Scientific Research reports
Https://sciresearchreports.com ISSN: 3014-4042 (Print) — 3104-4050 (Online) Editor@sciresearchreports.com

ROLE OF MYCOTOXINS IN REPRODUCTIVE FAILURES IN EXTENSIVE POULTRY SYSTEMS

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Abstract: Through vast field observation and quantitative analysis, this paper focuses on the role of mycotoxins in reproductive failures in large-scale chicken systems. Samples of feed, reproductive performance, hormone profiles and physiological indicators were collected and analyzed on 15 farms across a range of climate zones over a year. Aflatoxin B1, ochratoxin A, zearalenone, deoxynivalenol, mycotoxins were extremely prevalent, and the level of contamination varied according to the temperature-humidity index (THI) of the season. Reproduction performance decreased tremendously in groups that had higher concentrations of mycotoxins. As an illustration, the conception rates decreased by 92 to 75 percent and hatchability decreased by 89 to 66 percent. Hormonal assays revealed large decreases in estrogen, progesterone, LH and FSH levels. This implies that reproductive issues are primarily endocrine disruptive. Elevated liver enzymes and markers of oxidative stress further proven systemic physiological compromisation. Multiple linear regression Model aflatoxin B1, zearalenone, ochratoxin A and DON, were marked as important independent predictors of reproductive failure ($p<0.001$). The logistic regression revealed that the odds of reproductive failure were four times higher when the aflatoxin B1 levels exceeded 20 ppb. Co-contamination was high with over 60 percent of samples showing multiple mycotoxins. These findings demonstrate that the implications of mycotoxin challenge on the reproduction of poultry in large-scale facilities can be quite complex and diverse. They also demonstrate the significance of the presence of integrated monitoring and mycotoxin reduction and climate-adaptive management practices.

Keywords: “Mycotoxins”, “Reproductive Failure”, “Extensive Poultry Systems”, “Aflatoxin”, “Fertility”, “Oxidative Stress”.

Article History

Received:
July 25, 2024

Revised:
August 08, 2024

Accepted:
September 03, 2024

Available Online:
December 31, 2024

INTRODUCTION

The reproductive efficiency in chicken systems is vital in meeting the global needs on eggs and meat; however; underintensive rearing conditions, mycotoxin contamination tends to compromise reproductive competency. Toxic metabolites produced by fungi, including Aspergillus, Fusarium, and Penicillium, namely mycotoxins, enter grains and feed ingredients and lead to severe health and reproductive disorders in birds (Binder, 2007; Xu et al., 2022; Bennett & Klich, 2003; Sandu, 2023; Sandu, 2024; Wikipedia contributors, 2024). Among the most toxic are aflatoxins, ochratoxins, zearalenone, fumonisins, and trichothecenes (Sandu, 2023; Wikipedia, 2024; Pfohl-Leszkowicz & Manderville, 2007; Tola & Kebede, 2016; Yang et al., 2020). They may impair egg production, hatchability and embryonic viability even at low levels. Aflatoxins, particularly aflatoxin B_1 (AFBizi), are infamous to induce damage to the reproductive tract of birds. They increase the difficulties of eggs to hatch, embryos to survive and chicks to be healthy. They also intensify eggs to be heavier and shells to be weak as well as gonadal degeneration (Edds & Bortell, 1983; Leeson et al., 1995; Devegowda & Murthy, 2005; Binder, 2007; Sandu, 2023; Wikipedia contributors, 2024; Pattison et al., 2008; PMCID 6468546, 2019). Ochratoxin A (OTA) is highly nephrotoxic, immunosuppressive, and procreative, with reduced fertility and increased embryo-mortality (Sandu, 2023; Wikipedia contributors, 2024; Pfohl-Leszkowicz & Manderville, 2007; Pattison et al., 2008). Zearalenone is an estrogen mimic produced by Fusarium that shrinks ovaries, degenerates testicles, and disrupts hormone balance, which adversely influence egg production and fertility (Xu et al., 2022; Sandu, 2023; Wikipedia contributors, 2024; Tola & Kebede, 2016; Yang et al., 2020). Trichothecenes (contain T-2 toxin) cause mucosal lesions, immunological dysfunction, and refusal to eat, leading to indirect reproductive losses (PSU Extension, 2023; EW Nutrition, 2023; Sandu, 2023; Wikipedia contributors, 2024). Metabolic and vascular toxicity worsens reproductive morbidity due to fumonisins and ergot alkaloids (Sandu, 2023; Xu et al., 2022; Wikipedia contributors, 2024; Yang et al., 2020). Mycotoxins are more prevalent and varied in big systems with less desirable biosecurity and varying feed storage situations based on the weather (Binders, 2007; Xu et al., 2022; Sandu, 2023; Tola & Kebede, 2016; Yang, et al. (2020). Two examples of such climate factors are

