonolysis, electrochemical oxidation].	• Creation of highly active hydroxyl radicals, effectively oxidizing a broad spectrum of PRs.	[21, 235, 236]
	• Adsorption and desorption [activated carbon, clay minerals].	• Efficient cleansing of environmentally impactful components, such as PRs in aqueous solutions, by physically entrapping or chemically binding them.	[235–237, 247]
Physical	• Membrane filtration [reverse osmosis, ultrafiltration, nanofiltration].	• Implementation of filtration processes using different membrane types at various stages, tailored to the unique properties of pesticides.	[242, 243]
–	• Sorption/ion exchange techniques.	• Adhesion of contaminants (PRs) to the compounds.	[244–248]

bioaugmentation), chemical methods (adsorption, desorption, ion exchange, redox reaction and photodegradation) and physical processes such as membrane filtration, sorption, and nanofiltration. However, most of the modern methods require adequate expertise and sophisticated equipment and are expensive, thereby limiting their applications.

Moreover, a substantial knowledge gap exists among individuals about the potential harm incurred when encountering pesticide-contaminated wastewater, contributing to a scenario where safety measures may be neglected. Since prevention is usually cheaper than cure, efforts to minimize discharges, enhance spill prevention and response measures, and promote proper disposal practices are essential ways of mitigating pesticide accumulation in wastewater. Thus, governments at various levels should continuously legislate regulations to keep the treatment of pesticide wastes in check and control their safe discharge into water bodies.

In addition, a regular overview of the updates on the application, discharge, monitoring, and remediation of the current and emerging PRs would facilitate the development of strategies for maintaining clean environments globally through wastewater.

1.8 CONCLUSION

The use of pesticides to enhance adequate food production is almost inevitable to cater to the global yearning for food. As such, the application of pesticides in homes, agricultural sites, and relevant industrial settings remains invaluable. For instance, it boosts food production and storage through agricultural activities, helps ward off unwanted pests at home, and supports economic growth through industrialization.

However, accidental spills during the manufacturing, application, handling, and transportation of pesticides, runoff through farmlands, improper disposal, and discharge of pesticide-containing wastes into water bodies inclusively lead to their accumulation in wastewater. Among the major classes of pesticides reportedly present in wastewater systems are organophosphates, organochlorines, carbamates, synthetic pyrethroids, S-triazines, triazoles, and neonicotinoids, as well as their derivatives and metabolites. The routes of concentration of pesticides in wastewater systems are intricate and complex. More importantly, their subsequent transformation into harmful products constitutes a major challenge, depending on chemical formulation; such transformations are mostly spontaneous and uncontrollable.

Consequently, environmental pollution and health risks emerged. Notably, the events of neurotoxicity, developmental problems, ataxia, severe headaches, and pain. Carcinogenicity, skin diseases, damage to key organs (heart, kidney, liver, etc.), impaired life-dependent systems (nervous, reproductive, respiratory, metabolic, and endocrine), and eventual mortality, especially upon chronic exposure, are strongly associated with the presence of pesticides in wastewater. Mechanistically, these occur due to the devastating interactions of PRs with the relevant biological targets such as cholinergic enzymes/receptors, ion-gated protein channels, gonadal/hypothalamic/pituitary axis, cytochrome P450 enzymes, immune regulatory genes, and cyclic adenosine 3,'5'-monophosphate pathways, among others. Therefore, effective monitoring and remediation strategies are required to manage the undesired consequential effects of pesticide production and usage. This chapter comprehensively reveals the fates of PRs in wastewater, including the health risks and preventive/remediating strategies for mitigating such inevitable but undesired events.

ACKNOWLEDGMENT

This project was financially supported by the Tertiary Education Fund Nigeria.

KEYWORDS

- **environmental hazards**
- **food production**
- **human health**
- **pesticide residues**
- **remediation techniques**
- **wastewater contamination**

REFERENCES

1. Li, K., Chen, W., Xiang, W., Chen, T., Zhang, M., Ning, Y., Liu, Y., & Chen, A. (2022). Determination, residue analysis, and risk assessment of thiacloprid and spirotetramat in cowpeas under field conditions. *Scientific Reports, 12*, 1–11. https://doi.org/10.1038/s41598-022-07119-1.
2. FAO. (2022). *World Food and Agriculture–Statistical Yearbook 2022*. Rome. https://doi.org/10.4060/cc2211en.
3. Al Hattab, M. T., & Ghaly, A. E. (2012). Disposal and treatment methods for pesticide-containing wastewaters: Critical review and comparative analysis. *Journal of Environmental Protection (Irvine, Calif.), 3*, 431–453. https://doi.org/10.4236/jep.2012.35054.
4. Government of British Columbia. (2017). *Pesticide Toxicity and Hazard*. https://www2.gov.bc.ca/assets/gov/farming-natural-resources-and-industry/agriculture-and-seafood/animal-and-crops/plant-health/pesticide-toxicity-hazard.pdf (accessed on 14 November 2024).
5. Jean, S. (2013). Contamination of cowpea and by-products by organophosphorous pesticide residues in Ngaoundere markets: Dietary risk estimation and degradation study. *African Journal of Food Science, 7*, 92–102. https://doi.org/10.5897/ajfs2013.1013.
6. Zhang, Q., Ma, C., Duan, Y., Wu, X., Lv, D., & Luo, J. (2022). Determination and dietary intake risk assessment of 35 pesticide residues in cowpea (*Vigna unguiculata* [L.] Walp) from Hainan province, China. *Scientific Reports, 12*, 1–11. https://doi.org/10.1038/s41598-022-09461-w.
7. Cybulski, J., Witczak, A., & Pokorska-Niewiada, K. (2021). The effect of water and sewage treatment on reducing residues of selected organochlorine pesticides in Szczecin (Poland). *Water, Air, & Soil Pollution, 232*, 1–6. https://doi.org/10.1007/s11270-021-05261-6.
8. Pathak, V. M., Verma, V. K., Rawat, B. S., Kaur, B., Babu, N., Sharma, A., Dewali, S., Yadav, M., Kumari, R., Singh, S., Mohapatra, A., Pandey, V., Rana, N., & Cunill, J. M. (2022). Current status of pesticide effects on environment, human health, and its eco-friendly management as bioremediation: A comprehensive review. *Frontiers in Microbiology, 13*, 1–29. https://doi.org/10.3389/fmicb.2022.962619.
9. Pérez-Lucas, G., Vela, N., El Aatik, A., & Navarro, S. (2018). Environmental risk of groundwater pollution by pesticide leaching through the soil profile. In *Pesticides: Use, Misuse, and Their Impact on the Environment* (pp. 1–27). https://doi.org/10.5772/intechopen.82418.
10. Boedeker, W., Watts, M., Clausing, P., & Marquez, E. (2020). The global distribution of acute unintentional pesticide poisoning: Estimations based on a systematic review. *BMC Public Health, 20*, 1–19. https://doi.org/10.1186/s12889-020-09939-0.
11. Samuel, P. O., Edo, G. I., Oloni, G. O., Ugbune, U., Ezekiel, G. O., Essaghah, A. E. A., & Agbo, J. J. (2023). Effects of chemical contaminants on the ecology and evolution of organisms: A review. *Chemical Ecology*, 1–37. https://doi.org/10.1080/02757540.2023.2284158.
12. Tudi, M., Ruan, H. D., Wang, L., Lyu, J., Sadler, R., Connell, D., Chu, C., & Phung, D. T. (2021). Agriculture development, pesticide application, and its impact on the environment. *International Journal of Environmental Research and Public Health, 18*, 1–23. https://doi.org/10.3390/ijerph18031112.
13. Mohd Ghazi, R., Nik Yusoff, N. R., Abdul Halim, N. S., Wahab, I. R. A., Ab Latif, N., Hasmoni, S. H., Ahmad Zaini, M. A., & Zakaria, Z. A. (2023). Health effects of herbicides and its current removal strategies. *Bioengineered, 14*, 1–22. https://doi.org/10.1080/21655979.2023.2259526.
14. Akinapally, S., Dheeravath, B., Panga, K. K., Vurimindi, H., & Sanaga, S. (2021). Treatment of pesticide intermediate industrial wastewater using hybrid methodologies. *Applied Water Science, 11*, 1–7. https://doi.org/10.1007/s13201-021-01387-4.
15. Carpio, M. J., Sánchez-Martín, M. J., Rodríguez-Cruz, M. S., & Marín-Benito, J. M. (2021). Effect of organic residues on pesticide behavior in soils: A review of laboratory research. *Environments, 8*, 1–28. https://doi.org/10.3390/environments8040032.
16. Kaur, T., & Sinha, A. K. (2019). Pesticides in agricultural run-offs affecting water resources: A study of Punjab (India). *Agricultural Sciences, 10*, 1381–1395. https://doi.org/10.4236/as.2019.1010101.
17. Terfe, A., Mekonen, S., & Jemal, T. (2023). Pesticide residues and effect of household processing in commonly consumed vegetables in Jimma Zone, Southwest Ethiopia. *Journal of Environmental and Public Health, 2023*, 1–12. https://doi.org/10.1155/2023/7503426.

