AMINO ACIDS

Effects of Arginine on ascites syndrome in broiler chickens

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Abstract

Ascites syndrome is a complex problem in poultry production. The etiology of ascites is multifactorial, including environmental causes such as high altitude, low temperature and incubator environment. Genetic selection, growth rate and oxygen requirements also influences ascites. All of these factors causes ascites by inducing hypoxia in bird. Arginine is an indispensable amino acid for poultry and plays a crucial role in metabolic pathways associated with growth and immune. Results from numerous studies have shown that arginine reduces ascites-related mortality and attenuates adverse effects. Arginine supplementation decreased the ascites mortality and improved the intestinal morphology and performance in broiler chickens. Moreover, in ovo feeding of arginine increased serum nitric oxide concentration and decreased ascites mortality. This review provides insights into the optimal supplementation of arginine above NRC recommendation to improve growth, imm unity, intestinal health and antioxidant abilities.

How does Ascites syndrome develop

Ascites syndrome was first noticed as a problem for commercial poultry producers and has become a significant problem for producers everywhere[1].
The most apparent sign of ascites is an accumulation of fluid in the abdominal cavity of the bird, which has been described as waterbelly[1]. Pulmonary hypertension syndrome (PHS) and cardiac dysfunction are the most important features of ascites[2]. Genetic selection strategies of broiler have focused on higher body weight gain and feed efficiency increase the susceptibility to to PHS[3].
The etiology of ascites syndrome appears to be multifactorial. In 1955, Smith et al., first reported that growth is decreased and with the appearance of ascites at high altitude in domestic birds[4]. Low temperatures are a critical factor causing PHS.
Cold temperatures increase ascites by increasing both metabolic oxygen requirements and by increasing pulmonary hypertension[5]. Huchzermeyer et al. (1989) reported that a drop in environmental temperature induced 32.7% increase in PHS and are subjected to hypoxia resulting in an overall reduction in growth rate and compromised pulmonary vasculature development[6].
Diets high in protein increased the incidence of ascites[7]. When PHS happened, a series of pathophysiological changes including endothelial dysfunction and vascular remodeling, as a result of death caused by right ventricular failure[8]. Unfortunately, the most effective methods for reducing ascites is feed restriction which will reduced the growth rate of the animal.

Arginine and its role in alleviating ascites

In birds, arginine is an indispensable amino acid due to the unavailability of two separate enzymes in the urea cycle in the kidney[9].
Arg is a multipurpose amino acid and essential for the biosynthesis of polyamines, ornithine, creatine, proline, citrulline, nitric oxide and enhances secretion of insulin, growth hormone and insulin-like growth hormone and insulin-like growth factor 1 in animal[10].

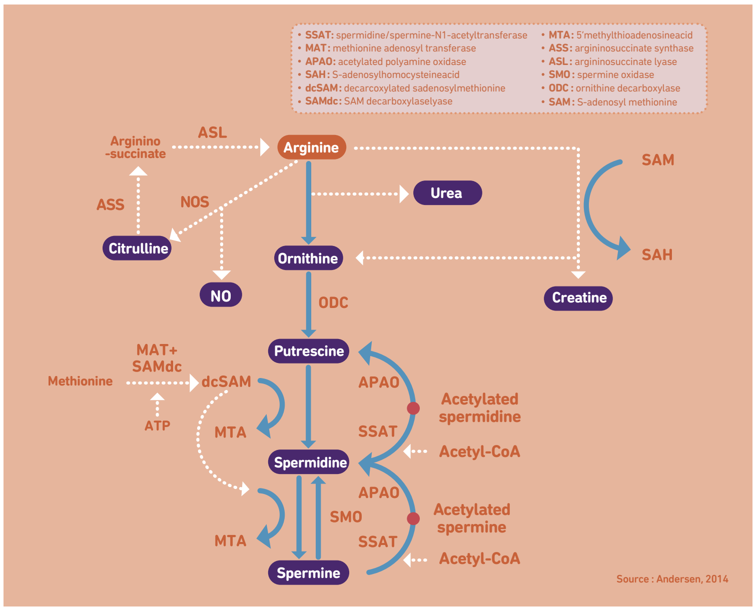

Figure 1. Arginine and polyamine synthesis

Several studies have shown that Arg play an important role in alleviate PHS. Abdulkarimi et al. (2019) reported that Arg supplementation improved the intestinal morphology and performance and decreased the ascites mortality in broiler chickens with cold induced ascites[11]. The Arg requirements are increased to maximized growth in broiler chickens reared at high altitude[12]. Saki et al. (2013) reported in ovo and in-feed Arg supplementation could improve broiler hypertensive response and reduce mortality from ascites (18.8% vs 43.8% and 28.1% vs 43.8%)[13]. Moreover, in ovo feeding of arginine increased serum nitric oxide concentration but decreased ascites mortality induced by subnormal eggshell temperature[14].