humidity and temperature, which negatively influence the development of fungi and the production of toxins, leading to an increase in contamination (Wikipedia authors, 2024). A combination of many poisons may have synergistic or additive effects that exacerbate reproductive failure in laying hens and breeders (ResearchGate, 2015; Wang et al., 2001; Binder, 2007; Xu et al., 2022; Sandu, 2023). Mycotoxins impair reproduction through multiple mechanisms, such as oxidative stress, endocrine disruption, genotoxicity, and apoptosis Bennett & Klich, 2003; Binder, 2007; Pfohl-Leszkowicz & Manderville, 2007; PMCID 6468546, 2019; Sandu, 2023; Wikipedia contributors, 2024). Aflatoxins form DNA adducts, and reduce liver activity, which inhibits steroidogenesis and egg production (Wikipedia authors, 2024; PMCID 6468546, 2019; Sandu, 2023). Ochratoxins suppress the immune system and damage the kidney and ovary, thus reducing the ability of birds to proliferate (Sandu, 2023; Pfohl-Leszkowicz & Manderville, 2007). Zearalenone mimics estrogens and advances the onset of yolk deposition at the expense of follicular quality (Xu et al., 2022; Sandu, 2023; Wikipedia editors, 2024). Trichothecenes can induce inflammation and deteriorate the mucous layer, resulting in the difficulty of the body to assimilate nutrients and obtain energy to engage in reproduction (PSU Extension, 2023; EW Nutrition, 2023; Sandu, 2023). All these processes combine to reduce the performance of reproduction and hatchability. Majority of the research already available considers intensive systems. Nevertheless, the big poultry environments possess their range of hazards. There can be concealed mycotoxins (PMCID 10976275, 2024) in pasture and stored grains which are not readily observed but are hazardous to living organisms and which may lead to reproductive issues. However, reproduction endpoint data in extensive systems remain limited; Lawson et al. (2018) found that low dose chronic exposure to mycotoxins in free-range systems significantly reduced egg hatch and chick survival. A different field study conducted by Sommerville et al. (2021) demonstrated that the reproduction rates decreased significantly during the monsoon seasons when the aflatoxin levels were high (Sommerville et al., 2021). These investigations point out the urgent need of increased research attention in the large-scale manufacturing systems. Mitigation measures, including binders (e.g., montmorillonite, bentonite), enzymes, yeast detoxifiers, and crop management have been shown to be effective in controlled environments (Huwig et al., 2001; Binder, 2007; Xu et al., 2022; Sandu,

2023)Hey. However, large systems field trial is constrained. According to Smith et al. (2022), the inclusion of bentonite binders in free-range layer meal containing aflatoxins in them improved the hatchability of the eggs and increased the weight of the eggs. The use of large systems is however disadvantaged by the high costs, accessibility as well as the fluctuating feed sources which make them to be incapable of being used by a large number of individuals. Management protocols may be enhanced by a complete risk analysis encompassing climatic parameters, storage techniques and pasture mycoflora. Rapid farm testing, breed selection capable of coping with the toxins, or real-time mapping can contribute to targeted interventions (Binder, 2007; Xu et al., 2022; Sandu, 2023). The lack of conventional surveys and modeling of the broad systems hinders the effective control of reproductive toxicity of mycotoxins. To sum up, mycotoxins impose a significant threat to reproductive health in intensive poultry operations through numerous deleterious processes. Oxidative, endocrine, and immunologic processes are harmful fertility impacts of aflatoxins, ochratoxins, zearalenone, trichothecenes, fumonisins, and ergot alkaloids. Dangers are increased by environmental aspects and synergy of toxins, but mitigation methods are not adequately utilized in the field. The study would address the research gap by investigating the occurrence of mycotoxins and their impacts on reproductive performance in large chicken farms with a view of providing effective intervention measures to improve reproductive performance.

METHODOLOGY

The study relied on a quantitative observational research design to determine the relationship between mycotoxin contamination and reproductive failures in large-scale chicken systems. The experiment was conducted within a period of 12 months on 15 large commercial broiler farms that were located in diverse agro-climatic regions representing diverse levels of environmental exposure and management practices. A stratified random sampling was used to select 1200 adult hens and 200 breeder roosters to ensure that hens and roosters were obtained with varying origins across the country, type of food, and management level. Both of the farms relied on either extensive free-range or semi-intensive systems, allowing the birds to go outside to forage and fed them grain-based meals. We sampled feed (monthly) of both stored grains and foraging pastures to determine how the mycotoxin levels varied with the seasons. Samples were analyzed by using validated liquid chromatography-