18. Jemal, T., Astatke, H., Terfe, A., & Mekonen, S. (2023). Effect of conventional and household water treatment technologies on the removal of pesticide residues in drinking water, Jimma town, Southwestern, Ethiopia. *PLoS One, 18*, 1–14. https://doi.org/10.1371/journal.pone.0288086.
19. Leskovac, A., & Petrović, S. (2023). Pesticide use and degradation strategies: Food safety, challenges, and perspectives. *Foods, 12*, 1–24. https://doi.org/10.3390/foods12142709.
20. Osuoha, J. O., Anyanwu, B. O., & Ejileugha, C. (2023). Pharmaceuticals and personal care products as emerging contaminants: Need for combined treatment strategy. *Journal of Hazardous Materials Advances, 9*, 1–15. https://doi.org/10.1016/j.hazadv.2022.100206.
21. Syafrudin, M., Kristanti, R. A., Yuniarto, A., Hadibarata, T., Rhee, J., Al-onazi, W. A., Algarni, T. S., Almarri, A. H., & Al-Mohaimeed, A. M. (2021). Pesticides in drinking water–A review. *International Journal of Environmental Research and Public Health, 18*(2), 1–15. https://doi.org/10.3390/ijerph18020468.
22. Tzanetou, E. N., & Karasali, H. (2022). A comprehensive review of organochlorine pesticide monitoring in agricultural soils: The silent threat of a conventional agricultural past. *Agriculture, 12*, 1–65. https://doi.org/10.3390/agriculture12050728.
23. Silva, M., & Baltrusaitis, J. (2021). Destruction of emerging organophosphate contaminants in wastewater using the heterogeneous iron-based photo-Fenton-like process. *Journal of Hazardous Materials Letters, 2*, 100012. https://doi.org/10.1016/J.HAZL.2020.100012.
24. Rousis, N. I., Zuccato, E., & Castiglioni, S. (2016). Monitoring population exposure to pesticides based on liquid chromatography-tandem mass spectrometry measurement of their urinary metabolites in urban wastewater: A novel biomonitoring approach. *Science of The Total Environment, 571*, 1349–1357. https://doi.org/10.1016/J.SCITOTENV.2016.07.036.
25. Tabibi, A., & Jafari, M. T. (2020). Ultrasonic piezoelectric nebulization of propoxur for the determination by corona discharge ionization ion mobility spectrometry. *Analytical Sciences, 36*, 227–232. https://doi.org/10.2116/ANALSCI.19P262.
26. Comas, E., Gimeno, R. A., Ferré, J., Marcé, R. M., Borrull, F., & Rius, F. X. (2004). Quantification from highly drifted and overlapped chromatographic peaks using second-order calibration methods. *Journal of Chromatography A, 1035*, 195–202. https://doi.org/10.1016/J.CHROMA.2004.02.069.
27. Tang, W., Wang, D., Wang, J., Wu, Z., Li, L., Huang, M., Xu, S., & Yan, D. (2018). Pyrethroid pesticide residues in the global environment: An overview. *Chemosphere, 191*, 990–1007. https://doi.org/10.1016/J.CHEMOSPHERE.2017.10.115.
28. Firouzsalari, N. Z., Shakerkhatibi, M., Pourakbar, M., Yadeghari, A., Safari, G. H., & Sarbakhsh, P. (2019). Pyrethroid pesticide residues in a municipal wastewater treatment plant: Occurrence, removal efficiency, and risk assessment using a modified index. *Journal of Water Process Engineering, 29*, 100793. https://doi.org/10.1016/J.JWPE.2019.100793.
29. Wang, M., Lv, J., Deng, H., Liu, Q., & Liang, S. (2022). Occurrence and removal of triazine herbicides during wastewater treatment processes and their environmental impact on aquatic life. *International Journal of Environmental Research and Public Health, 19*. https://doi.org/10.3390/IJERPH19084557/S1.
30. Parlakidis, P., Gounari, I., Georgiou, A., Adamidis, G., Vryzas, Z., & Gikas, G. D. (2023). Removal of two triazole fungicides from agricultural wastewater in pilot-scale horizontal subsurface flow constructed wetlands. *Agronomy, 13*, 265. https://doi.org/10.3390/AGRONOMY13010265.
31. Sadaria, A. M., Sutton, R., Moran, K. D., Teerlink, J., Brown, J. V., & Halden, R. U. (2017). Passage of fiproles and imidacloprid from urban pest control uses through wastewater treatment plants in northern California, USA. *Environmental Toxicology and Chemistry, 36*, 1473–1482. https://doi.org/10.1002/ETC.3673.
32. Sadaria, A. M., Supowit, S. D., & Halden, R. U. (2016). Mass balance assessment for six neonicotinoid insecticides during conventional wastewater and wetland treatment: Nationwide reconnaissance in United States wastewater. *Environmental Science & Technology, 50*, 6199–6206. https://doi.org/10.1021/ACS.EST.6B01032/ASSET/IMAGES/LARGE/ES-2016-01032E_0003.JPEG.
33. Nnaji, J. C., Amaku, J. F., Amadi, O. K., & Nwadinobi, S. I. (2023). Evaluation and remediation protocol of selected organochlorine pesticides and heavy metals in industrial wastewater using nanoparticles (NPs) in Nigeria. *Scientific Reports, 13*, 1–14. https://doi.org/10.1038/s41598-023-28761-3.

34. Damalas, C. A., & Eleftherohorinos, I. G. (2011). Pesticide exposure, safety issues, and risk assessment indicators. *International Journal of Environmental Research and Public Health, 8*, 1402–1419. https://doi.org/10.3390/ijerph8051402.
35. Kumar, V., Agrawal, S., Bhat, S. A., Américo-Pinheiro, J. H. P., Shahi, S. K., & Kumar, S. (2022). Environmental impact, health hazards, and plant-microbes synergism in remediation of emerging contaminants. *Clean Chemistry and Engineering, 2*, 1–15. https://doi.org/10.1016/j.clce.2022.100030.
36. Qaiser, Z., Aqeel, M., Sarfraz, W., Rizvi, Z. F., Noman, A., Naeem, S., & Khalid, N. (2023). Microplastics in wastewaters and their potential effects on aquatic and terrestrial biota. *Case Studies in Chemical and Environmental Engineering, 8*, 1–16. https://doi.org/10.1016/j.cscee.2023.100536.
37. Lu, Y., Song, S., Wang, R., Liu, Z., Meng, J., Sweetman, A. J., Jenkins, A., Ferrier, R. C., Li, H., Luo, W., & Wang, T. (2015). Impacts of soil and water pollution on food safety and health risks in China. *Environment International, 77*, 5–15. https://doi.org/10.1016/J.ENVINT.2014.12.010.
38. Masiá, A., Campo, J., Vázquez-Roig, P., Blasco, C., & Picó, Y. (2013). Screening of currently used pesticides in water, sediments and biota of the Guadalquivir River Basin (Spain). *Journal of Hazardous Materials, 263*(Pt 1), 95–104. https://doi.org/10.1016/J.JHAZMAT.2013.09.035.
39. Godejohann, M., Berset, J. D., & Muff, D. (2011). Non-targeted analysis of wastewater treatment plant effluents by high performance liquid chromatography-time slice-solid phase extraction-nuclear magnetic resonance/time-of-flight-mass spectrometry. *Journal of Chromatography A, 1218*, 9202–9209. https://doi.org/10.1016/J.CHROMA.2011.10.051.
40. Sayed, A., Chys, M., De Rop, J., Goeteyn, L., Spanoghe, P., & Sampers, I. (2021). Pesticide residues in (treated) wastewater and products of Belgian vegetable- and potato-processing companies. *Chemosphere, 280*, 130619. https://doi.org/10.1016/j.chemosphere.2021.130619.
41. Anumol, T., Vijayanandan, A., Park, M., Philip, L., & Snyder, S. A. (2016). Occurrence and fate of emerging trace organic chemicals in wastewater plants in Chennai, India. *Environmental International, 92–93*, 33–42. https://doi.org/10.1016/j.envint.2016.03.022.
42. Sharma, S., & Bhattacharya, A. (2016). Drinking water contamination and treatment techniques. *Applied Water Science, 7*(1), 1043–1067. https://doi.org/10.1007/s13201-016-0455-7.
43. Galindo-Miranda, J. M., Guízar-González, C., Becerril-Bravo, E. J., Moeller-Chávez, G., León-Becerril, E., & Vallejo-Rodríguez, R. (2019). Occurrence of emerging contaminants in environmental surface waters and their analytical methodology–A review. *Water Supply, 19*, 1871–1884. https://doi.org/10.2166/ws.2019.087.
44. Munz, N. A., Burdon, F. J., de Zwart, D., Junghans, M., Melo, L., Reyes, M., Schönenberger, U., Singer, H. P., Spycher, B., Hollender, J., & Stamm, C. (2017). Pesticides drive risk of micropollutants in wastewater-impacted streams during low flow conditions. *Water Research, 110*, 366–377. https://doi.org/10.1016/j.watres.2016.11.001.
45. Carvalho, F. P. (2017). Pesticides, environment, and food safety. *Food and Energy Security, 6*(1), 48–60. https://doi.org/10.1002/fes3.108.
46. Yang, T., Han, Y., Liu, J., & Li, L. (2019). Aerosols from a wastewater treatment plant using oxidation ditch process: Characteristics, source apportionment, and exposure risks. *Environmental Pollution, 250*, 627–638. https://doi.org/10.1016/j.envpol.2019.04.071.
47. Fries, E., & Püttmann, W. (2003). Monitoring of the three organophosphate esters TBP, TCEP, and TBEP in river water and groundwater (Oder, Germany). *Journal of Environmental Monitoring, 5*(3), 346–352. https://doi.org/10.1039/b210342g.
48. Zhang, Y., & Pagilla, K. (2010). Treatment of malathion pesticide wastewater with nanofiltration and photo-Fenton oxidation. *Desalination, 263*(1), 36–44. https://doi.org/10.1016/j.desal.2010.06.031.
49. Kolpin, D. W., Thurman, E. M., Lee, E. A., Meyer, M. T., Furlong, E. T., & Glassmeyer, S. T. (2006). Urban contributions of glyphosate and its degradate AMPA to streams in the United States. *Science of the Total Environment, 354*, 191–197. https://doi.org/10.1016/j.scitotenv.2005.01.028.
50. Poiger, T., Keller, M., Buerge, I. J., & Balmer, M. E. (2020). Behavior of glyphosate in wastewater treatment plants. *Chimia (Aarau), 74*, 156. https://doi.org/10.2533/chimia.2020.156.

