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The science behind poultry aspergillosis

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Abstract
Fungal infections in poultry, though less prevalent than bacterial and viral diseases, are increasingly recognized as significant respiratory threats, with aspergillosis being the most common mycosis. The disease, primarily caused by *Aspergillus fumigatus* and *A. flavus*, arises from inhalation of spores found in contaminated litter, feed, hatcheries, or ventilation systems. Stress, poor ventilation, overcrowding, nutritional deficiencies, and immunosuppressive conditions increase susceptibility, particularly in young chicks, turkeys, and ducklings. The infection manifests in acute or chronic forms, depending on spore load and host immunity, and may affect the respiratory and nervous systems. Seasonality, environmental humidity, and housing practices strongly influence outbreaks, with higher prevalence during warm, damp conditions. While moldy substrates such as feed, feathers, and litter act as reservoirs, hatchery contamination remains a key source of infection. Clinical outcomes vary with immune status, co-infections, and environmental stressors, often leading to high mortality. Interestingly, certain *Aspergillus* species, such as *A. niger* and *A. awamori*, exhibit probiotic effects when used as dietary supplements, highlighting a paradoxical role of the genus in poultry health. Effective management requires strict biosecurity, proper nutrition, and improved housing practices.

Keywords: Aspergillosis, poultry health, respiratory infections, *Aspergillus fumigatus*, *Aspergillus flavus*, biosecurity, mycosis

Introduction
Fungal infections are common across the globe in poultry but, less prevalent to bacterial and viral infections (Abd El-Ghany, 2021; Agbato *et al*, 2024) [2, 5] and emerged as major respiratory disease owing to extensive use of antibiotics, corticosteroids and changed in farming systems (Biswas *et al*, 2010; Radwan *et al*, 2016; Mersha and Dawit, 2017) [31, 159, 124]. Aspergillosis holds a very special place in poultry medicine being the main mycosis affecting birds. Initial contamination of farms may occur through the use of a moldy litter or introduction of day-old chicks whose down has retained the conidia in hatchery facilities (Arne *et al*, 2011) [14]. This disease is a fatal airborne illness, widely distributed in chickens young or old including broiler breeders (Martin *et al*, 2007) [125], turkeys, ducks, geese, pigeon, wild birds (Zeinab *et al*, 2024) [227] caused by toxins produced from fungus affecting the lower respiratory system (Fedde, 1998; Kunkle, 2003) [65, 112] and nervous system. All ages of avian species are susceptible (Pazhanivel *et al*, 2018, Talbot *et al*, 2018; Hauck *et al*, 2020) [148, 204, 84] mainly as respiratory infection (Nururrozi *et al*, 2020) [139]. Chickens are particularly susceptible to aspergillosis because of their natural behavior of soil pecking and scratching which exposes to spores in the environment.
Stress is the main predisposing factor (Saif *et al*, 2003; Copetti *et al*, 2004) [112, 43] that results from mismanagement in commercial operations or backyard poultry (Jordan *et al*, 2002) [100]. Clinical manifestations depend on the infective conidia inhaled, pre-existing disease and the immune status of the host (Romina *et al*, 2019) [178]. *Aspergillus fumigatus* infection (Amer and Karim, 2019) suppress immunity against Newcastle disease in chickens. An immunosuppressive metabolite from *Aspergillus fumigatus* - a galactosaminogalactan predispose birds to fungal, bacterial and viral infections (Javed *et al*, 2005; Fontaine *et al*, 2011) [93, 69]. Aspergillus spores are ubiquitous in the environment and grow anywhere in hot organic matters under humid environments. Dry litter conditions promote aerosolization of spores (Barbale, 2002; Flammer 2002) [21, 68].

Organic substrates like egg yolk, cardboard boxes, litter, old feed and feathers fulfil their nutrient requirements (Santos *et al*, 1996; Latge, 1999, 2001) [187, 114, 115].
Acute aspergillosis occurs on ingestion of large number of spores or through eggshell penetration and the chronic form in immunologically compromised birds. Hatchery provides the optimum environment for Aspergillus spores to thrive upon their entry through the eggs or incoming air. Contaminated eggs are the major source of spores that enter air handling units and ventilation system. The temperature and humidity in ventilation ducts are ideal for propagation of the molds especially when organic derbies have accumulated.
Few species of aspergillus have been used as dietary supplementation in birds. Aspergillus Niger improved broiler growth and meat quality (Saleh *et al*, 2011) [182]. A. awamori have probiotic and antibiotics effect (Saleh *et al*, 2014) [183] and combination of A. awamori with lactic acid bacteria increased unsaturated fatty acid content and reduces saturated fatty acid in egg yolk (Saleh *et al*, 2017) [184].