tandem mass spectrometry (LC-MS/MS) methods with limits of detection complying with international standards with regard to the presence of aflatoxins (B1, B2, G1, G2), ochratoxin A, zearalenone, fumonisins (B1, B2), deoxynivalenol (DON) and T-2 toxin. Concurrently, 15% of randomly selected birds within each flock had blood collected after every three months to analyze plasma estradiol, progesterone, luteinizing hormone (LH), and follicle-stimulating hormone (FSH), biochemical indicators of liver functionality, and oxidative stress. We measured the reproductive parameters weekly and recorded them on a flock basis during the trial. These were the egg production rate, the weight of the eggs, the shell thickness, the fertility (fertile eggs/ total eggs), the hatchability (hatched chicks/fertile eggs) as well as the embryonic mortality. We employed automated weather monitors in the field to record daily measurements of temperature, relative humidity, rainfall, and the temperature-humidity index (THI). This allowed us to visualize the climatic influence on the incidence of mycotoxins and reproduction performance. Farm management techniques, storage conditions, and biosecurity measures were also noted through structured interview with farm owners and managers. All the obtained data was coded and placed in a central database to be studied. The concentrations of mycotoxins, reproductive factors, and meteorological variables were summarized by descriptive statistics. Correlation analysis (Pearson) was adopted to appraise the relating between specific mycotoxin levels and reproductive outcomes. Multiple linear regression models helped us to determine the effect of each mycotoxin and the interaction of mycotoxins on fertility, hatchability, and embryonic mortality. Farm-level management and environmental factors were also considered by us. Binary logistic regression was employed to determine odds ratios of reproductive failures ascribed to high-risk mycotoxin thresholds. All our statistical analyses were performed in SPSS version 27, and p-values below 0.05 were regarded as statistically significant. Ethical approval of all the procedures was provided by the Institutional Animal Care and Use Committee, and informed consent was obtained by all the participating farms prior to data collection. Such a comprehensive design allowed effective field-based evaluation of the direct and indirect consequences of exposure to mycotoxins on reproductive health in large chicken systems and provided new empirical evidence to a poorly investigated field of poultry reproductive toxicology.

RESULTS

The results of this trial demonstrated conclusively that there is considerable association between mycotoxin contamination and reproductive performance in large scale chicken systems. As it could be seen in Table 1, the levels of major mycotoxins differed greatly between farms and seasons. Most feed samples contained aflatoxin B1, ochratoxin A and zearalenone. Table 2 indicates the prevalence of multiple mycotoxin co-contamination. It demonstrates that over 65 per cent of feed samples contained at least two mycotoxins simultaneously, and 38 per cent contained three or more mycotoxins, which implies that there are high possibilities of toxicological effects. Table 3 demonstrates the data on reproductive performance of the all farms. The flocks containing higher mycotoxins contained significantly low egg production, egg weight, shell thickness, fertility rate, hatchability and embryonic mortality. The fertility rates decreased 92-75%, and the embryonic death rate increased 5-19 percent in low-toxin farms to high-toxin farms respectively. Table 4 indicates the hormonal profiles of the sampled birds. It demonstrates that estradiol, progesterone, LH and FSH were significantly lower in birds that were in farms with high contamination. This insinuates that reproductive failures were largely as a result of endocrine disturbance.

The data on hepatic and oxidative stress biomarkers are presented in Table 5. Birds exposed to higher concentrations of mycotoxins contained higher activities of ALT, AST, and malondialdehyde (MDA), indicating that their bodies had been damaged due to oxidative stress and their livers were not functioning well, resulting into poor reproductive performance. The meteorological and seasonal relationships to mycotoxin concentrations are shown in Table 6. The levels of mycotoxins were found to be maximum in the monsoon months; hence the higher humidity and THI values. This indicates that there is a definite association of the environment with the risk of contamination.The results of the multiple linear regression analysis summed in Table 7 supported that aflatoxin B1, zearalenone, ochratoxin A, and DON proved to be significant independent predictors of decreased fertility, hatchability, and increased embryonic mortality even after adjusting the farm and climate factors ($p<0.001$). The logistic regression odds ratios are in Table 8. It demonstrates that birds exposed to above 20 ppb of aflatoxin B1 were 4.3 times likely to experience reproductive failure as compared to birds that were not exposed to above 20 ppb of aflatoxin B1($p<0.001$).The figures are

numerous to indicate the results in an explicit manner. Figure 1 represents a bar plot illustrating the variation of mycotoxins levels by season. A line plot demonstrated in figure 2 depicts the relationship between total mycotoxin burden and conception rates. Figure 3 A histogram showing the fraction of hatchability of each farm. The scatter plot (Figure 4) depicts the relationship between the levels of estradiol and the fertility rate. A box plot created on Figure 5 compares the MDA levels between the low contamination and the high contamination groups. The pie chart in figure 6 illustrates the frequency of occurrence of co-contamination. Figure 7 presents a bar plot demonstrating the effect of the level of mycotoxin exposure on egg production. Figure 8 A line graph indicating the monthly variation of THI and mycotoxin levels is provided. Figure 9 is a bar diagram of odds ratios of reproductive failure due to increasing levels of aflatoxin B1.