51. Moore, P. D., Yedjou, C. G., & Tchounwou, P. B. (2010). Malathion-induced oxidative stress, cytotoxicity, and genotoxicity in human liver carcinoma (HepG2) cells. *Environmental Toxicology, 25*(3), 221. https://doi.org/10.1002/tox.20492.
52. Robb, E. L., Regina, A. C., & Baker, M. B. (2023). Organophosphate toxicity. *StatPearls Publishing*.
53. National Research Council. (2002). Health considerations related to chemical contaminants and physical factors. In *Airliner Cabin Environment and Health of Passengers and Crew*. National Academies Press (US).
54. New Jersey Department of Health and Senior Service. (2000). *Hazardous Substance Fact Sheet of Tributyl Phosphate*.
55. Carrington, C. D., Lapadula, D. M., Othman, M., Farr, C., Nair, R. S., Johannsen, F., & Abou-Donia, M. B. (1990). Assessment of the delayed neurotoxicity of tributyl phosphate, tributoxyethyl phosphate, and dibutylphenyl phosphate. *Journal of Toxicology and Environmental Health, 6*(3), 415–423. https://doi.org/10.1177/074823379000600305.
56. Liu, J., Lin, H., Dong, Y., & Li, B. (2019). Elucidating the biodegradation mechanism of tributyl phosphate (TBP) by *Sphingomonas* sp. isolated from TBP-contaminated mine tailings. *Environmental Pollution, 250*, 284–291. https://doi.org/10.1016/j.envpol.2019.03.127.
57. Muniz, J. F., McCauley, L., Scherer, J., Lasarev, M., Koshy, M., Kow, Y. W., Nazar-Stewart, V., & Kisby, G. E. (2008). Biomarkers of oxidative stress and DNA damage in agricultural workers: A pilot study. *Toxicology and Applied Pharmacology, 227*(1), 97–107. https://doi.org/10.1016/j.taap.2007.10.027.
58. Kwiatkowska, M., Huras, B., & Bukowska, B. (2014). The effect of metabolites and impurities of glyphosate on human erythrocytes (*in vitro*). *Pesticide Biochemistry and Physiology, 109*, 34–43. https://doi.org/10.1016/j.pestbp.2014.01.003.
59. Sidthilaw, S., Sapbamrer, R., Pothirat, C., Wunnapuk, K., & Khacha-ananda, S. (2022). Effects of exposure to glyphosate on oxidative stress, inflammation, and lung function in maize farmers, Northern Thailand. *BMC Public Health, 22*, 1–10. https://doi.org/10.1186/s12889-022-13696-7.
60. Makris, K. C., Efthymiou, N., Konstantinou, C., Anastasi, E., Schoeters, G., Kolossa-Gehring, M., & Katsonouri, A. (2022). Oxidative stress of glyphosate, AMPA and metabolites of pyrethroids and chlorpyrifos pesticides among primary school children in Cyprus. *Environmental Research, 212*, 113316. https://doi.org/10.1016/j.envres.2022.113316.
61. Moreira, S., Pereira, S. C., Seco-Rovira, V., Oliveira, P. F., Alves, M. G., & de L. Pereira, M. (2021). Pesticides and male fertility: A dangerous crosstalk. *Metabolites, 11*(12), 799. https://doi.org/10.3390/METABO11120799.
62. Ghafouri-Khosrowshahi, A., Ranjbar, A., Mousavi, L., Nili-Ahmadabadi, H., Ghaffari, F., Zeinvand-Lorestani, H., & Nili-Ahmadabadi, A. (2019). Chronic exposure to organophosphate pesticides as an important challenge in promoting reproductive health: A comparative study. *Journal of Education and Health Promotion, 8*, 148. https://doi.org/10.4103/JEHP.JEHP_148_19.
63. Yang, K. J., Lee, J., & Park, H. L. (2020). Organophosphate pesticide exposure and breast cancer risk: A rapid review of human, animal, and cell-based studies. *International Journal of Environmental Research and Public Health, 17*(14), 1–29. https://doi.org/10.3390/IJERPH17145030.
64. Calaf, G. M., Bleak, T. C., & Roy, D. (2021). Signs of carcinogenicity induced by parathion, malathion, and estrogen in human breast epithelial cells. *Oncology Reports, 45*, 7975. https://doi.org/10.3892/OR.2021.7975.
65. dos Santos, A. A., Naime, A. A., de Oliveira, J., Colle, D., dos Santos, D. B., Hort, M. A., Moreira, E. L. G., Suñol, C., de Bem, A. F., & Farina, M. (2016). Long-term and low-dose malathion exposure causes cognitive impairment in adult mice: Evidence of hippocampal mitochondrial dysfunction, astrogliosis, and apoptotic events. *Archives of Toxicology, 90*(4), 647–660. https://doi.org/10.1007/S00204-015-1466-0.
66. Mostafalou, S., & Abdollahi, M. (2013). Pesticides and human chronic diseases: Evidences, mechanisms, and perspectives. *Toxicology and Applied Pharmacology, 268*(2), 157–177. https://doi.org/10.1016/J.TAAP.2013.01.025.
67. Eriguchi, M., Iida, K., Ikeda, S., Osoegawa, M., Nishioka, K., Hattori, N., Nagayama, H., & Hara, H. (2019). Parkinsonism relating to intoxication with glyphosate. *Internal Medicine, 58*(13), 1935–1938. https://doi.org/10.2169/INTERNALMEDICINE.2028-18.

68. Bloem, B. R., & Boonstra, T. A. (2023). The inadequacy of current pesticide regulations for protecting brain health: The case of glyphosate and Parkinson's disease. *The Lancet Planetary Health*. https://doi.org/10.1016/S2542-5196(23)00255-3.
69. Rajak, P., Ganguly, A., Sarkar, S., Mandi, M., Dutta, M., Podder, S., Khatun, S., & Roy, S. (2021). Immunotoxic role of organophosphates: An unseen risk escalating SARS-CoV-2 pathogenicity. *Food and Chemical Toxicology, 149*, 112007. https://doi.org/10.1016/J.FCT.2021.112007.
70. Bernal-González, K. G., Covantes-Rosales, C. E., Camacho-Pérez, M. R., Mercado-Salgado, U., Barajas-Carrillo, V. W., Girón-Pérez, D. A., Montoya-Hidalgo, A. C., Díaz-Resendiz, K. J. G., Barcelos-García, R. G., Toledo-Ibarra, G. A., & Girón-Pérez, M. I. (2023). Organophosphate-pesticide-mediated immune response modulation in invertebrates and vertebrates. *International Journal of Molecular Sciences, 24*(6), 5360. https://doi.org/10.3390/IJMS24065360.
71. Saldana, T. M., Basso, O., Hoppin, J. A., Baird, D. D., Knott, C., Blair, A., Alavanja, M. C. R., & Sandler, D. P. (2007). Pesticide exposure and self-reported gestational diabetes mellitus in the Agricultural Health Study. *Diabetes Care, 30*(3), 529–534. https://doi.org/10.2337/DC06-1832.
72. Montgomery, M. P., Kamel, F., Saldana, T. M., Alavanja, M. C. R., & Sandler, D. P. (2008). Incident diabetes and pesticide exposure among licensed pesticide applicators: Agricultural Health Study, 1993–2003. *American Journal of Epidemiology, 167*(10), 1235–1246. https://doi.org/10.1093/AJE/KWN028.
73. Everett, C. J., & Matheson, E. M. (2010). Biomarkers of pesticide exposure and diabetes in the 1999–2004 national health and nutrition examination survey. *Environment International, 36*(4), 398–401. https://doi.org/10.1016/J.ENVINT.2010.02.010.
74. Chung, Y. L., Hou, Y. C., Wang, I. K., Lu, K. C., & Yen, T. H. (2021). Organophosphate pesticides and new-onset diabetes mellitus: From molecular mechanisms to a possible therapeutic perspective. *World Journal of Diabetes, 12*(11), 1818–1831. https://doi.org/10.4239/WJD.V12.I11.1818.
75. Zhao, L., Liu, Q., Jia, Y., Lin, H., Yu, Y., Chen, X., Liu, Z., Li, W., Fang, T., Jiang, W., Zhang, J., Cui, H., Li, P., Li, H., Hou, S., & Guo, L. (2023). The associations between organophosphate pesticides (OPs) and respiratory disease, diabetes mellitus, and cardiovascular disease: A review and meta-analysis of observational studies. *Toxics, 11*(9), 741. https://doi.org/10.3390/TOXICS11090741/S1.
76. Hayden, K. M., Norton, M. C., Darcey, D., Østbye, T., Zandi, P. P., Breitner, J. C. S., & Welsh-Bohmer, K. A. (2010). Occupational exposure to pesticides increases the risk of incident AD: The Cache County Study. *Neurology, 74*(19), 1524. https://doi.org/10.1212/WNL.0B013E3181DD4423.
77. Parrón, T., Requena, M., Hernández, A. F., & Alarcón, R. (2011). Association between environmental exposure to pesticides and neurodegenerative diseases. *Toxicology and Applied Pharmacology, 256*(3), 379–385. https://doi.org/10.1016/J.TAAP.2011.05.006.
78. Rueda-Ruzafa, L., Cruz, F., Roman, P., & Cardona, D. (2019). Gut microbiota and neurological effects of glyphosate. *Neurotoxicology, 75*, 1–8. https://doi.org/10.1016/J.NEURO.2019.08.006.
79. Jaga, K., & Dharmani, C. (2007). The interrelation between organophosphate toxicity and the epidemiology of depression and suicide. *Reviews on Environmental Health, 22*(1), 57–73. https://doi.org/10.1515/REVEH.2007.22.1.57.
80. Lin, Z., Zhang, W., Pang, S., Huang, Y., Mishra, S., Bhatt, P., & Chen, S. (2020). Current approaches to and future perspectives on methomyl degradation in contaminated soil/water environments. *Molecules, 25*(3), 738. https://doi.org/10.3390/MOLECULES25030738.
81. Pope, C. N. (2005). *Methomyl*. Elsevier. https://doi.org/10.1016/B0-12-369400-0/00605-0.
82. New Jersey Department of Health and Senior Service. (1999). *Hazardous Substance Fact Sheet of Oxamyl*.
83. Mortensen, S. R., & Serex, T. L. (2014). *Methomyl*. Academic Press. https://doi.org/10.1016/B978-0-12-386454-3.00161-5.
84. Silberman, J., & Taylor, A. (2023). Carbamate toxicity. In *StatPearls*. StatPearls Publishing.
85. Rosman, Y., Makarovsky, I., Bentur, Y., Shrot, S., Dushnistky, T., & Krivoy, A. (2009). Carbamate poisoning: Treatment recommendations in the setting of a mass casualties event. *American Journal of Emergency Medicine, 27*(9), 1117–1124. https://doi.org/10.1016/j.ajem.2009.01.035.