Etiological Agents
Brooder pneumonia in poultry is caused by Aspergillus. These organisms are common soil saprophytes which grow on organic matter under warm and humid environment including the damaged eggs (Atlaman, 1997; Lobna, 2014) [17, 118]. The most common pathogens are *Aspergillus fumigatus* (Cortes *et al*, 2005; Souza and Degernes, 2005; Tell, 2005; Arne and Lee, 2019) [45, 200, 209,] and Aspergillus flavus (Kuldeep *et al*, 2013; Musa *et al*, 2014; Cheng *et al*, 2020; Ameji *et al*, 2020) [132, 41, 11]. Other species involved in a single or mixed infections in poultry includes A. Niger, A. glaucus, A. nidulans (J. Barton *et al*, 1992; Joseph, 2000) [94, 221]. Smaller sized spores of A. fumigatus are likely to be trapped completely in the nasal cavity, some able to reach trachea and lung, this is the most implicated reason associated with aspergillosis (Richard and Thurston, 1983) [175]. A new strain, Aspergillus sydowii (Romina *et al*, 2019) [178] with disease producing capability was isolated often as contaminants of papers, nuts, beans and straws. Mixed infections are possible (Perelman and Kuttin, 1992) [150]. Some influencing factors that contribute to aspergillosis are (Beernaert *et al*, 2010) [27]:

Anatomical and physiological characteristics of the bird's respiratory tract
Exposer to moldy organic matters like old feed, damp litter and compost piles favor Aspergillus growth. Mash feed can be contaminated with high number of molds (Akan *et al*, 2002; Nesic *et al*, 2005; Skrinjar *et al*, 2009; Kapetanov *et al*, 2013) [9, 135, 197,]. The biodiversity of litter microflora depends on material choice, litter aging and handling techniques (Dennis and Gee, 1973) [53]. Inhalation of large number of spores over a short period of time or chronic exposure to low level of spores remain the principal risk factor of disease development (Barakat *et al*, 2012; Queiroz *et al*, 2013) [20, 158]. If the spores are localized in the lungs, the fungi may be disseminated into other parts of the body, often leading to death (Powell *et al*,1994) [154].

Poor ventilation: Stuffy air and ammonia promote spores build up in the barn. In aspergillosis free farms, the concentration of aspergillus species in air inside poultry house (Sauter *et al*, 1981; De Bey *et al*, 1995; Gigli *et al*, 2005; Nieguitsila *et al*, 2007, 2011; Nichita *et al*, 2010) [189, 52, 75, 138, 136] varied from 10-10^4 CFU per cubic meter. These fluctuations may be related to seasons and husbandry practices (Fulleringer *et al*, 2006) [73]. Concentration of aspergillus species in the air and litter decreases when windows were open (Pinello *et al*, 1977) [152].
Stressors like extreme cold, overcrowding, molting, poor nutrition, vitamin deficiencies (McMillan and Petrak, 1989) [127], over use of antibiotics and corticosteroids (Oglesbee, 1997; Verstappen & Dorrestein, 2005) [142, 222] and irritant disinfectants compromise the host immune system. Shi Bao *et al* (2014) [190] and Nemanja *et al* (2019) [134] reported immunosuppressive effect of dexamethasone by decreasing the chemotaxis and phagocytic activity of lymphocytes and macrophages. The inhaled spore may not cause disease, and remain dormant in the bird's air sacs and lungs until a stressful event triggers. Chickens that have undergone heat stress have reduced neutrophil activity, making them more susceptible. Poultry coinfected with Colibacillosis and Pasteurellosis more likely develop fungal disease.