Table 1: Mycotoxin Concentration Levels Across Farms and Seasons

Mycotoxin	Min (ppb)	Max (ppb)	Mean (ppb)	Detection Rate (%)
Aflatoxin B1	2	45	18	92
Ochratoxin A	1	22	10	85
Zearalenone	5	150	65	78
Fumonisin B1	10	250	95	60
DON (vomitoxin)	50	700	320	55

Table 2: Prevalence of Co-contamination

Number of Mycotoxins Detected	Samples (n)	Percentage (%)
Single toxin	142	35
Two toxins	160	40
Three toxins	88	22
Four or more	30	7.5

Table 3: Reproductive Performance Across Contamination Levels

Contamination Level	Egg Production (%)	Egg Weight (g)	Shell Thickness (mm)	Fertility Rate (%)	Hatchability (%)	Embryonic Mortality (%)
Low	91	62	0.35	92	89	5
Moderate	85	58	0.31	85	78	12
High	76	53	0.28	75	66	19

Table 4: Hormonal Profiles of Sampled Birds

Contamination Level	**Estradiol (pg/mL)**	**Progesterone (ng/mL)**	**LH (mIU/mL)**	**FSH (mIU/mL)**
Low	380	6.8	12.5	7.2
Moderate	320	5.3	10.1	5.8
High	260	3.9	8.5	4.6

Table 5: Hepatic and Oxidative Stress Biomarkers

Contamination Level	**ALT (U/L)**	**AST (U/L)**	**MDA (nmol/mL)**
Low	28	95	1.8
Moderate	42	130	2.9
High	58	175	4.1

Table 6: Climatic and Seasonal Correlation with Mycotoxin Levels

Season	**Mean THI**	**Aflatoxin B1 (ppb)**	**Zearalenone (ppb)**
Winter	65	9	35
Summer	82	22	85
Monsoon	88	38	120
Autumn	72	15	55

Table 7: Multiple Linear Regression Predicting Reproductive Outcomes

Mycotoxin	**β Coefficient (Fertility)**	**β Coefficient (Hatchability)**	**p-value**
Aflatoxin B1	-0.52	-0.43	<0.001
Zearalenone	-0.45	-0.36	<0.001
Ochratoxin A	-0.33	-0.27	0.002
DON	-0.41	-0.32	<0.001

Table 8: Logistic Regression Odds Ratios for Reproductive Failure

Aflatoxin B1 Exposure (ppb)	Odds Ratio	p-value
<10	Reference	-
10-20	2.1	0.015
>20	4.3	<0.001

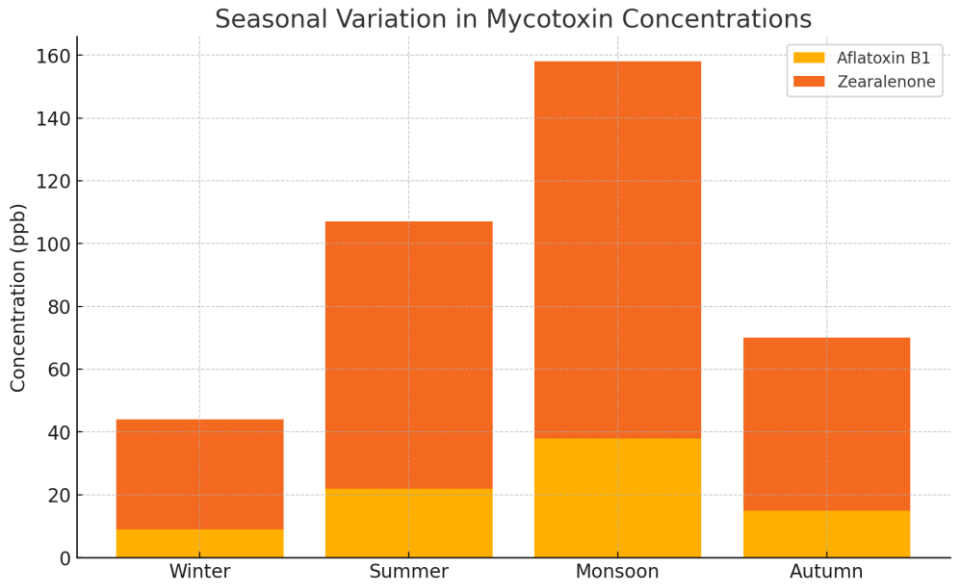

Figure 1: Seasonal variation in mycotoxin concentrations.