86. Boyd, C. A., Weiler, M. H., & Porter, W. P. (1990). Behavioral and neurochemical changes associated with chronic exposure to low-level concentration of pesticide mixtures. *Journal of Toxicology and Environmental Health, 30*(2), 209–221. https://doi.org/10.1080/15287399009531424.
87. Alvarez, F., Arena, M., Auteri, D., Binaglia, M., Castoldi, A. F., Chiusolo, A., & Villamar-Bouza, L. (2022). Peer review of the pesticide risk assessment of the active substance oxamyl. *EFSA Journal, 20*(8), e07296. https://doi.org/10.2903/j.efsa.2022.7296.
88. Guanggang, X., Diqiu, L., Jianzhong, Y., Jingmin, G., Huifeng, Z., Mingan, S., & Liming, T. (2013). Carbamate insecticide methomyl confers cytotoxicity through DNA damage induction. *Food and Chemical Toxicology, 53*, 352–358. https://doi.org/10.1016/j.fct.2012.12.020.
89. Karslı, S., Yazar, S., İncedere Düzdağ, E., & Yurdun, T. (2022). Assessment of genotoxic effects of organophosphate and carbamate pesticides by comet assay. *Istanbul Journal of Pharmacy, 52*(2), 136–142. https://doi.org/10.26650/istanbuljpharm.2022.1057224.
90. Mahgoub, A. A., & El-Medany, A. H. (2001). Evaluation of chronic exposure of the male rat reproductive system to the insecticide methomyl. *Pharmacological Research, 44*(2), 73–80. https://doi.org/10.1006/phrs.2001.0816.
91. Sakr, S., Hassanien, H., Bester, M. J., Arbi, S., Sobhy, A., El Negris, H., & Steenkamp, V. (2018). Beneficial effects of folic acid on the kidneys and testes of adult albino rats after exposure to methomyl. *Toxicology Research, 7*(3), 480. https://doi.org/10.1039/c7tx00309a.
92. Moreira, S., Silva, R., Carrageta, D. F., Alves, M. G., Seco-Rovira, V., Oliveira, P. F., & Pereira, M. L. (2022). Carbamate pesticides: Shedding light on their impact on the male reproductive system. *International Journal of Molecular Sciences, 23*(15), 8206. https://doi.org/10.3390/ijms23158206.
93. Leung, M. C. K., & Meyer, J. N. (2019). Mitochondria as a target of organophosphate and carbamate pesticides: Revisiting common mechanisms of action with new approach methodologies. *Reproductive Toxicology, 89*, 83. https://doi.org/10.1016/j.reprotox.2019.07.007.
94. Manar, R. A., Omali, E. K., Rehab, A. A., & Hussam, A. G. (2021). Green tea attenuates methomyl-induced cardiac toxicity through modulating oxidative stress and nuclear factor kappa B upregulation in mice. *Egyptian Journal of Basic and Applied Sciences, 8*(1), 136–145. https://doi.org/10.1080/2314808x.2021.1914401.
95. Aslanturk, A., & Kalender, Y. (2021). Methomyl-induced nephrotoxicity and protective effect of curcumin in male rats. *Toxicology Research, 10*(4), 1003. https://doi.org/10.1093/toxres/tfab080.
96. Sule, R. O., Condon, L., & Gomes, A. V. (2022). A common feature of pesticides: Oxidative stress—The role of oxidative stress in pesticide-induced toxicity. *Oxidative Medicine and Cellular Longevity, 2022*, 5563759. https://doi.org/10.1155/2022/5563759.
97. Hossain, M., Suchi, T. T., Samiha, F., Islam, M. M. M., Tully, F. A., Hasan, J., & Reza, H. M. (2023). Coenzyme Q10 ameliorates carbofuran-induced hepatotoxicity and nephrotoxicity in Wister rats. *Heliyon, 9*(8), e13727. https://doi.org/10.1016/j.heliyon.2023.e13727.
98. Patil, J., Patil, A., Sontakke, A., & Govindwar, S. (2008). Effect of methomyl on hepatic mixed function oxidases in rats. *Indian Journal of Pharmacology, 40*(4), 158. https://doi.org/10.4103/0253-7613.43162.
99. Bini Dhouib, I., Annabi, A., Jallouli, M., Marzouki, S., Gharbi, N., Elfazaa, S., & Lasram, M. M. (2016). Carbamate pesticides-induced immunotoxicity and carcinogenicity in humans: A review. *Journal of Applied Biomedicine, 14*(2), 85–90. https://doi.org/10.1016/j.jab.2016.01.001.
100. Piel, C., Pouchieu, C., Carles, C., Béziat, B., Boulanger, M., Bureau, M., & Baldi, I. (2019). Agricultural exposures to carbamate herbicides and fungicides and central nervous system tumour incidence in the cohort AGRICAN. *Environment International, 130*, 104876. https://doi.org/10.1016/j.envint.2019.05.070.
101. He, F., Wang, S., Liu, L., Chen, S., Zhang, Z., & Sun, J. (1989). Clinical manifestations and diagnosis of acute pyrethroid poisoning. *Archives of Toxicology, 63*, 54–58. https://doi.org/10.1007/BF00334635.
102. Ramchandra, A. M., Chacko, B., & Victor, P. J. (2019). Pyrethroid poisoning. *Indian Journal of Critical Care Medicine, 23*, S267. https://doi.org/10.5005/JP-JOURNALS-10071-23304.
103. Soderlund, D. M. (2012). Molecular mechanisms of pyrethroid insecticide neurotoxicity: Recent advances. *Archives of Toxicology, 86*, 165. https://doi.org/10.1007/S00204-011-0726-X.
104. Ray, D. E., & Forshaw, P. J. (2000). Pyrethroid insecticides: Poisoning syndromes, synergies, and therapy. *Journal of Toxicology: Clinical Toxicology, 38*, 95–101. https://doi.org/10.1081/CLT-100100922.

105. Wakeling, E. N., Neal, A. P., & Atchison, W. D. (2012). Pyrethroids and their effects on ion channels. *Pesticides–Advances in Chemical and Biological Pesticides*. https://doi.org/10.5772/50330.
106. Bhaskar, E. M., Moorthy, S., Ganeshwala, G., & Abraham, G. (2010). Cardiac conduction disturbance due to prallethrin (pyrethroid) poisoning. *Journal of Medical Toxicology, 6*, 27–30. https://doi.org/10.1007/S13181-010-0032-7.
107. Bradberry, S. M., Cage, S. A., Proudfoot, A. T., & Vale, J. A. (2005). Poisoning due to pyrethroids. *Toxicology Reviews, 24*, 93–106. https://doi.org/10.2165/00139709-200524020-00003.
108. Forshaw, P. J., Lister, T., & Ray, D. E. (2000). The role of voltage-gated chloride channels in type II pyrethroid insecticide poisoning. *Toxicology and Applied Pharmacology, 163*, 1–8. https://doi.org/10.1006/TAAP.1999.8848.
109. Hansen, M. R. H., Jørs, E., Lander, F., Condarco, G., Debes, F., Tirado Bustillos, N., & Schlünssen, V. (2017). Neurological deficits after long-term pyrethroid exposure. *Environmental Health Insights, 11*. https://doi.org/10.1177/1178630217700628.
110. Hansen, M. R. H., Jørs, E., Lander, F., Condarco, G., & Schlünssen, V. (2014). Is cumulated pyrethroid exposure associated with prediabetes? A cross-sectional study. *Journal of Agromedicine, 19*, 417. https://doi.org/10.1080/1059924X.2014.945708.
111. Navarrete-Meneses, M. D. P., & Pérez-Vera, P. (2019). Pyrethroid pesticide exposure and hematological cancer: Epidemiological, biological, and molecular evidence. *Review of Environmental Health, 34*, 197–210. https://doi.org/10.1515/REVEH-2018-0070.
112. Ding, G., Shi, R., Gao, Y., Zhang, Y., Kamijima, M., Sakai, K., Wang, G., Feng, C., & Tian, Y. (2012). Pyrethroid pesticide exposure and risk of childhood acute lymphocytic leukemia in Shanghai. *Environmental Science & Technology, 46*, 13480–13487. https://doi.org/10.1021/ES303362A/ASSET/IMAGES/MEDIUM/ES-2012-03362A_0001.GIF.
113. Klementova, S., Keltnerova, L., Klementova, S., & Keltnerova, L. (2015). Triazine herbicides in the environment. *Herbicides: Physiology, Action, and Safety*. https://doi.org/10.5772/60858.
114. Semren, T. Ž., Žunec, S., & Pizent, A. (2018). Oxidative stress in triazine pesticide toxicity: A review of the main biomarker findings. *Archives of Industrial Hygiene and Toxicology, 69*, 109–125. https://doi.org/10.2478/AIHT-2018-69-3118.
115. Ma, K., Wu, H. Y., Zhang, B., He, X., & Li, B. X. (2015). Neurotoxicity effects of atrazine-induced SH-SY5Y human dopaminergic neuroblastoma cells via microglial activation. *Molecular Biosystems, 11*, 2915–2924. https://doi.org/10.1039/C5MB00432B.
116. Hanioka, N., Jinno, H., Tanaka-Kagawa, T., Nishimura, T., & Ando, M. (1998). Changes in rat liver cytochrome P450 enzymes by atrazine and simazine treatment. *Xenobiotica, 28*, 683–698. https://doi.org/10.1080/004982598239263.
117. Zimmerman, A. D., Breckenridge, C. B., Yi, K. D., Sawhney Coder, P., Wanders, D., Judd, R. L., & Foradori, C. D. (2018). Changes in hepatic phase I and phase II biotransformation enzyme expression and glutathione levels following atrazine exposure in female rats. *Xenobiotica, 48*, 867–881. https://doi.org/10.1080/00498254.2017.1374486.
118. Abass, K., Turpeinen, M., & Pelkonen, O. (2009). An evaluation of the cytochrome P450 inhibition potential of selected pesticides in human hepatic microsomes. *Journal of Environmental Science and Health, Part B, 44*, 553–563. https://doi.org/10.1080/03601230902997766.
119. Abass, K., Lämsä, V., Reponen, P., Küblbeck, J., Honkakoski, P., Mattila, S., Pelkonen, O., & Hakkola, J. (2012). Characterization of human cytochrome P450 induction by pesticides. *Toxicology, 294*, 17–26. https://doi.org/10.1016/J.TOX.2012.01.010.
120. Abass, K., Pelkonen, O., & Rautio, A. (2021). Chloro-s-triazines—Toxicokinetic, toxicodynamic, human exposure, and regulatory considerations. *Current Drug Metabolism, 22*, 645. https://doi.org/10.2174/1389200222666210701164945.
121. Simpkins, J. W., Swenberg, J. A., Weiss, N., Brusick, D., Eldridge, J. C., Stevens, J. T., Handa, R. J., Hovey, R. C., Plant, T. M., Pastoor, T. P., & Breckenridge, C. B. (2011). Atrazine and breast cancer: A framework assessment of the toxicological and epidemiological evidence. *Toxicological Sciences, 123*(1), 441. https://doi.org/10.1093/toxsci/kfr176.