Weather and season: Most of the fungal diseases occur sporadically with a seasonal variation (Mandal *et al*, 2004) [123]. Warm, damp and humid weather are ideal for prolific spore production (Vaezi *et al*, 2018) [219]. The global warming and high humidity aggravate various diseases (Kapetanov *et al*, 2015) [103]. The percentage of positive isolates in lung and environment samples was more in summer than winter season. Sultana *et al* (2014) [201] stated the presence of aspergillosis all through the year but highest in rainy season followed by summer least in winter. Thermal discomfort, starvation and migration (Young *et al*, 1998) [225] pamper the disease. Incidence of aspergillosis was more common in winter when indoor gas levels tend to be highest leading to bad ventilation, increased humidity and greater accumulation of organic matter in the litter (Zafra *et al*, 2008; Fang and Latge, 2018) [226, 62]. Predominance of infection in closed housing during summer and presence of the fungi in the litter material during autumn make the eradication difficult (Solima *et al*, 2012) [199]. The distribution of the aspergillosis outbreaks is influenced by geographic location and seasonal variations, more frequent in areas with high temperature and rainfalls (Karwowska, 2005; Sajid *et al*, 2006) [104, 181]. This seasonality may result from how climatic conditions and fungal ecology interplay with the bird's intrinsic factors (Chate and Bhivgade, 2010) [39].
Dusty litter or feed carrying spores transmit infection in poultry barns. In areas that are heated with brooders, in damp spots near drinking system, temperature and humidity rise (Ruediger *et al*, 2020), conidial proliferation becomes very high to infect young chicks (Poultry Affairs, 2022) [153]. Aspergillosis can also be produced by inhalation of spores from contaminated feed and poultry house litter (Thomas *et al*.). Large number of spores were found in wet litter and get aerosolized when the litter dries. Extremely dry air and dust, dry out respiratory mucosa causing the birds susceptible to the disease (Kristensen and Wathes, 2000; Munir *et al*, 2017) [109, 131]. On parent stock level, wet litter or shavings are sources of aspergillosis. The eggshells become infected on litter and transfer this infection to hatchery. During incubation, the ventilation system becomes infected and on hatching, the day-old chicks will inhale the spore and develop pneumonia. Other sources in the hatchery includes wet carton. There is no true vertical transmission, but the eggshell can be the source of lateral transmission (Hybro, 2005) [89].

Age: The prevalence of aspergillosis in broiler is significantly influenced by age of the birds (Gull *et al*, 2024) [79]. Newly hatched chicks, turkey poults and ducklings are more susceptible. Higher body temperature in poultry allows quick

fungal growth. Sultana *et al* (2014) [201] reported higher incidence of aspergillosis in 6-11 days of age and lowest in 0-5 days old chicks (Islam *et al*, 2003; Uddin *et al*, 2010) [90, 215]. Increased susceptibility of young chicks to aspergillosis might be due to immature phagocytes or environment factors (Bennett, 1988) [26].

Chicken raised on saw dust than rice husk as bedding has increased incidence (Sultana S *et al*, 2014; Sajid *et al*, 2006) [201, 181] because of higher moisture content in saw dust. Wood shavings when stirred get dry out and spores become aerosolized and promote new cases (Rao *et al*, 1982; Dyar *et al*, 1984) [164, 56].

Eggs in the incubator are common places for the fungus growth. The other chicks in the hatcher can be infected and equipment contaminated if the contaminated eggs burst out releasing high levels of spores (Bauck, 1994) [22]. Aspergillus is the most recovered fungal genus from dead in shell embryos (Abd. El-Aziz, 2015) [3].

Nutritional deficiencies (Ranck and Miles, 2001) [163] weaken immune system, and the malnourished chickens become susceptible to fungal infections. Lack of vitamin A and D, zinc, copper and selenium impair immune function in chickens. Lead poisoning is another factor (Samour, 2000) [186]. Feeding a balanced diet is crucial for maintaining optimum flock health and robust immunity (The poultry Pro, 2025) [211].

Certain infectious diseases such as infectious bronchitis, laryngotracheitis, Newcastle disease and fowl pox (Jacobson, 1980) [91] and subclinical infectious bursal disease aggravate aspergillosis (Okoye *et al*, 1991) [144].

Prevalence

Among the diseases of poultry, aspergillosis constitute 4-7% (Talha *et al*, 2001; Badruzzaman *et al*, 2015) [205, 19]. In broilers, prevalence of aspergillosis ranges from 4-8%, while 4% in layer chicks. The warm environment of summer and the highest indoor gas during winter (Girma *et al*, 2016) [77] favors the disease development. The occurrence and pathogenicity of A. fumigatus varied according to the lineage: in a challenged study, no clinical signs and mortality in layer chickens, more than 50% mortality occurred in broilers (Simon Thierry *et al*, 2013) [193]. Koenen *et al* (2002) [106] assessed the immunological differences between layer and broiler chickens. Layer chicken, showed two types of immune responses – antigen specific and cellular response against the only antigen specific immunity in broilers whose part of the immune response dysfunction due to genetic selection against body weight and feed conversion (Faublee, 1975) [64]. Birds are more susceptible to aspergillosis due to their anatomical and physiological makeup: no diaphragm, no epiglottis, poor vascularization, limited mucociliary function of the air sacs and heterophils replacing neutrophils (Tell *et al*, 2005; Corbanie, 2007; Tell *et al*, 2019; Melo *et al*, 2020) [209, 44, 210, 128].