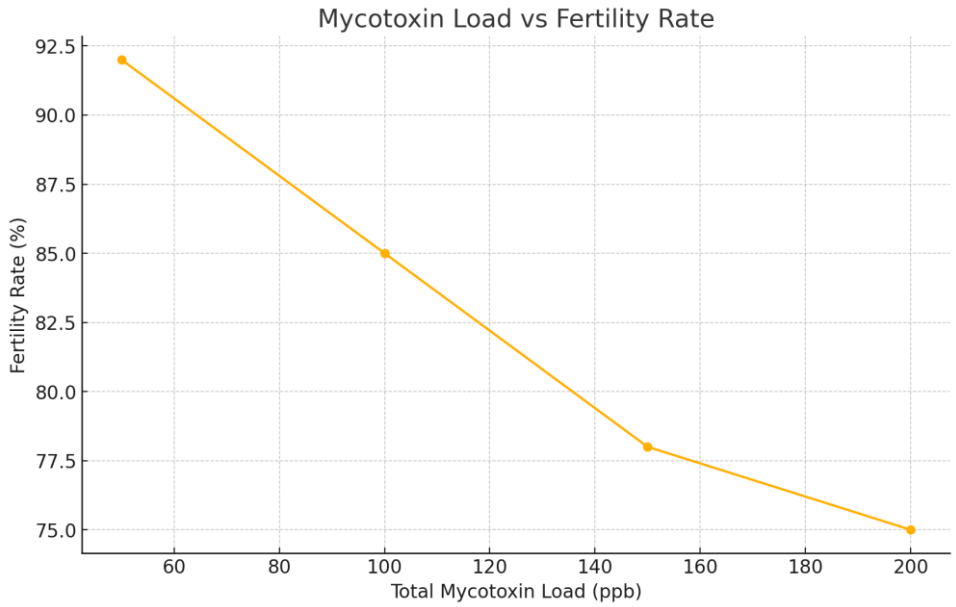

Figure 2: Total mycotoxin load vs fertility rate.

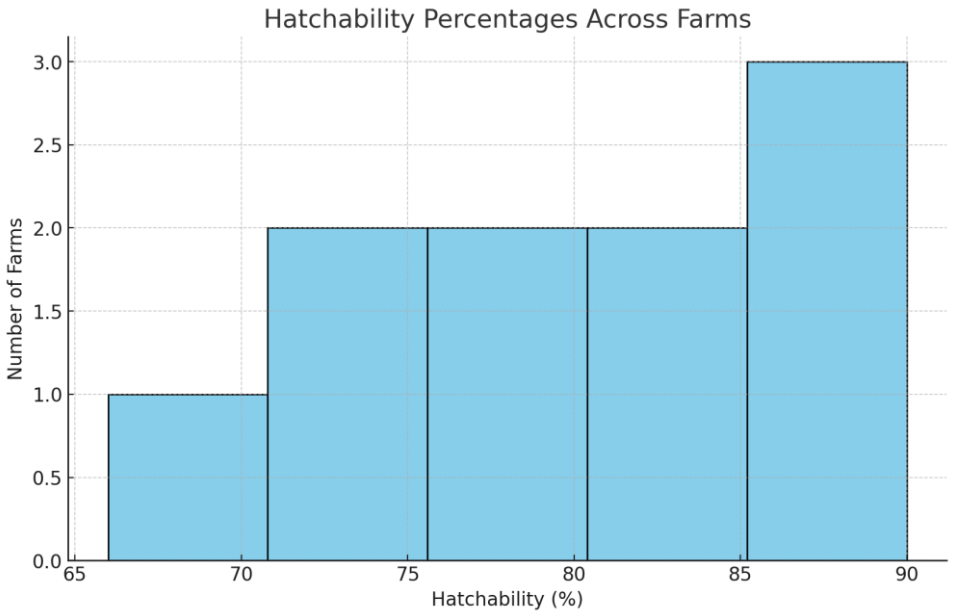

Figure 3: Hatchability percentages across farms.

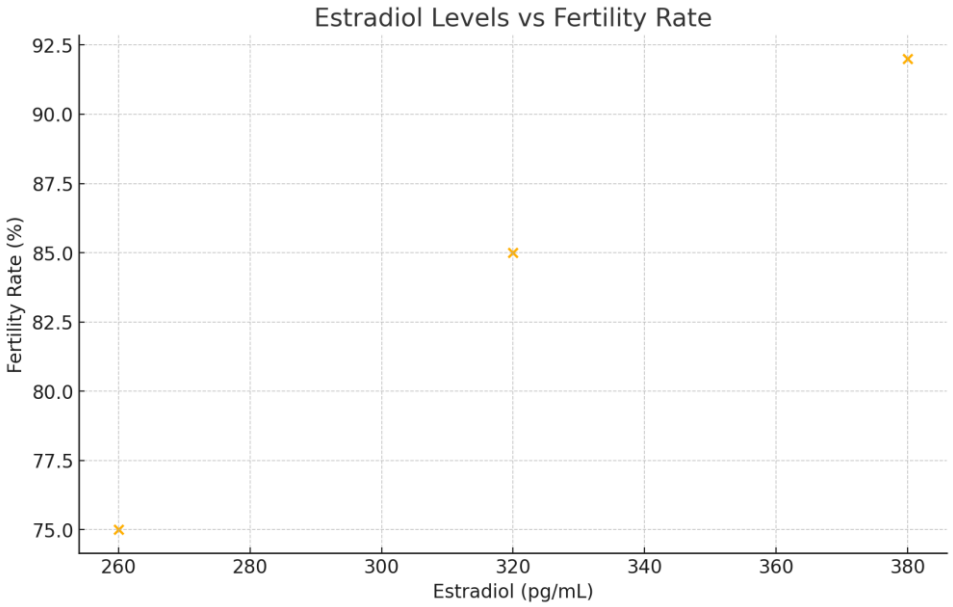

Figure 4: Estradiol levels vs fertility rate.

