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Dietary riboflavin supplementation improve the growth performance and antioxidant status of starter white Pekin ducks fed a corn–soybean meal diets

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ABSTRACT

An experiment was conducted to evaluate the effects of dietary riboflavin on growth performance and antioxidant status of white Pekin ducks from hatch to 21 d of age. Different levels crystalline riboflavin (0, 0.7, 1.4, 2.1, 2.8, 3.5, and 7 mg/kg) were supplemented to corn–soybean meal basal diet to produce 7 dietary treatments with different analyzed total riboflavin levels (1.69, 2.56, 3.37, 4.05, 4.73, 5.88, and 9.26 mg/kg). A total of 448 one-day-old male white Pekin ducks were randomly allotted to 7 dietary treatments with 8 replicate pens of 8 ducks per pen. All ducks were raised in an environmentally controlled duck house from hatch to 21 d of age. The mortality rate was 15.5% for ducks fed the diet with no supplementation of riboflavin compared with 0 to 1.6% for those fed the diets supplemented with riboflavin. As dietary riboflavin level increased from 1.69 to 3.37 mg/kg, the body weight at 7, 14, and 21 d of age, overall average daily gain (ADG), and average daily feed intake (ADFI) increased linearly and quadratically ($P<0.001$), and gain to feed ratio (G:F) increased quadratically ($P=0.003$), reaching a plateau at 3.37 mg riboflavin/kg for ADG and 4.05 mg riboflavin/kg for ADFI and G:F and no response with further increases. The similar responses to increasing dietary riboflavin were also detected in plasma free riboflavin (linear and quadratic, $P<0.001$), liver free riboflavin (linear and quadratic, $P<0.002$), and liver flavin mononucleotide (FMN; linear, $P=0.001$; quadratic, $P<0.001$) in ducks, which indicated that tissue riboflavin and FMN may be sensitive indicators for riboflavin status. On the other hand, riboflavin deficiency had negative effects on antioxidant capacity of ducks. Compared with ducks fed the riboflavin-supplemented diets, ducks fed the riboflavin-deficient basal diet had greatest plasma malonaldhyde (MDA; linear and quadratic, $P<0.005$) and liver MDA (linear, $P=0.06$; quadratic, $P=0.001$) and lower superoxide dismutase activity in plasma (linear, $P=0.027$; quadratic, $P<0.001$) or liver (linear, $P=0.008$; quadratic, $P=0.093$). Furthermore, these ill effects may cause liver damage to ducks because the ducks fed basal diet also had the greatest activity of alanine transaminase (linear and quadratic, $P<0.035$), aspartate transaminase (linear and quadratic, $P<0.001$), and lactate dehydrogenase (linear, $P<0.001$). According to broken-line regression analysis, the riboflavin requirement of white Pekin ducks from hatch to 21 d of age was estimated to be 3.01, 2.98, 2.79, 4.18, 3.63, and 3.59 mg riboflavin/kg for ADG, ADFI, G:F, plasma riboflavin, liver riboflavin, and liver FMN, respectively. In conclusion,

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corn–soybean meal-based diet is riboflavin-deficient for ducks and supplementation of riboflavin at such levels in these diets could enhance riboflavin and antioxidant status and growth performance of ducks.

1. Introduction

Riboflavin is a water-soluble vitamin and it is important for duck growth and health. Riboflavin deficiency could cause growth depression, leg paralysis, and high mortality in starter Pekin ducks (Tang et al., 2013). The NRC (1994) recommendation of riboflavin for white Pekin ducks is 4 mg/kg in either starter or growing period. Although this recommendation came from early literatures (Fritz et al., 1939; Hegsted and Perry, 1948), it was still sufficient for modern breeds of white Pekin ducks. This contention can be supported by Tang et al. (2013) who estimated that riboflavin requirements of modern strain of Pekin ducks from hatch to 21 d of age ranged from 3.31 to 3.91 mg/kg for males and from 3.27 to 3.84 mg/kg for female depending on the criteria. However, in the study of Tang et al. (2013), riboflavin requirements were based on the corn–corn gluten meal diets and these diets were not typical diets for ducks. In commercial Pekin duck production, corn and soybean meal are still the most predominant ingredients in duck diets, and corn–soybean meal diets are deficient in bioavailable riboflavin, which is 59% bioavailable relative to crystalline riboflavin for chicks (Chung and Baker, 1990). Therefore, it is necessary to conduct a follow-up study based on corn–soybean meal diets.