Susceptibility to Chemical and Physical Agents

Some species of Aspergillus are quite resistant and can grow in sanitizing fluids (Rippon, 1982) [177]. Commercial disinfectants with Enilconazole have been used to control spores in poultry houses and surrounding (Redmann *et al*, 1989) [167]. Cinnamaldehyde, an essential oil from cinnamon inhibits Aspergillus growth (Mahmoud, 1994) [121]. Formalin fumigation is effective and cost efficient.

Pathogenesis

The fungus secretes enzymes like proteases and toxic secondary metabolites (Latge, 2001; Tekaia and Latge, 2005) [115, 208]. Clinical signs and torticollis detected in turkey poults without central nervous system lesions indicated toxin involvement (Richard *et al*, 1981, 1983) [170, 175]. A. fumigatus produces (Rementeria *et al*, 2005) [169] gliotoxin an immunosuppressive cyclic peptide characterized by the presence of a disulfide bridge that regulate biological activity and toxicity (Gardiner *et al*, 2005) [74]. In poultry feedstuffs (Pena *et al*, 2010) [149] gliotoxin concentrations exceeding 20 µg/g, whereas 70 µg/g in tissues with air sac inflammation could be detected (Richard *et al*, 1996) [172]. Gliotoxin have cytocidal properties in chickens and turkeys causing mortality and torticollis after aerosol exposure (Richard, *et al*, 1996) [172]. Gliotoxin allow A. fumigatus to invade the epithelial cells of the lungs, and together with aflatoxin, suppresses the immune function of the host (J. Shankar, 2013) [97]. Galactosaminogalactan, another polysaccharide metabolite of *Aspergillus fumigatus* is also an immunosuppressive toxin (Fontaine *et al*, 2011) [69]. Extraction of gliotoxin from necrotic lesion of a cow udder infected with A. fumigatus suggest the biosynthesis and involvement of gliotoxin (Bauer *et al*, 1989; Beaver and Waring, 1994) [23, 24] in inducing necrosis, but mechanism is not fully elucidated (Hurne *et al*, 2002) [88]. The increased intracellular calcium flux, mediated by gliotoxin has been linked. Gliotoxin interaction with thiol residue on the plasma membrane of the calcium channel cause oxidative damage and develop necrosis(Veronica *et al*, 2017) [223]. Aspergillosis is caused by inhalation of huge small, hydrophobic fungal spores into the respiratory tract (Oglesbee, 1977) that are trapped in nares, trachea and enter into primary bronchi and delivers to the posterior thoracic and abdominal air sacs which further to epithelial surfaces in the lungs (Nardoni. *et al*, 2006) [133] and there, germinate granulomas. These spores are engulfed by alveolar macrophages of respiratory tract and lung (Maina, 2002) [120] to find their way into the blood circulation and lymphatic stream, spread further into multiple organs (Dahlhausen *et al*, 2004) [50] like brain, pericardium, bone marrow, kidney and other soft tissues. A. fumigatus conidia resist killing by alveolar macrophages (Van *et al*, 1996) [218]. Tissue invasion creates an inflammatory reaction with heterophils, lymphocytes and monocytes to produce lesions (Kunkle and Rimler, 1996) [111] their severity depends on chronicity of infection, organs affected and the number of spores inhaled (Perez and Carrasco, 2000) [151]. The aspergilli migrate from lung to other parts of birds such as the liver, oviduct and ovary leading to disease syndromes characterized by acute to chronic infection, thereby suppressing growth and cessation of egg laying (Jordan *et al*, 2002) [100]. Besides inhalation and hematogenous spread to tissues, angio-invasive hyphae are also possible in disseminated mycosis (Dahlhausen *et al*, 2004) [50].

Aspergillus penetrate the eggshell (Amal *et al*, 2015; Brajen, 2025: Arrow) [10], contaminate and infect the embryo (Hamet

et al, 1991; Chauhan and Roy, 2016) [80, 40], eggs broken during incubation spread the disease in hatchery thus, the death of the embryos and chicks hatched with pathological lesions (Gray *et al*, 2021) [78]. In Ovo vaccination may present an increased risk of contamination. Two types of tissue reactions have been recognized: 1. Granulomatous or deep nodular form with neither exudative inflammation nor vascular lesions in the neighboring tissues. Reactions develop both in aerated and non-aerated organs like lungs and air sacs (Nardoni, 2006; Femenia *et al*, 2007) [133, 66]. 2. Infiltrative or superficial diffuse form – the fungus frequently invades blood vessels and form aggregates of hyphae containing large numbers of conidia (Cacciuttolo *et al*, 2009) [36] that often-impending pulmonary blood flow leading to right ventricular dilatation and ascites. Another factor for mortality from aspergillosis includes exudates lodged in the trachea and syrinx producing respiratory embarrassment in birds (Pattisson *et al*, 2009) [147].