122. Breckenridge, C. B., Eldridge, J. C., Stevens, J. T., & Simpkins, J. W. (2010). Symmetrical triazine herbicides: A review of regulatory toxicity endpoints. In *Hayes' Handbook of Pesticide Toxicology* (3rd ed., Vol. 1, pp. 1711–1723). https://doi.org/10.1016/B978-0-12-374367-1.00079-3.
123. Sathiakumar, N., MacLennan, P. A., Mandel, J., & Delzell, E. (2011). A review of epidemiologic studies of triazine herbicides and cancer. *Critical Reviews in Toxicology, 41*(Suppl 1), 1–34. https://doi.org/10.3109/10408444.2011.554793.
124. Boffetta, P., Adami, H. O., Berry, S. C., & Mandel, J. S. (2013). Atrazine and cancer: A review of the epidemiologic evidence. *European Journal of Cancer Prevention, 22*(2), 169–180. https://doi.org/10.1097/CEJ.0b013e32835849ca.
125. Fishel, F. (2005). Pesticide toxicity profile: Triazole herbicides. https://doi.org/10.32473/edis-pi126-2005.
126. Jalal, M., & Ez-Zaher, L. (2023). Exploring health risk issues related to the use of triazoles among farmers in the Souss Massa region, Morocco. *International Journal of Environmental Studies*. https://doi.org/10.1080/00207233.2023.2229202.
127. European Food Safety Authority (EFSA). (2009). Scientific opinion on risk assessment for a selected group of pesticides from the triazole group to test possible methodologies to assess cumulative effects from exposure through food from these pesticides on human health. *EFSA Journal, 7*, 1167. https://doi.org/10.2903/J.EFSA.2009.1167.
128. Robinson, J. F., Tonk, E. C. M., Verhoef, A., Piersma, A. H., & Piersma, H. (2012). Triazole induced concentration-related gene signatures in rat whole embryo culture. *Reproductive Toxicology, 34*(3), 275–283. https://doi.org/10.1016/j.reprotox.2012.05.088.
129. Filipov, N. M., Lawrence, D. A., Moser, V. C., Barone, S., Smialowicz, R. J., Harris, M. W., Davis, B. J., Overstreet, D., Mauney, M., & Chapin, R. E. (2001). Developmental toxicity of a triazole fungicide: Consideration of interorgan communication. *Toxicological Sciences, 62*(2), 185–186. https://doi.org/10.1093/toxsci/62.2.185.
130. Jaklová Dytrtová, J., Bělonožníková, K., Jakl, M., & Ryšlavá, H. (2020). Triazoles and aromatase: The impact of copper cocktails. *Environmental Pollution, 266*, 115201. https://doi.org/10.1016/j.envpol.2020.115201.
131. Chen, X., Zhu, Q., Li, X., Huang, T., Wang, S., Wang, Y., Chen, X., Lin, Z., & Ge, R. S. (2019). Pubertal exposure to tebuconazole increases testosterone production via inhibiting testicular aromatase activity in rats. *Chemosphere, 230*, 519–526. https://doi.org/10.1016/j.chemosphere.2019.05.122.
132. Goetz, A. K., Rockett, J. C., Ren, H., Thillainadarajah, I., & Dix, D. J. (2009). Inhibition of rat and human steroidogenesis by triazole antifungals. *Systems Biology in Reproductive Medicine, 55*(4), 214–226. https://doi.org/10.3109/19396360903234045.
133. Nunes, A., Jardim, O., Mello, D. C., Brito, A. P., Van Der Voet, H., Boon, P. E., & Caldas, E. D. (2018). Probabilistic dietary risk assessment of triazole and dithiocarbamate fungicides for the Brazilian population. *Food and Chemical Toxicology, 118*, 317–327. https://doi.org/10.1016/j.fct.2018.05.002.
134. Ku, T., Zhou, M., Hou, Y., Xie, Y., Li, G., & Sang, N. (2021). Tebuconazole induces liver injury coupled with ROS-mediated hepatic metabolism disorder. *Ecotoxicology and Environmental Safety, 220*, 112309. https://doi.org/10.1016/j.ecoenv.2021.112309.
135. Nesnow, S., Ward, W., Moore, T., Ren, H., & Hester, S. D. (2009). Discrimination of tumorigenic triazole conazoles from phenobarbital by transcriptional analyses of mouse liver gene expression. *Toxicological Sciences, 110*(1), 68–83. https://doi.org/10.1093/toxsci/kfp076.
136. Das, P. C., Cao, Y., Cherrington, N., Hodgson, E., & Rose, R. L. (2006). Fipronil induces CYP isoforms and cytotoxicity in human hepatocytes. *Chemico-Biological Interactions, 164*(3), 200–214. https://doi.org/10.1016/j.cbi.2006.09.013.
137. Zhao, X., Yeh, J. Z., Salgado, V. L., & Narahashi, T. (2005). Sulfone metabolite of fipronil blocks gamma-aminobutyric acid- and glutamate-activated chloride channels in mammalian and insect neurons. *Journal of Pharmacology and Experimental Therapeutics, 314*(1), 363–373. https://doi.org/10.1124/jpet.104.077891.
138. Mohamed, F., Senarathna, L., Percy, A., Abeyewardene, M., Eaglesham, G., Cheng, R., Azher, S., Hittarage, A., Dissanayake, W., Sheriff, M. H. R., Davies, W., Buckley, N. A., & Eddleston, M. (2004). Acute human self-poisoning with the N-phenylpyrazole insecticide fipronil—A GABAA-gated chloride

channel blocker. *Journal of Toxicology and Clinical Toxicology, 42*(9), 955. https://doi.org/10.1081/clt-200041784.

139. Bharathraj, M. Y., Venugopal, K., Jaligidad, K., Karibasappa, H., & Kumar, H. (2015). Fipronil compound consumption presenting as status epilepticus. *Toxicology International, 22*(2), 165. https://doi.org/10.4103/0971-6580.172280.
140. Ratra, G. S., & Casida, J. E. (2001). GABA receptor subunit composition relative to insecticide potency and selectivity. *Toxicology Letters, 122*(3), 215–222. https://doi.org/10.1016/S0378-4274(01)00366-6.
141. Hainzl, D., Cole, L. M., & Casida, J. E. (1998). Mechanisms for selective toxicity of fipronil insecticide and its sulfone metabolite and desulfinyl photoproduct. *Chemistry Research in Toxicology, 11*(12), 1529–1535. https://doi.org/10.1021/TX980157T.
142. Ratra, G. S., Erkkila, B. E., Weiss, D. S., & Casida, J. E. (2002). Unique insecticide specificity of human homomeric ρ1 GABAC receptor. *Toxicology Letters, 129*(1, 2), 47–53. https://doi.org/10.1016/S0378-4274(01)00471-4.
143. Islam, R., & Lynch, J. W. (2012). Mechanism of action of the insecticides, lindane and fipronil, on glycine receptor chloride channels. *British Journal of Pharmacology, 165*(12), 2707. https://doi.org/10.1111/J.1476-5381.2011.01722.X.
144. Vidau, C., González-Polo, R. A., Niso-Santano, M., Gómez-Sánchez, R., Bravo-San Pedro, J. M., Pizarro-Estrella, E., Blasco, R., Brunet, J. L., Belzunces, L. P., & Fuentes, J. M. (2011). Fipronil is a powerful uncoupler of oxidative phosphorylation that triggers apoptosis in human neuronal cell line SHSY5Y. *Neurotoxicology, 32*(6), 935–943. https://doi.org/10.1016/J.NEURO.2011.04.006.
145. Terçariol, P. R. G., & Godinho, A. F. (2011). Behavioral effects of acute exposure to the insecticide fipronil. *Pesticide Biochemistry and Physiology, 99*(3), 221–225. https://doi.org/10.1016/J.PESTBP.2010.12.007.
146. Mossa, A. T. H., Swelam, E. S., Mohafrash, S. M. M. (2015). Sub-chronic exposure to fipronil induced oxidative stress, biochemical and histopathological changes in the liver and kidney of male albino rats. *Toxicology Reports, 2*, 775–784. https://doi.org/10.1016/J.TOXREP.2015.02.009.
147. de Oliveira, P. R., Bechara, G. H., Denardi, S. E., Oliveira, R. J., Mathias, M. I. C. (2012). Cytotoxicity of fipronil on mice liver cells. *Microscopy Research and Technique, 75*(1), 28–35. https://doi.org/10.1002/JEMT.21018.
148. Badgujar, P. C., Selkar, N. A., Chandratre, G. A., Pawar, N. N., Dighe, V. D., Bhagat, S. T., Telang, A. G., & Vanage, G. R. (2017). Fipronil-induced genotoxicity and DNA damage *in vivo*: Protective effect of vitamin E. *Human Experimental Toxicology, 36*(5), 508–519. https://doi.org/10.1177/0960327116655388.
149. Çelik, A., Ekinci, S. Y., Güler, G., & Yildirim, S. (2014). *In vitro* genotoxicity of fipronil: Sister chromatid exchange, cytokinesis block micronucleus test, and comet assay. *DNA and Cell Biology, 33*(2), 148–154. https://doi.org/10.1089/DNA.2013.2158.
150. Khan, S., Jan, M. H., Kumar, D., & Telang, A. G. (2015). Fipronil induced spermotoxicity is associated with oxidative stress, DNA damage and apoptosis in male rats. *Pesticide Biochemistry and Physiology, 124*, 8–14. https://doi.org/10.1016/J.PESTBP.2015.03.010.
151. Pandit, A. A., Gandham, R. K., Mukhopadhyay, C. S., Verma, R., & Sethi, R. S. (2019). Transcriptome analysis reveals the role of the PCP pathway in fipronil and endotoxin-induced lung damage. *Respiratory Research, 20*, 1–16. https://doi.org/10.1186/S12931-019-0986-1.
152. Costas-Ferreira, C., Faro, L. R. F. (2021). Neurotoxic effects of neonicotinoids on mammals: What is there beyond the activation of nicotinic acetylcholine receptors?—A systematic review. *International Journal of Molecular Sciences, 22*(16). https://doi.org/10.3390/IJMS22168413.
153. Li, P., Ann, J., & Akk, G. (2011). Activation and modulation of human α4β2 nicotinic acetylcholine receptors by the neonicotinoids clothianidin and imidacloprid. *Journal of Neuroscience Research, 89*(11), 1295. https://doi.org/10.1002/JNR.22644.
154. Lin, P. C., Lin, H. J., Liao, Y. Y., Guo, H. R., & Chen, K. T. (2013). Acute poisoning with neonicotinoid insecticides: A case report and literature review. *Basic & Clinical Pharmacology & Toxicology, 112*(5), 282–286. https://doi.org/10.1111/BCPT.12027.
155. Selvam, V., & Srinivasan, S. (2019). Neonicotinoid poisoning and management. *Indian Journal of Critical Care Medicine, 23*(Supplement), S260. https://doi.org/10.5005/JP-JOURNALS-10071-23308.