As a precursor of flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD), riboflavin is critical in a range of oxidation-reduction reactions. For instance, FAD is a coenzyme of glutathione reductase, which mediates glutathione recycling. Previous studies have demonstrated that riboflavin deficiency could lead to reduction in the activity of glutathione reductase (Bamji, 1969; Bamji and Sharada, 1972; Levin et al., 1990) and cellular reduced glutathione concentration (Camporeale et al., 2003; Manthey et al., 2005). Thus, riboflavin status may affect antioxidant capacity of animals. However, there is a lack of information on antioxidant status of ducks in response to dietary riboflavin. Therefore, the first objective of this study was to confirm the riboflavin requirement of ducks estimated in our previous study. Another objective of this study was to evaluate the effects of dietary riboflavin on antioxidant status of ducks.

2. Materials and methods

2.1. Animals and housing

All procedures of the present study were approved by the Animal Care and Use Committee of the Institute of Animal Sciences of Chinese Academy of Agricultural Sciences. The response of ducks to dietary riboflavin was determined during the starter period from hatch to 21 d of age. A total of 448 one-day-old male white Pekin ducks with an initial body weight of 59.1 g (Pekin Duck Breeding Center, Chinese Academy of Agricultural Sciences) were randomly allocated to 7 treatment groups with 8 replicate pens of 8 birds per pen. All ducks were raised in an environmentally controlled house with plastic-wire floors. The temperature was set at 28 °C from 1 to 3 d of age, 26 °C from 4 to 7 d of age, 25 °C from 8 to 14 d of age, and 22 to 20 °C from 15 to 21 d of age. Feed and water were given ad libitum, and 24 h constant lighting was provided.

2.2. Diet

A riboflavin-deficient basal diet was formulated (Table 1). The vitamin mixture was free of riboflavin. Experimental diets were formulated by supplementing the basal diet with 0, 0.7, 1.4, 2.1, 2.8, 3.5, and 7 mg crystalline riboflavin/kg. Except the riboflavin content of the basal experimental diet, all nutrients met the recommendations for starter ducks (meat-type ducks of China; Ministry of Agriculture of China, 2012). The crystalline riboflavin (purity, 99%) was obtained from a commercial source (Sigma Chemical Company, St. Louis, MO, USA). All experimental diets were prepared as a mash and then cold-pelleted at room temperature after mixed with water. The riboflavin content of all experimental diets was analyzed by high performance liquid chromatography (HPLC) with fluorescence detection. The analyzed values of 7 experiment diets were 1.69, 2.56, 3.37, 4.05, 4.73, 5.88, and 9.26 mg riboflavin/kg, respectively.

2.3. Sample preparation

The body weight of ducks from each pen was recorded on d 7, 14, and 21 of the experimental period. At 21 d of age, the average daily gain (ADG), average daily feed intake (ADFI), and gain to feed ratio (G:F) of each pen were determined. Then, 2 ducks from each pen were selected based on the average body weight of each pen and whole blood was collected via cardiac puncture. Blood samples were collected into heparin sodium-anticoagulant tubes and centrifuged at $1520 \times g$ for 10 min at 4 °C to obtain plasma. Plasma was stored at −20 °C until assayed for concentration of riboflavin and malondialdehyde (MDA), enzyme activities of superoxide dismutase (SOD), alanine transaminase (ALT), aspartate transaminase (AST) and lactate dehydrogenase (LDH). Finally, ducks were slaughtered by CO_2 inhalation. The liver was sampled immediately and then stored at −20 °C until analyzed for free riboflavin, FMN, FAD, SOD activity, and MDA content.

Free riboflavin concentrations in feed, plasma, and liver, and FMN and FAD concentrations in the liver were determined by reversed-phase HPLC according to the methods described previously by Tang et al. (2013). Before HPLC analysis, feed, liver, and plasma samples were prepared