EFFECT OF EARLY POSTMORTEM OXIDATIVE STRESS ON MITOCHONDRIAL REDOX STABILITY AND APOPTOSIS OF TWO PORCINE MUSCLES

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I. OBJECTIVES

Apoptosis has been suggested as a novel mechanism affecting meat quality attributes. Oxygen-deprivation-induced apoptosis is dependent on the intrinsic mitochondrial pathway, which is closely associated with metabolic status and mitochondria dysfunction of the muscle cells. Generation of reactive oxygen species (ROS) under oxidative stress could lead to further mitochondrial damage. However, the exact mechanisms of ROS-mediated mitochondrial apoptosis remain largely unknown. The objective of this study was to evaluate effects of oxidative stress on early postmortem muscles and its impacts on mitochondrial functionalities and apoptosis of 2 porcine muscles.

II. MATERIALS AND METHODS

Longissimus dorsi (LD) and *Psoas major* (PM) muscles were excised from 6 pork carcasses at 2 h postmortem. Each muscle sample was subdivided into 2 fractions and allocated to 2 treatments as follows; oxidizing condition as being immersed in 20 mM H_2O_2 for 60 min or control as being treated with distilled water. Upon treatments, all samples were vacuum packaged and stored for 0, 6, 24, and 48 h at 4°C. Biochemical attributes, including mitochondrial membrane permeability, mitochondrial lipid peroxidation, mitochondrial Ca^{2+}, and cytochrome c redox stability, were determined at each storage time. Data were analyzed by SAS (SAS Institute Inc., Cary, NC) to compare the traits across muscle types and treatments over time. Least-squares means were separated by least significant differences.

III. RESULTS

Overall, the mitochondrial membrane permeability was increased at 0–48 h post treatment in both muscles. PM had a higher membrane permeability than LD at 0–24 h ($P < 0.05$). The effect of oxidative stress on the mitochondrial membrane permeability was only significant in LD at 6–24 h. Oxidative stress resulted in a significant increase in mitochondrial lipid peroxidation and Ca^{2+} flux in both muscles. However, PM had a greater mitochondrial lipid peroxidation and Ca^{2+}, compared to LD ($P < 0.05$). While the cytochrome c redox stability decreased in both muscles, PM had a greater cytochrome c redox stability than LD ($P < 0.05$). These observations further support the hypothesis that ROS-mediated oxidative stress plays a major role in the activation of mitochondrial apoptosis by influencing important regulators of the mitochondrial apoptotic pathway. These results also imply that oxidative stress could induce the rate and extent of apoptosis process, but it could be muscle specific.

IV. CONCLUSION

The ROS generated by H_2O_2 significantly increased the mitochondrial oxidative stress levels. The results from the present study found that ROS-mediated oxidative stress enhanced the mitochondrial apoptotic pathway indicated by increased mitochondrial membrane permeability, mitochondrial lipid peroxidation, and overload of the production of

mitochondrial Ca^{2+}, from early postmortem muscles. However, the rate and extent of response to the given oxidative-stress-induced apoptosis could be muscle specific, where PM showed more ROS-mediated mitochondrial apoptosis compared to LD. Further research looking into the impact of oxidative-stress-mediated apoptosis on proteolytic enzyme activity and subsequent myofibrillar protein degradation is currently underway.

Keywords: apoptosis, pork loins, reactive oxygen species