Clinical Signs

Birds placed in environments contaminated with aerosolized conidia develop symptoms after a short exposure. Acute aspergillosis has 2-5 days of incubation period in chicken and turkeys (Richard *et al*, 1984; Melloul *et al*, 2014) [176,129].

Birds of different age groups are susceptible (Charlton *et al*, 2008) [38] involving many body systems. Manifestations of the disease depend on which organs involved and the infection either localized or disseminated. Extensive involvement of the respiratory tract occurs before symptoms are apparent. However, aspergillosis is generally reflected as a disease of respiratory tract (Singh and Malhotra, 1980; Sajid *et al*, 2006) [195,181] and occasionally of the central nervous system (Atlaman, 1997) [17]. Clinical signs depend on the infective dose and distribution of spores, presence of pre-existing diseases and the immune response of the host (Dahlhausen *et al*, 2004) [50]. The main clinical signs of the disease in broilers (Eassa *et al*, 2017) [57] include a third day onset of labored breathing and neurologic disorders as incoordination, torticollis and paralysis. Three forms of aspergillosis based on local involvement to systemic dissemination (Leishangthem *et al*, 2015; Fagbohun *et al*, 2020) [116, 61] are as follows:

Acute aspergillosis occurs by inhaling large number of spores. Within the first 3-5 days newly hatched chicks infected in the hatchery show very rapid and difficult breathing with gasping due to obstruction of the air passage (Aguilar and Redig, 1995) [6]. When these signs are complicated by other respiratory diseases - infectious bronchitis, infectious laryngotracheitis, there is gurgling or rattling noises with scratch sound during expiration (Okwara, 2016) [145], but in aspergillosis dyspnea there is no respiratory noise (Vegad, 2015) [220]. Respiratory distress led to cyanosis (Richard, 1997) [173]. Young chicks and turkey poults that inhaled large number of spores during hatching or when placed on bedding contaminated with spores, potentially exhibit high morbidity and mortality often without showing clinical signs. Symptoms of anorexia represent 50%, dyspnea 48%, lethargy 40%, gasping 29% (In Video: Gasping and dyspnea)
and torticollis 3% of signs in birds (Nururrozi, 2020) [139]. These findings are in consistent with Singh *et al* (2009) [196], Beernaert *et al* (2010) and Melo *et al* (2020) [128] that clinical signs of acute aspergillosis manifest in a few days to 2 weeks of age. Nasal discharge occurs in acute aspergillosis (Sajid *et al*, 2006; Arne *et al*, 2011) [14, 181]. Nasal aspergillosis causes exudative rhinitis and nasal sinusitis possibly leading to malformation of the nostrils and beak (Bauck *et al*, 1994) [22]. Mycotic keratitis can cause blepharospasm, photophobia, periorbital swelling, turbid discharge, swollen eyelids, cloudy cornea and cheesy exudates within the conjunctival sac (Bechman *et al*, 1994; Hoppes *et al*, 2000) [87]. Otomycosis invasive infection due to aspergillus species have been reported by Henry *et al* (2000) [85]. Per acute form in chicks may bring sudden death.

Chronic form is associated with immunosuppression (Vanderheyden, 1993) [217] and common in older birds (Hassanain *et al*, 2018; Kannoju *et al*, 2021) [83, 102]. Clinical signs include anorexia, increased thirst, diarrhea, pyrexia, lethargy, ruffled feathers, dehydration, stunting and gasping. Ocular changes include ophthalmitis, photophobia, periorbital swelling with cheesy exudates in the conjunctival sacs (Backman *et al*, 1994; Hoppes *et al*, 2000) [25, 87] and granulomatous dermatitis. Cloudiness of eye and blindness are not rare. Chronic form is sporadic, has lower mortality (Shoukat *et al*, 2018) [192] due to gradual loss of lung function and ascites (Vegad, 2015) [220]. In breeders, Akan *et al* (2002) [9] reported aspergillosis as chronic infection with brain lesions confining to cerebellum or cerebellar meninges. Chronicity is the result of long-term malnutrition, pre-existing illness, over exposure to antibiotics, steroids and stress (Bauck L, 1994; Rahul *et al*, 2024) [22]. The fungus may invade skin or surgical wounds causing necrotic granulomatous dermatitis or systemic aspergillosis.