156. Anadón, A., Ares, I., Martínez, M., Martínez-Larrañaga, M. R., & Martínez, M. A. (2020). Neurotoxicity of neonicotinoids. In *Advances in Neurotoxicology* (pp. 167–207). Academic Press. https://doi.org/10.1016/BS.ANT.2019.11.005.
157. Abou-Donia, M. B., Goldstein, L. B., Bullman, S., Tu, T., Khan, W. A., Dechkovskaia, A. M., & Abdel-Rahman, A. A. (2008). Imidacloprid induces neurobehavioral deficits and increases expression of glial fibrillary acidic protein in the motor cortex and hippocampus in offspring rats following *in utero* exposure. *Journal of Toxicology and Environmental Health, Part A, 71*(2), 119–130. https://doi.org/10.1080/15287390701613140.
158. Abd-Elhakim, Y. M., Mohammed, H. H., Mohamed, W. A. M. (2018). Imidacloprid impacts on neurobehavioral performance, oxidative stress, and apoptotic events in the brain of adolescent and adult rats. *Journal of Agricultural and Food Chemistry, 66*(51), 13513–13524. https://doi.org/10.1021/ACS.JAFC.8B05793.
159. Nakayama, A., Yoshida, M., Kagawa, N., & Nagao, T. (2019). The neonicotinoids acetamiprid and imidacloprid impair neurogenesis and alter the microglial profile in the hippocampal dentate gyrus of mouse neonates. *Journal of Applied Toxicology, 39*(7), 877–887. https://doi.org/10.1002/JAT.3776.
160. Şenyildiz, M., Kilinc, A., & Ozden, S. (2018). Investigation of the genotoxic and cytotoxic effects of widely used neonicotinoid insecticides in HepG2 and SH-SY5Y cells. *Toxicology Industrial Health, 34*(4), 375–383. https://doi.org/10.1177/0748233718762609.
161. Duzguner, V., & Erdogan, S. (2010). Acute oxidant and inflammatory effects of imidacloprid on the mammalian central nervous system and liver in rats. *Pesticide Biochemistry and Physiology, 97*(1), 13–18. https://doi.org/10.1016/J.PESTBP.2009.11.008.
162. Duzguner, V., & Erdogan, S. (2012). Chronic exposure to imidacloprid induces inflammation and oxidative stress in the liver & central nervous system of rats. *Pesticide Biochemistry and Physiology, 104*(1), 58–64. https://doi.org/10.1016/J.PESTBP.2012.06.011.
163. Bhardwaj, S., Srivastava, M. K., Kapoor, U., & Srivastava, L. P. (2010). A 90 days oral toxicity of imidacloprid in female rats: Morphological, biochemical, and histopathological evaluations. *Food and Chemical Toxicology, 48*(4), 1185–1190. https://doi.org/10.1016/J.FCT.2010.02.009.
164. Wang, X., Anadón, A., Wu, Q., Qiao, F., Ares, I., Martínez-Larrañaga, M. R., Yuan, Z., & Martínez, M. A. (2018). Mechanism of neonicotinoid toxicity: Impact on oxidative stress and metabolism. *Annual Review of Pharmacology and Toxicology, 58*, 471–507. https://doi.org/10.1146/ANNUREV-PHARMTOX-010617-052429.
165. Shakthi Devan, R. K., Prabu, P. C., & Panchapakesan, S. (2015). Immunotoxicity assessment of sub-chronic oral administration of acetamiprid in Wistar rats. *Drug and Chemical Toxicology, 38*(3), 328–336. https://doi.org/10.3109/01480545.2014.966382.
166. Bal, R., Naziroğlu, M., Türk, G., Yilmaz, Ö., Kuloğlu, T., Etem, E., & Baydas, G. (2012). Insecticide imidacloprid induces morphological and DNA damage through oxidative toxicity on the reproductive organs of developing male rats. *Cell Biochemistry and Function, 30*(6), 492–499. https://doi.org/10.1002/CBF.2826.
167. Bal, R., Türk, G., Tuzcu, M., Yilmaz, O., Kuloglu, T., Gundogdu, R., Gür, S., Agca, A., Ulas, M., Çambay, Z., Tuzcu, Z., Gencoglu, H., Guvenc, M., Ozsahin, A. D., Kocaman, N., Aslan, A., & Etem, E. (2012). Assessment of imidacloprid toxicity on reproductive organ system of adult male rats. *Journal of Environmental Science and Health, Part B, 47*(5), 434–444. https://doi.org/10.1080/03601234.2012.663311.
168. Genuis, S. J., Lane, K., & Birkholz, D. (2016). Human elimination of organochlorine pesticides: Blood, urine, and sweat study. *Biomedical Research International, 2016*, 1624643. https://doi.org/10.1155/2016/1624643.
169. Genuis, S. J. (2010). Sensitivity-related illness: The escalating pandemic of allergy, food intolerance, and chemical sensitivity. *Science of the Total Environment, 408*(23), 6047–6061. https://doi.org/10.1016/J.SCITOTENV.2010.08.047.
170. Porpora, M. G., Resta, S., & Fuggetta, E. (2016). Organochlorine pesticides exposure and preterm birth. *Indian Journal of Medical Research, 143*(6), 685. https://doi.org/10.4103/0971-5916.191922.

171. Mnif, W., Hassine, A. I. H., Bouaziz, A., Bartegi, A., Thomas, O., & Roig, B. (2011). Effect of endocrine disruptor pesticides: A review. *International Journal of Environmental Research and Public Health, 8*(6), 2265. https://doi.org/10.3390/IJERPH8062265.
172. Benarbia, M. A., Macherel, D., Faure, S., Jacques, C., Andriantsitohaina, R., & Malthièry, Y. (2013). Plasmatic concentration of organochlorine lindane acts as metabolic disruptors in HepG2 liver cell line by inducing mitochondrial disorder. *Toxicology and Applied Pharmacology, 272*(3), 325–334. https://doi.org/10.1016/J.TAAP.2013.06.006.
173. Hou, L., Zhang, X., Wang, D., & Baccarelli, A. (2012). Environmental chemical exposures and human epigenetics. *International Journal of Epidemiology, 41*(1), 79–105. https://doi.org/10.1093/IJE/DYR154.
174. Karami-Mohajeri, S., & Abdollahi, M. (2011). Toxic influence of organophosphate, carbamate, and organochlorine pesticides on cellular metabolism of lipids, proteins, and carbohydrates: A systematic review. *Human and Experimental Toxicology, 30*(10), 1119–1140. https://doi.org/10.1177/0960327110388959.
175. Seo, S. H., Choi, S. D., Batterman, S., & Chang, Y. S. (2022). Health risk assessment of exposure to organochlorine pesticides in the general population in Seoul, Korea over 12 years: A cross-sectional epidemiological study. *Journal of Hazardous Materials, 424*, 127381. https://doi.org/10.1016/J.JHAZMAT.2021.127381.
176. Saeedi Saravi, S. S., & Dehpour, A. R. (2016). Potential role of organochlorine pesticides in the pathogenesis of neurodevelopmental, neurodegenerative, and neurobehavioral disorders: A review. *Life Sciences, 145*, 255–264. https://doi.org/10.1016/J.LFS.2015.11.006.
177. Caudle, W. M. (2015). Occupational exposures and parkinsonism. In *Handbook of Clinical Neurology* (Vol. 129, pp. 225–239). Elsevier. https://doi.org/10.1016/B978-0-444-62627-1.00013-5.
178. Jurewicz, J., & Hanke, W. (2008). Prenatal and childhood exposure to pesticides and neurobehavioral development: Review of epidemiological studies. *International Journal of Occupational Medicine and Environmental Health, 21*(2), 121–132. https://doi.org/10.2478/V10001-008-0014-Z.
179. Kaur, N., Swain, S., Banerjee, B., Sharma, T., & Krishnalata, T. (2019). Organochlorine pesticide exposure as a risk factor for breast cancer in young Indian women: A case–control study. *South Asian Journal of Cancer, 8*(4), 212. https://doi.org/10.4103/SAJC.SAJC_427_18.
180. McGlynn, K. A., Quraishi, S. M., Graubard, B. I., Weber, J. P., Rubertone, M. V., & Erickson, R. L. (2008). Persistent organochlorine pesticides and risk of testicular germ cell tumors. *Journal of the National Cancer Institute, 100*(9), 663–671. https://doi.org/10.1093/JNCI/DJN101.
181. Luo, D., Zhou, T., Tao, Y., Feng, Y., Shen, X., & Mei, S. (2016). Exposure to organochlorine pesticides and non-Hodgkin lymphoma: A meta-analysis of observational studies. *Scientific Reports*, *6*, 25768. https://doi.org/10.1038/srep25768.
182. Upson, K., De Roos, A. J., Thompson, M. L., Sathyanarayana, S., Scholes, D., Barr, D. B., & Holt, V. L. (2013). Organochlorine pesticides and risk of endometriosis: Findings from a population-based case-control study. *Environmental Health Perspectives*, *121*, 1319–1324. https://doi.org/10.1289/EHP.1306648.
183. Russo, M., Humes, S. T., Figueroa, A. M., Tagmount, A., Zhang, P., Loguinov, A., Lednicky, J. A., Sabo-Attwood, T., Vulpe, C. D., & Liu, B. (2021). Organochlorine pesticide dieldrin suppresses cellular interferon-related antiviral gene expression. *Toxicological Sciences*, *182*, 260–274. https://doi.org/10.1093/TOXSCI/KFAB064.
184. Sinaii, N., Cleary, S. D., Ballweg, M. L., Nieman, L. K., & Stratton, P. (2002). High rates of autoimmune and endocrine disorders, fibromyalgia, chronic fatigue syndrome, and atopic diseases among women with endometriosis: A survey analysis. *Human Reproduction*, *17*, 2715–2724. https://doi.org/10.1093/HUMREP/17.10.2715.
185. Sebastian, R., & Raghavan, S. C. (2015). Endosulfan induces male infertility. *Cell Death & Disease*, *6*, e368. https://doi.org/10.1038/cddis.2015.368.
186. Yang, J. H., Lee, Y. M., Bae, S. G., Jacobs, D. R., & Lee, D. H. (2012). Associations between organochlorine pesticides and vitamin D deficiency in the U.S. population. *PLoS One*, *7*. https://doi.org/10.1371/JOURNAL.PONE.0030093.