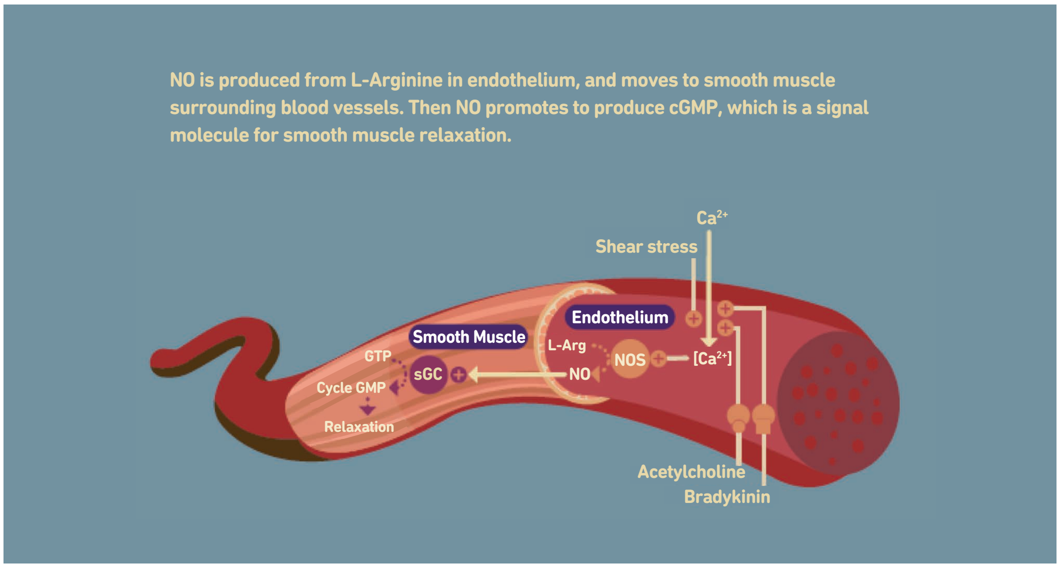

Figure 2. Functions of nitric oxide

Arg serves as a substrate for the synthesis of nitric oxide (NO) and potentiates vasodilation. Ahmadipour et al. (2018) reported that serum NO levels increased as a consequence of ARG supplementation, indicating that Arg is a precursor of NO synthesis in broiler chicken[15]. In mammals, nitric oxide, which is produced during the metabolism of Arg, acts as a potent pulmonary vasodilator[16]. In haled nitric oxide is effective pulmonary vasodilator in an in-vivo porcine model[17]. In broilers, administration of NO synthesis inhibitor markedly enhanced the amplitude and duration of the lipopolysaccharide (LPS) induced pulmonary hypertension, indicating NO attenuates the pulmonary vascular response to LPS8.

Widemanet al. (2005) showed that LPS-initiated inflammatory cascade that are correlated with the magnitude of the evoked pulmonary hypertensive response[18]. In human, augmented expression of cytokines (IL-18 and CXCL10) may perpetuate an inflammatory condition that eventually contributes to the vascular obstruction characteristic of pulmonary arterial hypertension (PAH)[19]. Thus, an improved immune response is an effective way against PHS. Arg, often found in immunonutrition regimens, is an important modulator of immune system activation[20]. Supplementation of Arg increased heterophil oxidative burst and IgG level to an Eimeria challenge of broiler chicken, suggesting Arg may increase humoral and innate immune response[21].

Moreover, Arg supplementation increase CD4+ and CD8+cells and attenuated the ileal expression of IL1 β and TLR4, indicating the effects of Arg on cell-surface antigens for T cells, as well as cytokine and chemokine responses in yellow-feathered chickens[22].

As we known, the intestine serves not only as a digestive absorptive organ, it is also one of the largest immune organs.[23]. Arg plays an important role in development of small intestinal mucosa and growth is correlated with polyamines synthesis[24]. Supplementation of 10 g/kg Arg, has reduced the susceptibility to PHS and improved gut function through increase in villus height of small intestine in broiler reared at high altitude[25]. Additionally, Arg is substrate for the formation of glutamate, which is an immunoregulator of intestinal immune system in broiler chickens[26].

Moreover, the onset of PHS was associated with the production of oxygen species (ROS) and endothelial cell damage[7]. Nezhad et al. (2011) reported that MDA content in plasma and liver was significantly higher when used cold temperature, suggesting PHS may be initiated by increased production of ROS[27]. In yellow-feathered chickens, Arg increased intestinal antioxidative capacity through increasing the activities of glutathione peroxidase and total antioxidative capacity in jejunum and ileum[22].

Dietary supplementation of 1.36% digestible Arg increased the total antioxidant capacity levels and decreased the MDA concentration in the serum and egg yolk, indicating Arg improved antioxidant capacity of broiler breeder[28]. According to these research, the supplementation of Arg is an effective pathway to improve the performance and consequently decreased the ascites in broiler chicken.

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