

Chronic Effect of Heavy Metal Exposure on Poultry Health and Performance

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ABSTRACT

The most hazardous heavy metals that are known to be continually released into the environment are cadmium (Cd), lead (Pb), mercury (Hg), and arsenic (As). These metals are released through natural processes, the manufacture of artificial fertilizers, industrial processes, and the disposal of waste. Due to continuous exposure to heavy metals, chickens bio accumulate As, Cd, Pb, and Hg in a variety of organs, primarily the liver, kidneys, reproductive organs, and lungs. The histological changes seen in the liver, kidneys, and reproductive organs are negatively correlated with serum biochemical markers and enzyme activity. The toxicity of metals is dependent on the route of exposure, length of exposure, and absorbed dosage (acute or chronic).

Introduction

The class of metalloids and metals with atomic densities more than or equal to 4000 kg/m³ includes heavy metals [1]. When present in more than certain amounts, toxic elements such as Cd, Pb, nickel (Ni), chromium (Cr), and Hg can be dangerous to living things. These elements are usually linked to pollution. Not only do non-essential elements not appear to be harmful in any appreciable quantity, but they also have no recognized specialized function in the body. Certain heavy metals, such as As, Cd, Pb, Ni, Cr, and Hg are present in water, fish, poultry, and birds in trace amounts. [2]. The spread of heavy metals has been increased in particular by growing anthropogenic activity patterns such as industrialization, mining, the use of chemical pesticides and fertilizers, unregulated sewage outflow, and widespread groundwater irrigation. Toxic metals can be found in agricultural soils for a variety of reasons, including air deposition, wastewater irrigation, agrochemicals, and animal and bird waste products. Reproductive problems and hepato-renal dysfunctions are only a few of the fatal signs that are brought on by high levels of heavy metal accumulation [3]. The two most frequent and toxic heavy metals that accumulate in the food chain are lead and copper. At large concentrations, copper can result in a hemolytic crisis and affect liver, kidney, and brain functions, just like other metals. Poultry exposed to heavy metals may experience weight loss, organ failure, and even death. The path of exposure, duration of exposure, and absorbed dosage acute or chronic determine the toxicity of metals. This review's objective is to provide a thorough overview of the toxicity mechanism, consequences, and histopathological alterations in various poultry tissues that result from exposure to heavy metals [4].

Sources and Effects of Heavy Metal Transmission on Poultry

Sources and Effects of Cadmium

Cadmium is continuously released into the environment at significant levels by both industrial and natural sources. Cd is one of the many contaminants found in the atmosphere, originating from both natural and manmade sources [5]. Anthropogenic sources include the smelting of Cu and Ni, the burning of fossil fuels, the creation of phosphate fertilizers from rocks with varying Cd concentrations, and the application of sewage sludge to the soil. When blood levels of Cd surpass the capacity of metallothionein to bind it, free Cd releases reactive oxygen species and lipid peroxidases that damage the kidneys and liver. High consumption of Cd causes histopathological damage, lowers feed intake, and increases stress sensitivity in chickens, all of which reduce egg production [6].

Sources and Effects of Lead

In industrialized regions of the world, Pb is one of the biggest environmental toxins, and animals are regularly exposed to it. Pb poisoning is especially common in animals and can be caused by several environmental factors, including as industrial pollution, agricultural activities, cars, contaminated feed, and contaminated soil [7]. By producing free radicals, Pb can both induce oxidative stress and act as a catalyst for oxidative reactions in biological molecules. Pb poisoning reduces lysosome activity and influences polymorphonuclear neutrophil phagocytic activity. Many antioxidant defenses are negatively impacted by Pb, and low antioxidant levels can harm organ systems such as the neurological system, liver, kidneys, and reproductive system. Pb poisoning is also known to be fatal in severe conditions in chickens.

Sources and Effects of Arsenic

Poultry in the affected regions is also exposed to hazardous levels of harmful metals, much like animals are. As is a known source of toxicity and is commonly used in animal spray solutions intended to control ectoparasites. [8]. Sources of As contamination include contaminated drinking water, feed, plants, grasses, and air pollution. The primary sources of As include grasses, vegetables, tainted drinking water, and feed additives. Even at very low levels in food, arsenic is extremely dangerous and its significance in poultry nutrition is strongly disputed. Acute As poisoning in poultry can result in hypothermia, watery diarrhea, increased salivation, stomach pain, circulatory collapse, and even death. Prolonged exposure to low concentrations of As in poultry can cause skin coloring, stomach pains, and persistent indigestion.