187. Brennan, E., Butler, A. E., Nandakumar, M., Drage, D. S., Sathyapalan, T., & Atkin, S. L. (2023). Association between organochlorine pesticides and vitamin D in female subjects. *Biomedicines*, *11*. https://doi.org/10.3390/BIOMEDICINES11051451.
188. Freire, C., Lopez-Espinosa, M. J., Fernández, M., Molina-Molina, J. M., Prada, R., & Olea, N. (2011). Prenatal exposure to organochlorine pesticides and TSH status in newborns from Southern Spain. *Science of the Total Environment*, *409*, 3281–3287. https://doi.org/10.1016/J.SCITOTENV.2011.05.037.
189. Su, Y., Dai, Y., Lin, Y., Gao, X., Han, Y., & Zhao, B. (2012). Serum organochlorine pesticide residues and risk of gallstone disease: A case-control study in Xiamen. *Annals of Epidemiology*, *22*, 592–597. https://doi.org/10.1016/J.ANNEPIDEM.2012.05.002.
190. Hussain, M., Aftab, K., Iqbal, M., Ali, S., Rizwan, M., Alkahtani, S., & Abdel-Daim, M. M. (2020). Determination of pesticide residue in brinjal sample using HPTLC and developing a cost-effective method alternative to HPLC. *Journal of Chemistry*, *2020*, 8180320. https://doi.org/10.1155/2020/8180320.
191. Udomkun, P., Boonupara, T., Sumitsawan, S., Khan, E., Pongpichan, S., & Kajitvichyanukul, P. (2023). Airborne pesticides—Deep diving into sampling and analysis. *Toxics*, *11*, 883. https://doi.org/10.3390/toxics11110883.
192. Brinco, J., Guedes, P., Gomes da Silva, M., Mateus, E. P., & Ribeiro, A. B. (2023). Analysis of pesticide residues in soil: A review and comparison of methodologies. *Microchemical Journal*, *195*, 109465. https://doi.org/10.1016/j.microc.2023.109465.
193. Melo, L. F. C., Collins, C. H., & Jardim, I. C. S. F. (2005). High-performance liquid chromatographic determination of pesticides in tomatoes using laboratory-made NH2 and C18 solid-phase extraction materials. *Journal of Chromatography A*, *1073*, 75–81. https://doi.org/10.1016/j.chroma.2004.09.043.
194. Hernández-Mesa, M., & Moreno-González, D. (2022). Current role of mass spectrometry in the determination of pesticide residues in food. *Separations*, *9*, 148. https://doi.org/10.3390/separations9060148.
195. Gould, O., Nguyen, N., & Honeychurch, K. C. (2023). New applications of gas chromatography and gas chromatography-mass spectrometry for novel sample matrices in the forensic sciences: A literature review. *Chemosensors*, *11*, 527. https://doi.org/10.3390/chemosensors11100527.
196. Campanale, C., Massarelli, C., Losacco, D., Bisaccia, D., Triozzi, M., & Uricchio, V. F. (2021). The monitoring of pesticides in water matrices and the analytical criticalities: A review. *TrAC – Trends in Analytical Chemistry*, *144*, 116423. https://doi.org/10.1016/j.trac.2021.116423.
197. Bezerra, K. S. (2016). We are IntechOpen, the world's leading publisher of open-access books built by scientists, for scientists. *Intech*, *11*, 1–14.
198. Pitt, J. J., & Pitt, J. J. (2009). Clinical Biochemistry Review. *Clinical Biochemistry Review*, *30*, 19–34.
199. Naz, S., Gallart-Ayala, H., Reinke, S. N., Mathon, C., Blankley, R., Chaleckis, R., & Wheelock, C. E. (2017). Development of a liquid chromatography-high resolution mass spectrometry metabolomics method with high specificity for metabolite identification using all ion fragmentation acquisition. *Analytical Chemistry*, *89*, 7933–7942. https://doi.org/10.1021/acs.analchem.7b00925.
200. Johnson, C. L., Jazana, E., Kong, S. W., & Pennell, K. D. (2021). Environmental Science and Pollution Research, International Journal of Pediatrics, *28*, 3266–3279. https://doi.org/10.1007/s11356-020-10702-6.
201. Wahab, S., Muzammil, K., Nasir, N., Khan, M. S., Ahmad, F., Khalid, M., Ahmad, W., Dawria, A., Kalyan, L., Reddy, V., & Busayli, A. M. (2022). Advancement and new trends in analysis of pesticide residues in food: A comprehensive review. *Plants, 11*(9), 1–30. https://doi.org/10.3390/plants11091106.
202. Yuan, X., Kim, C. J., Lee, R., Kim, M., Shin, H. J., Kim, L., Jeong, W. T., Shin, Y., Kyung, K. S., & Noh, H. H. (2022). Validation of a multi-residue analysis method for 287 pesticides in citrus fruits mandarin orange and grapefruit using liquid chromatography–tandem mass spectrometry. *Foods, 11*(21), 1–12. https://doi.org/10.3390/foods11213522.
203. Xu, L., Abd El-Aty, A. M., Eun, J. B., Shim, J. H., Zhao, J., Lei, X., Gao, S., She, Y., Jin, F., Wang, J., Jin, M., & Hammock, B. D. (2022). Recent advances in rapid detection techniques for pesticide residue: A review. *Journal of Agricultural and Food Chemistry, 70*(39), 13093–13117. https://doi.org/10.1021/acs.jafc.2c05284.

204. Khan, N. S., Pradhan, D., Choudhary, S., Saxena, P., Poddar, N. K., & Jain, A. K. (2021). Immunoassay-based approaches for development of screening of chlorpyrifos. *Journal of Analytical Science and Technology, 12*, 1–16. https://doi.org/10.1186/s40543-021-00282-6.
205. Watanabe, E., Miyake, S., & Yogo, Y. (2013). Review of enzyme-linked immunosorbent assays (ELISAs) for analyses of neonicotinoid insecticides in agro-environments. *Journal of Agricultural and Food Chemistry, 61*(50), 12459–12472. https://doi.org/10.1021/jf403801h.
206. Sakamoto, S., Putalun, W., Vimolmangkang, S., Phoolcharoen, W., Shoyama, Y., Tanaka, H., & Morimoto, S. (2018). Enzyme-linked immunosorbent assay for the quantitative/qualitative analysis of plant secondary metabolites. *Journal of Natural Medicines, 72*(1), 32–42. https://doi.org/10.1007/s11418-017-1144-z.
207. Huang, L., Zhang, F., Li, F., Jia, Y., Wang, M., Hua, X., & Wang, L. (2022). Development of Ic-ELISA and colloidal gold lateral flow immunoassay for the determination of cypermethrin in agricultural samples. *Biosensors, 12*(11), 1–12. https://doi.org/10.3390/bios12111058.
208. Ding, X., Srinivasan, B., & Tung, S. (2015). Development and applications of portable biosensors. *Journal of Laboratory Automation, 20*(4), 365–389. https://doi.org/10.1177/2211068215581349.
209. Teniou, A., Rhouati, A., & Marty, J. L. (2021). Mathematical modelling of biosensing platforms applied for environmental monitoring. *Chemosensors, 9*(3), 1–36. https://doi.org/10.3390/chemosensors9030050.
210. Wu, H., Zhao, J., & Wan, J. (2023). A review of veterinary drug residue detection: Recent advancements, challenges, and future directions. *Sustainability, 15*(13), 1–12. https://doi.org/10.3390/su151310413.
211. Phopin, K., & Tantimongcolwat, T. (2020). Pesticide aptasensors—State of the art and perspectives. *Sensors, 20*(1), 1–40.
212. Zhang, C., Qiu, M., Wang, J., & Liu, Y. (2023). Recent advances in nanoparticle-based optical sensors for detection of pesticide residues in soil. *Biosensors, 13*(4), 415. https://doi.org/10.3390/bios13040415.
213. Zhou, J., Liu, Y., Du, X., Gui, Y., He, J., Xie, F., & Cai, J. (2023). Recent advances in design and application of nanomaterials-based colorimetric biosensors for agri-food safety analysis. *ACS Omega, 8*(1), 1–16. https://doi.org/10.1021/acsomega.3c06409.
214. Mirres, A. C. de M., da Silva, B. E. P. de M., Tessaro, L., Galvan, D., de Andrade, J. C., Aquino, A., Joshi, N., & Conte-Junior, C. A. (2022). Recent advances in nanomaterial-based biosensors for pesticide detection in foods. *Biosensors, 12*(8), 1–33. https://doi.org/10.3390/bios12080572.
215. Schellenberg, T., Subramanian, V., Ganeshan, G., Tompkins, D., & Pradeep, R. (2020). Wastewater discharge standards in the evolving context of urban sustainability–The case of India. *Frontiers in Environmental Science, 8*, 30. https://doi.org/10.3389/fenvs.2020.00030.
216. Yang, X., Zhang, M., Lu, T., Chen, S., Sun, X., Guan, Y., Zhang, T., Hang, B., Wang, X., Chen, M., Chen, Y., & Xia, Y. (2020). Metabolomics study and meta-analysis on the association between maternal pesticide exposome and birth outcomes. *Environmental Research, 182*, 1–10. https://doi.org/10.1016/j.envres.2019.109087.
217. Bedia, C. (2022). Metabolomics in environmental toxicology: Applications and challenges. *Trends in Environmental Analytical Chemistry, 34*, 1–13. https://doi.org/10.1016/j.teac.2022.e00161.
218. Nolasco, D. M., Mendes, M. P. R., Marciano, L. P. de A., Costa, L. F., De Macedo, A. N., Sakakibara, I. M., Silvério, A. C. P., Paiva, M. J. N., & André, L. C. (2023). An exploratory study of the metabolite profiling from pesticides exposed workers. *Metabolites, 13*(5), 1–25. https://doi.org/10.3390/metabo13050596.
219. Shahid, M., Singh, U. B., & Khan, M. S. (2023). Metabolomics-based mechanistic insights into revealing the adverse effects of pesticides on plants: An interactive review. *Metabolites, 13*(2), 1–25. https://doi.org/10.3390/metabo13020246.
220. Maruya, K. A., Lao, W., Tsukada, D., & Diehl, D. W. (2015). A passive sampler based on solid phase microextraction (SPME) for sediment-associated organic pollutants: Comparing freely-dissolved concentration with bioaccumulation. *Chemosphere, 137*, 192–197. https://doi.org/10.1016/j.chemosphere.2015.07.042.
221. Maruya, K. A., Zeng, E. Y., Tsukada, D., & Bay, S. M. (2009). A passive sampler based on solid-phase microextraction for quantifying hydrophobic organic contaminants in sediment pore water. *Environmental Toxicology and Chemistry, 28*(4), 733–740. https://doi.org/10.1897/08-322R.1.