CNS granuloma formation in the brain, abscesses in the cerebellum were also noticed (Pattison *et al*, 2009) [147]. Neurological signs include paralysis, ataxia, tremor, torticollis, lameness, lateral recumbency, seizures, convulsions (Dyar *et al*, 1984; Jensen *et al*, 1997; Akan *et al*, 2002; Throne *et al*, 2003; Bongomin *et al*, 2020) [56, 96, 9, 213, 32]. Torticollis and loss of equilibrium are the significant signs in experimental and natural aspergillosis in poultry (Kunkle, 2003) [112].

In farms, mortality may increase suddenly, peak over a few days and return to the initial state in a biphasic manner. In chicks contaminated during hatching, aspergillosis is highly fatal in the first 10 days of life causing 50-80% mortality (Kuldeep *et al*, 2013; Eassa, 2017; Wondimu *et al*, 2019) [57, 224] within 5-14 days of symptoms development. Unilateral drooping of the wing due to infection of the thoracic and clavicular air sac or proximal humerus bone and repeated vomiting because of lesions in the anterior air sac also often accompanied (Forbes, 1991, 1992) [70, 71].

Gross Lesions

The primary location of lesions are the lungs, air sacs and other organs – esophagus, proventriculus, gizzard, small intestine, liver, kidney, spleen, skin, trachea and peritoneum (Ahamad *et al*, 2018) [7]. Extensive involvement of the respiratory tract can be seen prior to the development of clinical signs. Typical lesions consist of white to yellowish granulomas (Arrow) of millet seed to a pea size on the serosa

and parenchyma of multiple organs (Reece *et al*, 1986; Perelman and Kuttin, 1992; Singh H *et al*, 1994; Ashok *et al*, 2014, Musa *et al*, 2014) [168, 150, 194, 132] in broilers and matured broiler breeders. These granulomas contain the organisms which could be rarely penetrated by therapeutic agents (Krishnan, 2005) [108].

The colonization of spores in the lungs and air sacs promote proliferation of the organism, development of granulomatous lesions in individual organs and tissues through evocation of the host defense mechanism (Cacciuttolo e *et al*, 2009) [36]. Pulmonary lesions are characterized by multiple hard creams to yellow circumscribe plaques a few mm to centimeter in diameter throughout the lung surface and inside, scattered on ventral surface of sternum and air passages (Beytut, 2007; Singh *et al*, 2009; Ruediger *et al*, 2020) [29, 196,]. The lungs, liver and spleen from aspergillus infected geese and emu appeared darker red than normal pink appearance (McDougle and Vaught, 1968; Sunitha *et al*, 2010; Emin Karakurt, 2021) [126, 60]. Aspergillosis has been the primary cause of death within 2-7 weeks of age ostriches (Saravia-Gomez, 1978) [188]. Dyspnea and gasping due to hyphae growth led to necrosis and inflammation in the air sacs and lungs (Ahamad *et al*, 2018) [7]. Necrotic cells in the respiratory tract cause hypoxia and birds increased respiratory frequency in attempt to compensate body oxygen, while the presence of plaques in the respiratory tract inhibit oxygen exchange of the lung and disrupt oxygen circulation (Chu *et al*, 2017; Eassa *et al*, 2017) [42, 57]. Aspergillus Niger produce oxalic acid which combine with calcium ions form insoluble calcium oxalate (Pradeep and Uma, 2009) [156] on being deposited in the lungs - disrupt its functioning.

Abscesses in the cerebellum or cerebrum of 3-5 mm could be seen as circumscribed white to greyish areas in broiler breeders, turkeys and layers (Kureljusic *et al*, 2012; Gino L, 2023) [113,]. Ocular changes, such as mycotic keratitis with plaques can also be observed (Rafael Lera). Ascites in chickens has been a frequent after effect of hypoxia from aspergillosis leading to pulmonary hypertension and heart failure (Julian, 1993; Zafra *et al*, 2008) [101, 226].

Diagnosis

Diagnosis based on clinical signs and postmortem lesions are incomplete, may be confused with other infections. Therefore, confirmation requires combination of evidence from history, clinical signs, postmortem, hematology, biochemistry, serology, radiographic changes, or endoscopy and fungal cultures (Jones and Orosz, 2000) [99]. Field cases are diagnosed through postmortem findings of white to yellowish caseous nodules in the lungs or air sacs (Olias *et al*, 2010, 2011) [140]. History of the birds reveal stressful events, environmental factors associated with immune suppression, drug therapy and chronic debilitating changes (Hofle, *et al*, 2001) [86].