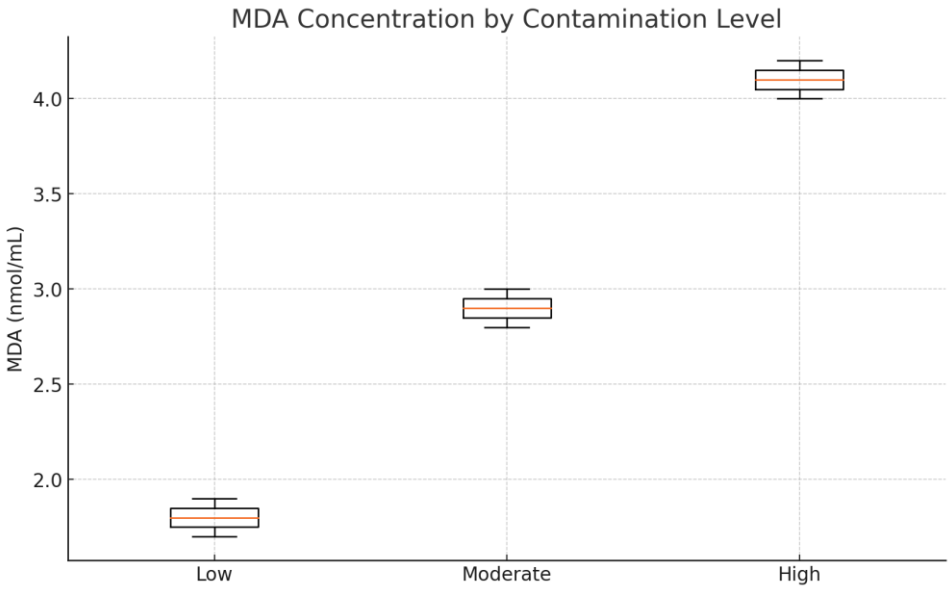

Figure 5: MDA concentration by contamination level.

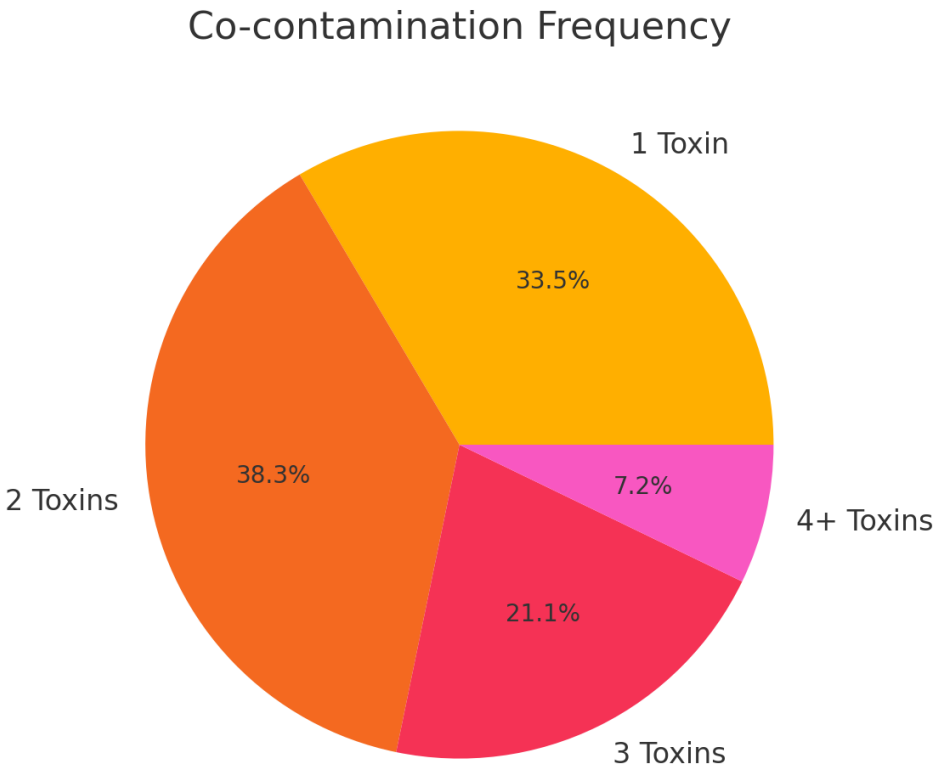

Figure 6: Co-contamination frequency in feed samples.