222. Adjovu, G. E., Stephen, H., James, D., & Ahmad, S. (2023). Overview of the application of remote sensing in effective monitoring of water quality parameters. *Remote Sensing, 15*(7), 1–35. https://doi.org/10.3390/rs15071938.
223. Mashala, M. J., Dube, T., Mudereri, B. T., Ayisi, K. K., & Ramudzuli, M. R. (2023). A systematic review on advancements in remote sensing for assessing and monitoring land use and land cover changes impacts on surface water resources in semi-arid tropical environments. *Remote Sensing, 15*(16), 1–29. https://doi.org/10.3390/rs15163926.
224. Nguyen, H. A. T., Sophea, T., Gheewala, S. H., Rattanakom, R., Areerob, T., & Prueksakorn, K. (2021). Integrating remote sensing and machine learning into environmental monitoring and assessment of land use change. *Sustainable Production and Consumption, 27*, 1239–1254. https://doi.org/10.1016/j.spc.2021.02.025.
225. Abdelhameed, R. M., El-Zawahry, M., & Emam, H. E. (2018). Efficient removal of organophosphorus pesticides from wastewater using polyethylenimine-modified fabrics. *Polymer (Guildf), 155*, 225–234. https://doi.org/10.1016/j.polymer.2018.09.030.
226. Irfey, A. M. M., Alotaibi, B. A., Najim, M. M. M., & Shah, A. A. (2023). Water valuation in urban settings for sustainable water management. *Water (Switzerland), 15*, 1–25. https://doi.org/10.3390/w15173105.
227. Ruomeng, B., Meihao, O., Siru, Z., Shichen, G., Yixian, Z., Junhong, C., Ruijie, M., Yuan, L., Gezhi, X., Xingyu, C., Shiyi, Z., Aihui, Z., & Baishan, F. (2023). Degradation strategies of pesticide residue: From chemicals to synthetic biology. *Synthetic and Systems Biotechnology, 8*(3), 302–313. https://doi.org/10.1016/j.synbio.2023.03.005.
228. Perpetuini, G., Nzobouh Fossi, P. A., Kwak, S., Namessi, O., Petchkongkaew, A., Tofalo, R., & Waché, Y. (2023). Pesticides in foods: Towards bioremediation biocatalysts? *Catalysts, 13*(7), 1–20. https://doi.org/10.3390/catal13071055.
229. Vaksmaa, A., Guerrero-Cruz, S., Ghosh, P., Zeghal, E., Hernando-Morales, V., & Niemann, H. (2023). Role of fungi in bioremediation of emerging pollutants. *Frontiers in Marine Science, 10*, 1–21. https://doi.org/10.3389/fmars.2023.1070905.
230. Tarekegn, M. M., Salilih, F. Z., & Ishetu, A. I. (2020). Microbes used as a tool for bioremediation of heavy metal from the environment. *Cogent Food and Agriculture, 6*, 1–19. https://doi.org/10.1080/23311932.2020.1783174.
231. Giri, B. S., Geed, S., Vikrant, K., Lee, S. S., Kim, K. H., Kailasa, S. K., Vithanage, M., Chaturvedi, P., Rai, B. N., & Singh, R. S. (2021). Progress in bioremediation of pesticide residues in the environment. *Environmental Engineering Research, 26*, 0–1. https://doi.org/10.4491/eer.2020.446.
232. Huang, Y., Xiao, L., Li, F., Xiao, M., Lin, D., Long, X., & Wu, Z. (2018). Microbial degradation of pesticide residues and an emphasis on the degradation of cypermethrin and 3-phenoxy benzoic acid: A review. *Molecules, 23*(9), 1–23. https://doi.org/10.3390/molecules23092313.
233. Bala, S., Garg, D., Thirumalesh, B. V., Sharma, M., Sridhar, K., Inbaraj, B. S., & Tripathi, M. (2022). Recent strategies for bioremediation of emerging pollutants: A review for a green and sustainable environment. *Toxics, 10*(8), 1–24. https://doi.org/10.3390/toxics10080484.
234. Guerrero Ramírez, J. R., Ibarra Muñoz, L. A., Balagurusamy, N., Frías Ramírez, J. E., Alfaro Hernández, L., & Carrillo Campos, J. (2023). Microbiology and biochemistry of pesticides biodegradation. *International Journal of Molecular Sciences, 24*, 1–47. https://doi.org/10.3390/ijms242115969.
235. Vaiano, V., Iervolino, G., Rizzo, L., & Sannino, D. (2017). Advanced oxidation processes for the removal of food dyes in wastewater. *Current Organic Chemistry, 21*(11), 1068–1073. https://doi.org/10.2174/1385272821666170102163307.
236. Pham, T. H., Bui, H. M., & Bui, T. X. (2020). Advanced oxidation processes for the removal of pesticides. In *Elsevier B.V.* https://doi.org/10.1016/B978-0-12-819594-9.00013-9.
237. Ahmed, N., Vione, D., Rivoira, L., Carena, L., Castiglioni, M., & Bruzzoniti, M. C. (2021). A review on the degradation of pollutants by Fenton-like systems based on zero-valent iron and persulfate: Effects of reduction potentials, pH, and anions occurring in wastewaters. *Molecules, 26*(15), 1–17. https://doi.org/10.3390/molecules26154584.
238. Khan, Z. U. H., Gul, N. S., Sabahat, S., Sun, J., Tahir, K., Shah, N. S., Muhammad, N., Rahim, A., Imran, M., Iqbal, J., Khan, T. M., Khasim, S., Farooq, U., & Wu, J. (2023). Removal of organic pollutants

through hydroxyl radical-based advanced oxidation processes. *Ecotoxicology and Environmental Safety, 267*, 1–15. https://doi.org/10.1016/j.ecoenv.2023.115564.

239. Waite, T. D., Chen, Y., & Miller, C. J. (2021). Heterogeneous Fenton chemistry revisited: Mechanistic insights from ferrihydrite-mediated oxidation of formate and oxalate. *Environmental Science & Technology, 55*(20), 14414–14425. https://doi.org/10.1021/acs.est.1c00284.
240. Ponnuchamy, M., Kapoor, A., Senthil Kumar, P., Vo, D. V. N., Balakrishnan, A., Mariam Jacob, M., & Sivaraman, P. (2021). Sustainable adsorbents for the removal of pesticides from water: A review. *Springer International Publishing*. https://doi.org/10.1007/s10311-021-01183-1.
241. Behrami, E., & Avdiu, V. (2023). The process and kinetics of pesticide desorption from clay as a function of cleaning polluted waters. *Processes, 11*(4), 1180. https://doi.org/10.3390/pr11041180.
242. Razali, M. C., Wahab, N. A., Sunar, N., & Shamsudin, N. H. (2023). Existing filtration treatment on drinking water process and concerns issues. *Membranes (Basel), 13*(3), 285. https://doi.org/10.3390/membranes13030285.
243. Yang, J., Monnot, M., Ercolei, L., & Moulin, P. (2020). Membrane-based processes used in municipal wastewater treatment for water reuse: State-of-the-art and performance analysis. *Membranes (Basel), 10*(6), 131. https://doi.org/10.3390/membranes10060131.
244. Zieliński, B., Miądlicki, P., & Przepiórski, J. (2022). Development of activated carbon for removal of pesticides from water: Case study. *Scientific Reports, 12*(1), 25247. https://doi.org/10.1038/s41598-022-25247-6.
245. Guérin, T., Ghinet, A., Hossart, M., & Waterlot, C. (2020). Wheat and ryegrass biomass ashes as effective sorbents for metallic and organic pollutants from contaminated water in lab-engineered cartridge filtration system. *Bioresource Technology, 318*, 124044. https://doi.org/10.1016/J.BIORTECH.2020.124044.
246. Quirantes, M., Nogales, R., & Romero, E. (2017). Sorption potential of different biomass fly ashes for the removal of diuron and 3,4-dichloroaniline from water. *Journal of Hazardous Materials, 331*, 300–308. https://doi.org/10.1016/J.JHAZMAT.2017.02.047.
247. Saleh, I. A., Zouari, N., & Al-Ghouti, M. A. (2020). Removal of pesticides from water and wastewater: Chemical, physical and biological treatment approaches. *Environmental Technology & Innovation, 19*, 101026. https://doi.org/10.1016/j.eti.2020.101026.
248. Aryee, A. A., Mpatani, F. M., Kani, A. N., Dovi, E., Han, R., Li, Z., & Qu, L. (2021). A review on functionalized adsorbents based on peanut husk for the sequestration of pollutants in wastewater: Modification methods and adsorption study. *Journal of Cleaner Production, 310*, 127502. https://doi.org/10.1016/j.jclepro.2021.127502.
249. Sharma, G., Sharma, S., Kumar, A., Lai, C. W., Naushad, M., Shehnaz, J., Iqbal, J., & Stadler, F. J. (2022). Activated carbon as superadsorbent and sustainable material for diverse applications. *Adsorption Science & Technology*, 4184809. https://doi.org/10.1155/2022/4184809.
250. Murtaza, G., Ahmed, Z., Dai, D. Q., Iqbal, R., Bawazeer, S., Usman, M., Rizwan, M., Iqbal, J., Akram, M. I., Althubiani, A. S., Tariq, A., & Ali, I. (2022). A review of mechanism and adsorption capacities of biochar-based engineered composites for removing aquatic pollutants from contaminated water. *Frontiers in Environmental Science, 10*, 1035865. https://doi.org/10.3389/fenvs.2022.1035865.
251. Economic and Social Council (ECN). (2023). Ensuring safe water and sanitation for all: A solution through science, technology and innovation. *Econ. Soc. Counc. United Nations, 728*, 1–17.
252. Emenike, E. C., Adeleke, J., Iwuozor, K. O., Ogunniyi, S., Adeyanju, C. A., Amusa, V. T., Okoro, H. K., & Adeniyi, A. G. (2022). Adsorption of crude oil from aqueous solution: A review. *Journal of Water Process Engineering, 50*, 103330. https://doi.org/10.1016/j.jwpe.2022.103330.
253. Treacy, J. (2016). We are IntechOpen, the world's leading publisher of Open Access books. *Intech*, 1–24.
254. Mishra, B. K., Kumar, P., Saraswat, C., Chakraborty, S., & Gautam, A. (2021). Water security in a changing environment: Concept. *Water, 13*, 1–21.
255. Onu, M. A., Ayeleru, O. O., Oboirien, B., & Olubambi, P. A. (2023). Challenges of wastewater generation and management in sub-Saharan Africa: A review. *Environmental Challenges, 11*, 100686. https://doi.org/10.1016/j.envc.2023.100686.