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Heat and chemical stress modulate the expression of the α-RYR gene in broiler chickens

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ABSTRACT. The biological cause of Pork Stress syndrome, which leads to PSE (pale, soft, exudative) meat, is excessive release of Ca^{2+} ions, which is promoted by a genetic mutation in the ryanodine receptors (RyR) located in the sarcoplasmic reticulum of the skeletal muscle cells. We examined the relationship between the formation of PSE meat under halothane treatment and heat stress exposure in chicken αRYR hot spot fragments. Four test groups were compared: 1) birds slaughtered without any treatment, i.e., the control group (C); 2) birds slaughtered immediately after halothane treatment (H); 3) birds slaughtered immediately after heat stress treatment (HS), and 4) birds exposed to halothane and to heat stress (H+HS), before slaughtering. Breast muscle mRNA was extracted, amplified by RT-PCR, and sequenced. PSE meat was evaluated using color determination (L* value). The most common alteration was deletion of a single nucleotide, which generated a premature stop codon, resulting in the production of truncated proteins. The highest incidence of nonsense transcripts came with exposure to halothane; 80% of these abnormal

transcripts were detected in H and H+HS groups. As a consequence, the incidence of abnormal meat was highest in the H+HS group (66%). In HS, H, and C groups, PSE meat developed in 60, 50, and 33% of the samples, respectively. Thus, halothane apparently modulates αRYR gene expression in this region, and synergically with exposure to heat stress, causes Avian Stress syndrome, resulting in PSE meat in broiler chickens.

Key words: PSE (pale, soft, exudative) meat; Abnormal meat color; Ryanodine receptor