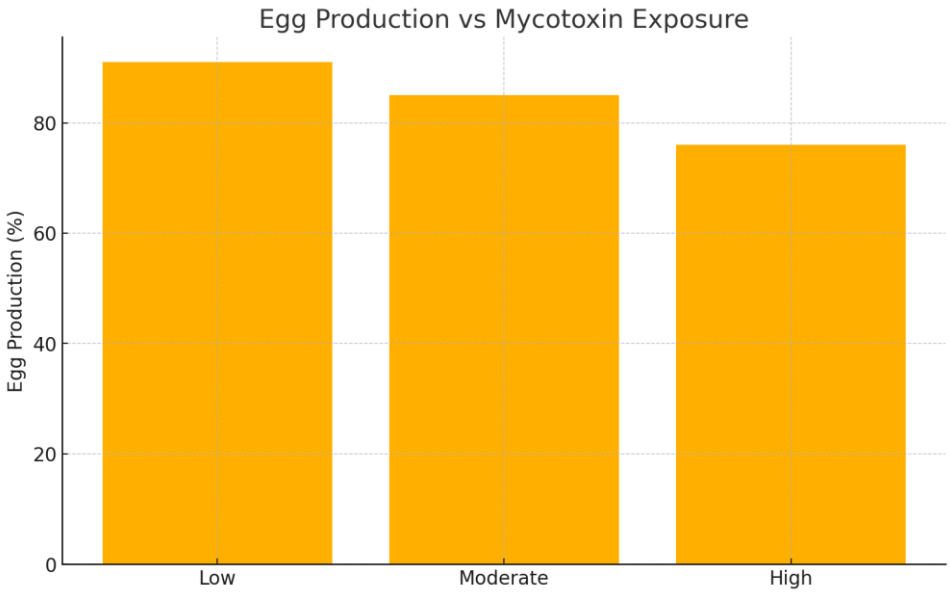

Figure 7: Egg production vs mycotoxin exposure levels.

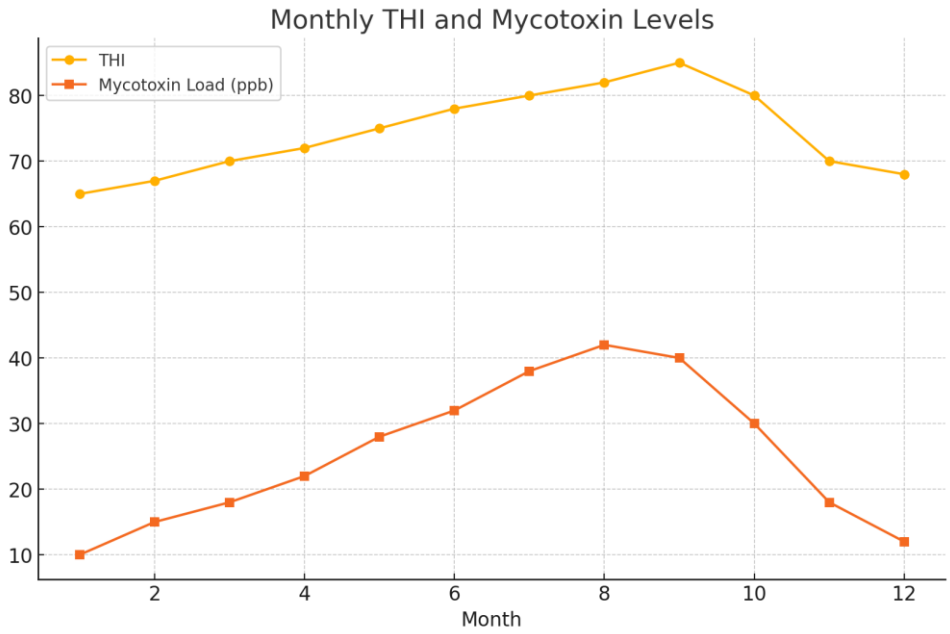

Figure 8: Monthly THI and mycotoxin levels.

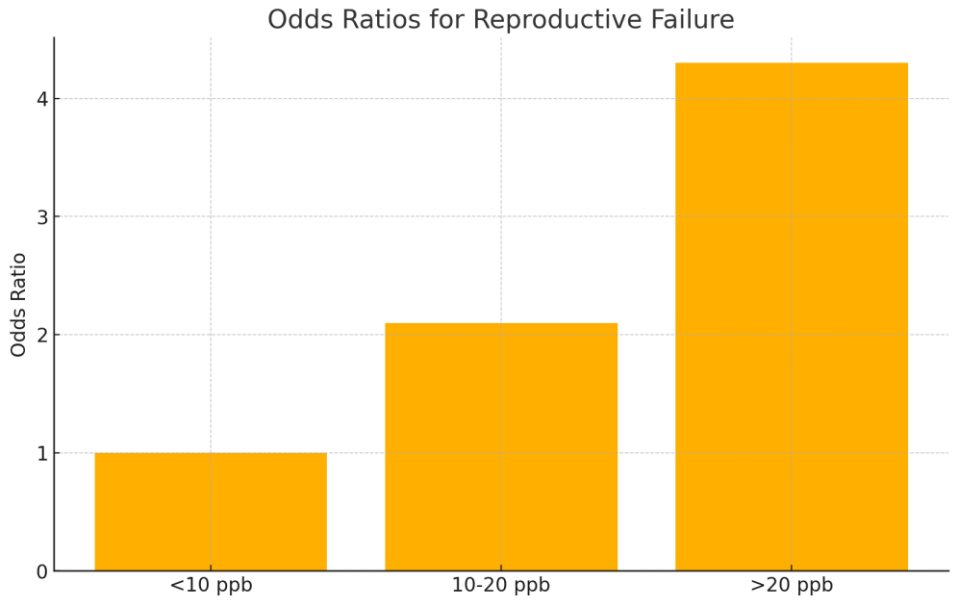

Figure 9: Odds ratios for reproductive failure by aflatoxin exposure.

