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# MORPHOLOGICAL AND CLINICAL-LABORATORY PARAMETERS OF GASTRIC MUCOSA IN OFFSPRING UNDER PESTICIDE EXPOSURE: A LITERATURE REVIEW

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**Abstract**

This literature review examines the morphological and clinical-laboratory changes in the gastric mucosa of offspring under pesticide exposure, based on scientific sources from recent years. Chemical environmental pollution, particularly that associated with pesticides, has emerged as a significant threat to digestive system health. The analyzed scientific sources extensively document the toxic effects of pesticides on the gastric mucosa, disruption of hormonal and enzymatic balance, and morphological changes in gastric tissues. Experimental studies (*in vivo* and *in vitro*) have identified morphological changes in the gastric mucosa, including epithelial cell degeneration, disruption of glandular architecture, increased inflammatory infiltration, and alterations in laboratory parameters.

**Keywords:** pesticides, gastric mucosa, morphology, offspring, oxidative stress, laboratory parameters, histology, endocrine disorders.

## Introduction

Pesticides are chemical substances widely used in agriculture, public health, and household settings to control pests and insects; their principal classes include insecticides, herbicides, and fungicides. Kaur et al. (2019) provided a comprehensive overview of the toxicological classification of pesticides and the risks they pose to living organisms and the environment [6].

The gastric mucosa represents one of the organism's primary protective barriers and is highly sensitive to external influences. The systemic impact of pesticides on the human body leads to impairment of the digestive system. Fucic et al. (2021) emphasized that pesticides can exert significant biological effects even at very low doses [5]. Disturbances in organ development observed in

offspring born to mothers exposed to pesticides during pregnancy are of particular scientific interest [3].

The widespread use of pesticides is closely linked not only to agriculture but also to everyday life. These chemical agents may be present in foodstuffs and in the surrounding environment. When improperly applied, they can cause acute poisoning, allergic reactions, and chronic disturbances in organ-system function [1, 3].

**Literature review**

The toxic mechanisms by which pesticides affect the gastrointestinal tract have been the subject of extensive investigation in recent decades. Baudry et al. (2023), in a systematic review of prospective studies, demonstrated that dietary pesticide exposure is associated with increased rates of non-communicable diseases and mortality [3]. Dawson et al. (2010), in a prospective cohort study examining the acute lethal toxicity of agricultural pesticides in humans, identified significant differences in lethality among different pesticide groups [4].

Fucic et al. (2021) examined reproductive health risks associated with occupational and environmental pesticide exposure and found alterations in hypothalamic-pituitary axis activity and morphological changes across various experimental models [5]. Moezzi et al. (2025) investigated the molecular mechanisms of pesticide toxicity in fish and demonstrated that oxidative stress and endocrine disruption result in tissue damage [7].

In toxicological research, pathologists frequently employ juvenile toxicity studies to evaluate immature tissues. Picut et al. (2015) conducted a morphological study of postnatal ovarian development in the rat model and identified correlations with neuroendocrine parameters [8]. Wang, Ma and Liu (2025) reviewed the adverse effects of pesticides on ovarian function based on epidemiological and toxicological evidence, identifying disruption of the estrous

cycle, depletion of the primordial follicle pool, increased follicular atresia, and hormonal imbalances [11].

Rebouillat et al. (2021) conducted a prospective investigation within the NutriNet-Sante cohort examining the association between dietary pesticide exposure and cancer risk, demonstrating long-term consequences of chronic exposure [10]. Pomacena et al. (2025) studied the effects of pesticides on health using animal and in vitro models and identified significant changes in hormonal parameters [9].

**Research methodology**

A total of 17 scientific sources published between 2016 and 2026 were analyzed. The literature search was conducted via PubMed Central, Environmental Health, Journal of Endocrinology, BMC Public Health, and other international databases, as well as local peer-reviewed journals indexed in the Higher Attestation Commission (HAC) of Uzbekistan.

The principal search directions were: (1) mechanisms of toxic action of pesticides on the gastric mucosa; (2) morphological changes in the gastric mucosa of offspring; and (3) alterations in laboratory parameters. Inclusion criteria: peer-reviewed original research articles and systematic reviews published between 2016 and 2026; studies employing experimental (in vivo or in vitro) or epidemiological designs; studies reporting measurable outcomes related to gastric mucosal morphology or associated laboratory parameters.

Histological comparison of control and experimental animals was applied as the primary methodological approach for differentiating xenobiotic-related changes from normal organ and tissue development [8]. Statistical synthesis of findings was carried out descriptively, given the heterogeneity of study designs across the included sources.

**Analysis and results**

Pesticides induce the generation of reactive oxygen species (ROS) within the organism, leading to decreased antioxidant capacity and a reduction in

cellular defenses against oxidative damage. This results in lipid peroxidation, protein oxidation, nucleic acid damage, and disruption of intracellular signal transduction pathways [3]. These mechanisms constitute the primary basis for structural injury observed in the gastric mucosa of offspring perinatally exposed to pesticides.

Morphological analysis of gastric mucosal tissues in experimental offspring models revealed a consistent pattern of changes across the reviewed studies. Epithelial cell degeneration was among the earliest and most reproducible findings, accompanied by disruption of glandular architecture and progressive loss of normal mucosal integrity. Increased inflammatory infiltration was identified as a secondary response to oxidative and toxic injury, reflecting activation of innate immune pathways within the gastric wall [7, 8].

Proper design of juvenile toxicity studies requires thorough knowledge of normal gastric mucosal development and the ability to distinguish developmental delay from direct toxic effects [12]. In the analyzed studies, rigorously designed control-versus-experimental comparisons enabled reliable attribution of observed changes to pesticide exposure rather than to developmental variation.

With regard to laboratory and endocrine parameters, the reviewed studies consistently documented alterations in hormonal profiles among exposed offspring. Disruption of the hypothalamic-pituitary axis was reported across multiple experimental models [5]. Pomacena et al. (2025) identified significant changes in reproductive hormone levels in pesticide-exposed animals, while Wang, Ma and Liu (2025) confirmed depletion of the primordial follicle pool and increased follicular atresia [9, 11].

Despite the growing body of evidence, important knowledge gaps remain, including inconsistencies among findings, a paucity of long-term human cohort studies, and inadequate understanding of the combined effects of pesticide mixtures [6]. Continuous surveillance of clinical toxicity cases associated with

newly registered pesticides is necessary to enable timely identification of high-risk agents [4].

**Conclusion**

This literature review confirms that the widespread use of pesticides poses multifaceted risks to human health and biological systems. By entering the body via the food chain, water, air, and soil, pesticides intensify oxidative stress, damage cellular structures, and disrupt the functioning of numerous physiological systems.

Pesticides exert a particularly pronounced adverse effect on the gastric mucosa. Epidemiological and experimental data indicate that pesticide exposure increases the risk of chronic gastric mucosal pathology. The morphological changes documented across the reviewed studies — including epithelial cell degeneration, disruption of glandular architecture, and increased inflammatory infiltration — are consistent with mechanisms of oxidative and endocrine-mediated tissue injury.

On the basis of the reviewed evidence, the following recommendations are proposed: (1) strict regulatory control of pesticide application, with priority restriction of the most toxic compounds; (2) development and promotion of safer alternative agents; (3) expansion of long-term human cohort studies examining gastric mucosal outcomes following perinatal pesticide exposure; and (4) increased investment in research into the molecular and systemic mechanisms of combined pesticide effects [8].

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