Sources and Effects of Mercury

One of the most powerful neurotoxins, Hg can have a variety of detrimental effects on an animal's or human's health. High toxicity and capacity of Hg for biomagnification and bioaccumulation make it, along with other non-essential trace elements, an important environmental pollutant. The primary industries that release Hg into the atmosphere are those that deal with paper, fungicides, chemicals, pesticides, paint, and geothermal steam used to produce power [9]. Hg is a well-known hazardous substance that, even at extremely low exposure levels, can have catastrophic consequences on poultry, including damage to the kidneys and liver. Poultry exposed to toxic levels of mercury may have symptoms like anemia and slowed growth. Mercury exposure can harm tissues and organs, and it is absorbed and dispersed in poultry's liver and kidneys.

Histopathological Changes in the kidney of poultry

The second most seriously affected organ by Cd poisoning is the kidneys, which are in charge of eliminating toxic compounds from the body. Necrotic lesions and eosinophilic intranuclear inclusion in the renal tubule epithelial cells were observed in the kidney's histopathological alterations. Increased renal tubules, tubular hyalinization, fibrosis, a fold rise in nucleosome content, elevated levels of malondialdehyde (MDA), and decreased levels of intracellular glutathione (GSH) in the kidney were among the histopathological abnormalities brought on by mercury deposits in the kidney [11]. Renal tubular fibrosis, expansion of the renal tubules, severe hyalinization, lowered renal SOD activity, reduced renal GSH-Px activity, decreased CAT and GR activity, and higher renal MDA level are among the histopathological alterations in the kidney caused by exposure to As. Variations in autophagic responses, cell adhesion, and cellular signaling cascades are brought about by the toxicity caused by Cd in the kidneys [13].

Histopathological changes in brain tissues of Poultry

Histological alterations in the brain following As poisoning in chicken induce lesions in the brain, mitochondrial enlargement, infiltration in glial cells, vacuolation, and acute hemorrhage, all of which contribute to As toxicity and neuronal cell death. Histopathological changes in the reproductive system of Poultry Chickens were found to have damaged the blood-testis barrier, and some seminiferous tubules [12]. Pb deposition caused spermatogenic cells to be arranged irregularly, producing more spermatogonium, and distorting spermatogenic tubes.

Histopathological Changes in the Liver of Poultry

Cd is mostly transported to the liver by portal blood circulation, where it is then absorbed by hepatocytes from the liver's sinusoidal capillaries. The increasing dosage of Cd led to large macrophage infiltrations in the liver, as well as an increase in the size, damage, and necrosis of hepatic cells. The daily treatment of 50 mg/L of Cd caused hepatocytes, macrophages, plasma cells, lymphocytes, and livers to swell, become fragile, and develop localized necrotic patches and sinusoidal gaps. Because of the underlying hepatic injury caused by Cd, endothelial cell destruction results in ischemia [14]. Acute exposure to copper stimulates a variety of different types of liver cells, Kupffer cells, and several cytotoxic mediators and inflammatory, resulting in secondary liver injury. Pb exposure can cause histological abnormalities in chicken livers, as Pb accumulates in the liver. The animals' livers showed abnormal changes following exposure to high Pb concentrations, including severe sinusoid congestion, hepatocellular cytoplasm hyalinization vacuolization, hepatic lipid vacuolization, and irregularity and dilatation of the blood sinusoids. As accumulation causes histopathological alterations in the liver, such as increased MDA levels, reduced GSH levels, reduced hepatic SOD activity, and reduced CAT, GR, and GSH-Px activity [15].

Conclusion

Four heavy metals (As, Cd Pb, and Hg) accumulate in different organs as a result of chronic exposure; As, Pb and Hg indicate declining concentration patterns, whereas Cd has greater concentrations. The liver is the organ most affected by these heavy metals, followed by the reproductive system, kidney, and brain. Heavy metals also affect other organs in poultry.

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