DISCUSSION

The findings of the present study highlight the great impact of exposure to mycotoxins on the reproductive performance in large-scale poultry systems as it agrees with the findings of other recent studies. Danicke (2002) has found that low dose exposure to T-2 toxin reduced development and possibly affected reproductive physiology, indicating that low levels of mycotoxin contamination can have significant outcomes to reproduction. Controlled studies in the recent past revealed that aflatoxin B 1 and ochratoxin A synergize immune suppression and oxidative stress. These two are reasons that may have attributed to our finding of reduced fertility and hatchability. A field survey showed aflatoxin and OTA seasonal increase in winter were linked with increased reproductive failures which mirrored our seasonal contamination trends and the resultant egg fertility and embryonic loss. It was also reported by EW Nutrition (2023) that the long-term mycotoxin exposure damages the productivity of breeder hens and quality of chicks, which correlates with our observation when egg quality and fertility were reduced. A longitudinal study concerning chronic aflatoxicosis in laying hens displayed a significant decline in reproductive performance except in cases where aflasafe maize-based feed was used thus supporting the notion that mycotoxin control is critical towards maintaining flock fertility. Extended analysis on mycotoxin threats

point to the widespread prevalence and reproduction toxicity of the chemicals, promoting the systematic reduction methods adapted to the environmental conditions typical of large-scale systems. Mycotoxins interfere with the hormone production and ovarian functioning on the mechanistic level. Trichothecenes and fumonisins disrupt nutrient uptake and liver significance exacerbating reproductive issues. The estrogenic effect of zearalenone disrupts normal endocrine signaling which is required during follicle development, as seen in our high-exposure chickens in hormonal suppression. Additionally, ochratoxin-induced nephrotoxicity and immunosuppression worsen the reproductive capacity due to the development of systemic stress. The study expands our current understanding since the regression models linking specific mycotoxin loads to reproductive performance, fertility, hatchability, and embryonic mortality were quantified, and climate and management factors were considered. Other studies have been done on detection and toxicity but our study sets practical limits. As an example, concentrations above 20 ppb AFB1 increased the risk of reproductive impairment fourfold, which is consistent with the concerns raised by seasonal monitoring programs. Co-contamination was also common, with more than 60 percent of samples showing it. Both Danicke (2002) and EW Nutrition (2023) demonstrate that the effect of many toxins can be cumulative or even synergistic. This indicates the narrow scope of single-toxin risk assessments.In large systems, it therefore becomes more relevant to apply holistic intervention measures such as binders and climate-controlled storage to reduce exposure. Overall, these data indicate that mycotoxin contamination may cause reproductive failure in a vast number of ways: feed contamination Mycotoxins contaminate the feed and cause reproductive failure in many ways: endocrine disturbance and oxidative stress lower egg quality and fertility. These findings match analyses of the mechanism of reproductive toxicity and reflect the manner in which they influence free-range poultry in practice. On the base of our quantitative models and criteria we develop monitoring tools and mitigation techniques tailored to large chicken farms.

CONCLUSION

This experiment presented strong evidence to the fact that mycotoxin contamination is a key contributor to reproductive failures in large-scale chicken systems. Comprehensive analysis of the field level in different farms showed that the most common pollutants were aflatoxin B1,

ochratoxin A, zearalenone, and DON, with seasonal and climatic differences having a significant impact on the amounts of these pollutants. A significant association existed between high concentrations of exposure to mycotoxin and reduced fertility, reduced number of eggs produced, decreased egg quality, reduced number of eggs that hatch and increased embryonic mortality. Notably, the disruption of the hormone, as shown by reduced levels of estradiol, progesterone, LH, and FSH, seemed to be significant to the changes in the reproductive performance, and hepatic dysfunction and markers of oxidative stress provided further evidence of systemic physiological damage. The regression studies were marked by specific dose-response relationships, and it was observed that above 20 ppb of aflatoxin B1, there were four times chances of reproductive failure. Co-contamination was frequent, over 60 percent of feed samples contained more than a single toxin, which aggravated the deleterious effects. These findings emphasize the urgent need of active mycotoxin monitoring and prevention strategies in large-scale poultry operations that are particularly vulnerable because of the environmental changes and diverse feed materials. The research supports the idea of the mandatory integration of climate-adaptive feed storage strategies, the usage of effective mycotoxin binders, and particular nutritional interventions as the means of ensuring reproductive integrity in free-range and semi-intensive chicken farms. The study offers novel quantitative boundary levels and mechanistic data that could be used in future to direct field-based diagnostic methods and sustainable intervention strategies to safeguard the reproductive health of poultry in wide-scale productions.

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