Definitive diagnosis is based on the isolation of Aspergillus species by culture and detection of the organism during histological examination (Charlton *et al*, 2008; Beernaert *et al*, 2010; Kim *et al*, 2011) [38, 105] on Saburou's glucose agar or antibiotics, incubated at 37°C for 24 hours show characteristic conidial head and colony (Fowler, 1993; Aguilar and Redig, 1995; Dahlhausen *et al*, 2004) [72, 6, 50]. However, negative culture cannot confirm the absence of aspergillus infection (Redig, 1994) [34]. Species can be identified by colony morphology and microscopic characterization of spores (Latge, 1999) [114]. Other serological tests that confirm aspergillosis infections are:

Enzyme-Linked immunosorbent assay (ELISA): Brown and Redig, 1994; Redig *et al*,1997; and Arca-Ruibal *et al*,2006 [34, 166, 13].

Immunohistochemistry: Carrasco *et al*, 1993; Jensen *et al*, 1997; Beytut *et al*, 2004; Beytut *et al*, 2007 [37, 96, 28, 29].

Galactomannan assay and Plasma protein electrophoresis: Cray *et al*, 2005, 2009a.

Lateral and ventrodorsally radiograph (Jones and Orosz, 2000) [99], Endoscopy (Taylor, 1993; Oglesbee, 1997) [207, 142] detect abdominal air sac infection.

Recent molecular techniques like RT-PCR, Internal Transcribed Spacer (ITS) gene-based PCR for broiler breeders, dead in shell and hatched chicks (Radwan *et al* 2019) [160], environment and hospital patients (Hasab *et al*, 2019) [82] are now available to detect and quantify Aspergillus burden in lungs (Melloul *et al*, 2014; Tartor & Hassan, 2017) [129, 206]. The 26S gene of *Aspergillus fumigatus* was identified by amplification of 401 bp fragment in PCR test.

Treatment

The economic loss from aspergillosis is huge as treatments are ineffective and challenging in poultry (John, 2002; Arne, 2011; Ameji *et al*, 2020) [98, 14] because of: (a). Spontaneous recovery can occur if exposure to fungus is avoided, (b). Limited knowledge on the pharmacokinetics of antifungal agents in different bird species, (c). The granulomatous inflammations create barrier for the drugs reaching the targeted fungus, and (d). Presence of concurrent infections, immunosuppression and the late stage at which birds are presented make the treatment challenging (McMillan and Petrak, 1989; Flammer, 1993 and Orosz & Frazier, 1995) [127, 67, 146]. Effective treatment involves a comprehensive approach, including antifungal medication, symptomatic treatment and supportive therapy (Krautwald *et al*, 2015; Jenks *et al*, 2018) [107, 95,]. Best results achieved on combination of two antifungal drugs, therefore, a judicious consideration for less toxic alternative approach is advised (Tell *et al*, 2019) [210]. When the granulomatous lesion dried, topical treatment in conjunction with systemic therapy gave faster recovery. Since drug resistance has been increasingly reported in human medicine (Snelders *et al*, 2008) [198], standardized susceptibility testing is also becoming mandatory in avian medicine.

Itraconazole and posaconazole 10 mg/kg orally every 12 hours for 10 days is the drug of choice (Sultana *et al*, 2015, Bunskoek *et al*, 2017) [202, 35]. Amphotericin B, 1.5 mg/kg intravenously once daily for 3 days (Hasan *et al*, 2011) [81]. Clotrimazole as nasal flush or nebulization 30 minutes. Ketoconazole, 30 mg/kg in conjunction with Itraconazole is given for 21 days. Voriconazole, 10 mg/kg 12 hourlies through oral route is more potent but absorption and bioavailability depends on gastric pH (Lestrade *et al*, 2016)

[117]. Fluconazole 15 mg/kg 8-12 hours orally. Nystatin 50000 IU per kg once a week in drinking water significantly reduced chick mortality (Oladele *et al*, 2022) [143].

Enilconazole may be sprayed to decontaminate indoor volume. Spraying of thiabendazole (Fate *et al*, 1987) [63], nystatin and copper sulphate decrease fungal contamination of litter materials.

In outbreaks, a 1:2000 solution of copper sulfate in water prevents the spread or 600 gm Copper Sulfate per ton of feed for 5-7 days (Dyar *et al*, 1984) [56]. A fresh solution from 50 gm Copper Sulphate dissolve in 250 ml vinegar + 700 ml of water: 2 ml of this solution mixed with Griseofulvin @ 2 mg/liter water for 5 days reduced mortality). Tetracycline 200 mg a liter of water treat aspergillosis in chicks (Babras and Radhakrishnan, 1967) [18]. Chlorine bleach @ 5ppm in drinking water is effective (Janmaat and Morton, 2010) [92].

