EFFECT OF DIFFERENT ACUTE HEAT STRESS DEGREE ON AMP-ACTIVATED PROTEIN KINASE (AMPK) IN CHICKEN MEAT

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I. INTRODUCTION

Heat stress (HS) has long been recognized as one of the prominent environmental elements influencing the poultry industry [1]. Our previous research found that HS under 36 °C caused chicken breasts to have significant lighter color and higher cooking loss. A rapid pH decline was observed in the first 1 hour post-mortem. However, as the HS temperature increased to 38°C and 40°C, this phenomenon weakened or disappeared [2]. Further work in glycolysis changes is necessary to explain this phenomenon. As a downstream component of protein kinase cascades, AMPK has been described as a central regulator of cellular energy metabolism and plays an important role in the regulation of postmortem skeletal muscle glycolysis [3, 4]. Under such a circumstance, this experiment was designed to reveal the changes of AMPK activity under different HS conditions and to find out its potential function in glycolysis after slaughter.

II. MATERIALS AND METHODS

One hundred male broilers (Arbor Acres) were raised in same condition (25 ± 1°C). Eighty four broilers were randomly selected at six weeks of age and then randomly divided into 7 groups (12 broilers in each group) to receive different treatments, including 1 control group (no HS treatment) and 6 HS treatments: (1) 36 °C for 1 h, (2) 36 °C for 2 h, (3) 38 °C for 1 h, (4) 38 °C for 2 h, (5) 40 °C for 1 h, and (6) 40 °C for 2 h. Each group consisted of 6 replicates, with 2 broilers in each replicate. After treatments, birds were slaughtered and bled in 5 min. Then the left pectoralis major muscles were removed manually via knife-cutting after bleeding, and stored under - 80 °C for AMPK activity analysis. The AMPK activity was analyzed according to the method of Xing et al., (2016) [5].

III. RESULTS AND DISCUSSION

Heat stress exposure time (1 h & 2 h) had no effect on AMPK phosphorylation ($P > 0.05$). Figure 1 shows a representative immunoblot of the phosphorylation of breast muscle AMPK under different HS temperatures. AMPK phosphorylation increased significantly from 5min to 1 h postmortem ($P < 0.05$), and then decreased at 2 h postmortem ($P < 0.05$). This indicated that the AMPK regulates glycolysis mainly during the early postmortem period. It was in agreement with our previous results that there was a big pH decline in the first hour postmortem.

No difference among the four HS treatments was found at either 5 min or 24 h postmortem ($P > 0.05$). Significant difference was only observed at 1h postmortem. The AMPK phosphorylation in 36 °C HS group was the highest, followed by those in 38 °C HS group, 40°C HS group and control group ($P < 0.05$). It was significantly higher than the control group ($P < 0.05$). While, there was no difference between 40 °C HS group and control. It showed that with the HS temperature increased from 36 °C to 40 °C, the AMPK phosphorylation level dropped to the normal level. This was in accordance with our previous results that rapid pH decline, lower water holding capability and higher light values were found in 36 °C HS group, but this phenomenon

weakened or disappeared with HS temperature increased to 40 °C.

IV. CONCLUSION

Moderate high temperature (36 °C) HS could active the AMPK more rapidly with higher level than other higher HS temperatures (38 °C & 40 °C) , which then accelerated the glycolysis more rapidly in the early postmortem and caused more pale color and lower water holding capability chicken meat. However, the mechanism why higher temperature HS caused AMPK phosphorylation decline is not clear and still needs further research.

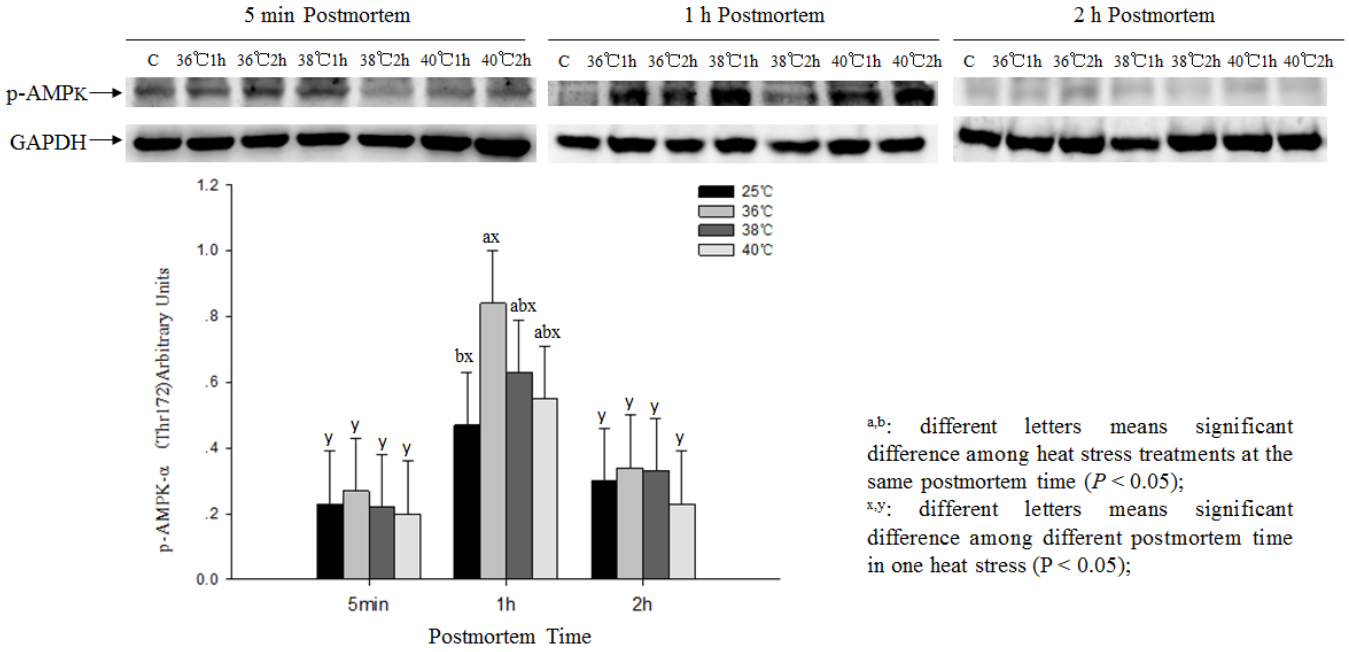

Figure 1 Effects of heat stress temperatures and time on phosphorylation of adenosine monophosphate-activated protein kinase (AMPK) (Thr172) as assessed by immunoblot technique (n = 6). (C: control, 25 °C)

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