Histopathological and biochemical evaluations of the kidney in broiler chickens under acute heat stress conditions

S.C. Huang¹², Y.F. Fu¹, Y.F. Lan², M.U. Rehman², and Z.X. Tong*¹

College of Animal Science and Veterinary Medicine, Henan Agricultural University, Zhengzhou 450002, People’s Republic of China. Received: 28-07-2016 Accepted: 13-12-2016 DOI: 10.18805/ijar.v0iOF.7652

ABSTRACT
The purpose of the present study was to investigate the correlation between acute heat stress and relevant histopathology and biochemical parameters of kidney function. A total of 80 healthy Arbor Acer (AA) broiler chickens were randomly divided into two groups: CT (Control Temperature; 22±1°C) group and HT (High Temperature; 38±1°C) group. Histopathological images revealed alteration in kidney (renal tubular lumen dilation with tubular necrosis, especially after 10h of heat stress) of broiler chickens in HT group leading to disturbance of acid base balance. Blood urea nitrogen (BUN) and creatinine (CREA) as serum markers of renal dysfunction were elevated significantly (p<0.05) after 5h, and especially, after 10h of heat stress (p<0.01) as compared with CT group. These results indicated that the evaluation of morphological and functional parameters in kidney is required, in order to monitor broiler chickens exposed to heat stress.

Key words: Biochemical parameters, Broiler chickens, Heat stress, Histopathology, Kidney.

Heat stress is one of the most challenging environmental conditions for commercial poultry, as broiler chickens are more sensitive to high temperature compared with other species of domestic animals (Geraert et al., 1993). During the past decade, many studies have been demonstrated that high temperature causes various damages to the animal organs, including injuries in heart, kidney, and liver (Bouchama et al., 2002; Yu et al., 2008; Liu et al., 2015).

The kidneys play a vital role in body homeostasis by adapting the renal excretion of fluid and electrolytes to body needs under the control of a systemic neurohumoral adjustment (Volker et al., 2006). However, only fewer studies are available to know the impact of short-term physiological changes in kidney under intense work for the period of heat stress. Therefore, we duplicated the chicken heat stress model that enabled us to study the interaction of the kidney and heat stress by way of histological alteration and biochemical parameters.

The animal experiments and procedures were performed in strict accordance with the guidelines of the regional Animal Ethics Committee and the protocol was approved by the Institutional Animal Care and Use Committee of Henan Agricultural University.

A total of 80 Arbor Acer (AA) broiler chickens, one day old, were purchased from a local Commercial Fowl Company (Henan, China) and were randomly divided into two groups: CT (Control Temperature; 22±1°C; n=40 and 10 per repetition) group and HT (High Temperature; 38±1°C; n=40 and 10 per repetition) group. On the 28th day of broiler chickens, room temperature was abruptly increased from 22±1°C to 38±1°C by using a heater and the relative humidity of the room was maintained at 50%±10% during the experiment. Six chickens were randomly selected from the 2h, 5h, and 10 of heat stress respectively. All experimental broiler chickens were killed rapidly by cervical dislocation.

To observe the histopathological changes in kidney tissue caused by heat stress, we fixed samples in 4% buffered formalin. Later, paraffin-embedded tissue sections were cut at 5-μm thicknesses, stained with Haematoxylin and eosin (H&E). The serum samples (n=6), obtained from blood collected in test tubes without the anticoagulant, were assayed for the concentrations of BUN and CREA. The tests were performed with the Idexx VetTest 8008 Chemistry Analyzer, according to the manufacturer’s instructions.

All biochemical data were statistically analyzed with SPSS statistical software for Windows (version 17; SPSS, Chicago, Illinois). A significance level of 0.05 was used. Data were expressed as means ± SEM.

The broiler chickens of HT group exhibited many clinic signs of decreased activity and feed intake with opened wings, increased drinking water, and diarrhoea during the...
heat stress, which were consistent with the previous reported (Kumar et al., 2013, Pearce et al., 2013). It showed that the model of heat stress in broiler chickens was copied successfully.

Heat stress might be associated with degenerative and necrotizing changes in the renal tubules and glomerulus. Several days after the occurrence of heat stress, there is evidence of renal failure due to degeneration and necrosis of renal tubules (Aengwanich et al., 2005). These changes are similar to the effects of acute heat stress on renal tubule and glomerulus in broilers in this study. The results revealed that kidney tissue was identified with the emergence of renal tubular luminal dilation with tubular necrosis, especially after 10h of heat stress compared to CT group (figure 1C, 1D). In addition, congestion could be found almost in every renal tubule, and lots of neutrophil granulocytes invaded accumulated in renal interstitial areas and the structure of glomerulus was changed (figure 1B-1D). It was suggested that the morphological structure of kidney was changed under heat stress condition in broiler chickens (Aengwanich 2009).

Serum creatinine is freely filtered by the glomerulus and is secreted by proximal tubular cells (Yuko et al., 2008); serum BUN level is very variable, depending on protein degradation and catabolism of amino acids (Marai et al., 2010). Koelkebeck et al. (1995), using white leghorn hens (50 weeks of age), documented that heat stress for 100 mins had no effect plasma enzymes, including uric acid and creatinine. However, in present study, serum results in broiler chickens showed that the level of serum BUN significantly increased ($p<0.05$) at 5h of heat stress, especially, markedly increased ($p<0.01$) at 10h of heat stress in the HT group. Compared with the control group, the level of serum CREA in serum significantly increased ($p<0.01$) during the heat stress from the HT group (figure 2). Further results indicated that the electrolyte balance was changed during heat stress and the young chickens had less heat tolerant ability.

In conclusion, the present study showed that acute heat stress can cause histopathological and biochemical parameters alterations that induce undesirable physiological changes in kidney, which may cause kidney dysfunction.

**Fig 1:** Histopathological characteristics of kidney tissue of broiler chickens after heat stress by H&E stain. Lots of red cells accumulation in renal tubules in kidney of heat stressed chickens was observed (arrow). (A) CT group (×400). (B-D) HT group: 2h, 5h, and 10h of heat stress, respectively (scale bar=20µm).
Fig 2: Serum biochemical parameters of kidney of broiler chickens after heat stress. (A) BUN, (B) CREA. Data were represented as the mean± SEM (n=6). Bars with different superscript letters were represented statistically significant differences (P<0.05) between the CT group and HT group. CT= Control Temperature, HT= High Temperature.

REFERENCES