Anti-mycotoxigenic, antifungal and antioxidant compounds from wild stevia restrain the growth of Aspergillus, Penicillium and Fusarium (Cowan, 1999; Dikhoba *et al*, 2019) [46, 54]. Essential oils inhibit growth of toxigenic fungi and their mycotoxins (da Cruz *et al*, 2013; Prakash *et al*, 2015; Powers *et al*, 2019) [49, 157, 155].

Broiler breeders experiencing chronic aspergillosis and broilers from these parents had symptoms of gasping, lameness, mortality from hatch, suboptimal growth among the survivors responded positively to essential oils Fytera Perform® at 150 gm + organic acids (Salacid GG®) 4 kg per ton feed after 10 days. Continued dietary inclusion eliminated recurrence and concurrent bacterial infections in parent flocks. Essential oils could successfully treat aspergillosis in poultry and decrease infectious agents in the environment (Ebani *et al*, 2018) [58]. Cinnamon oil has the highest synergy with organic acids and potentially effective natural method of controlling post-harvest aflatoxin contamination in poultry feed. Essential oils have antioxidant, antimicrobial, analgesic, antipyretic, antiulcer, anticonvulsant and anticarcinogenic properties (Uma *et al*, 2017) [216] thereby reducing pathogenic bacterial growth, improving gut health, weight gain and meat quality in broilers (Zhai *et al*, 2018; Ding *et al*, 2022) [228, 55]. Several organic acids inhibit the growth of aspergillus strains and minimize mycotoxin production, are effective against Salmonella and E. coli infections (El-Saadony *et al*, 2022) [59].

Prevention

Among the fungal diseases of poultry, Aspergillosis, Candidiasis and Mycotoxicosis have impact on poultry production (Mersha and Dawit, 2017) [124]. These pathogens mainly target the respiratory and nervous organs of poultry (Shiva Chandra *et al*, 2004) [191].

Prevention is the best way of control (Girma *et al*, 2016) [77] by ensuring adequate ventilation, cleaning and disinfection (Beckman *et al*, 1994) [25], reducing exposure to the fungus and associated risk factors, and early intervention to mitigate the outbreaks. Keeping the hatchery free of aspergillus prevent outbreak in chicks. Hatching eggs are collected and stored to reduce sweating, exposure to spore-laden dust, hatching equipment and air ducts be clean and disinfected. Contaminated premise is fumigated. Trace and eliminate the source of spores usually from nest boxes, litter, cardboard boxes, wooden walls or ceilings. In the setters and hatchers, either formalin or enilconazole (Clinafarm®) may be fogged to decontaminate indoor volumes without adverse effect on embryo development (Hybro, 2005) [89]. Removal of dust from poultry houses, improving ventilation and airflow reduces humidity which decrease fungal growth by 75% (Redmann and Schildger, 1989) [167]. An 0.5 cubic meters per bird per minute of fresh air exchange ensure adequate ventilation (Poultry Pro,2025) [225].

Providing a balanced diet rich in essential amino acids, minerals, vitamins and immunomodulators boost their immunity. Probiotics as supplements promote gut health and supplementation of arginine in diets enhance immune function reducing the susceptibility to aspergillosis (Danish *et al*, 2023) [51]. Improving feed management practices is crucial for preventing this disease (Agbato *et al*, 2024) [5].

Aspergillus fumigatus is the most dominant fungal pathogen in poultry (Radwan *et al*, 2019) [160] causing great economic losses. Poultry workers with weak immune system must use face mask to avoid inhalation of infective spores. According to P. Arne and Lee (2019), reducing spore load by sanitation of hatchery and rearing premises, removal of moldy feed and bedding, and good air quality reduce fungal disease. Host innate immunity is essential for defending against the pathogens in early stage of infection. When treating other illness, duration of antibiotic usage and doses of corticosteroids must be weighed against mycotic infections (Oglesbee, 1997) [142].

Conclusion

Aspergillosis continue to remain life threatening to immunocompromised poultry. An increased action to identify and isolate new bioactive compounds to treat the disease is imperative. Many plant extracts and their constituents have shown therapeutic alternatives, but still remain challenging. Interactions among different plant derived compounds should be evaluated to maximize the antifungal efficacy while minimizing toxicity.

Mycotoxins are the leading cause of immunosuppression in birds, making them prone to bacterial and viral infections. Formulating strategies with focus on diagnosis, prevention and control of fungal pathogens will find a solution. Provide no environment for the fungal spore to thrive in farm and hatchery will overcome the disease.

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