

## Expression of Inflammatory and Cell Death Genes and DNA Damage Induced by Endotoxic Shock in Laying Hens

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**Abstract :** This investigation was conducted to determine the physiological response and evaluate the expression of inflammatory and cell death genes and DNA damage induced by endotoxic shock in laying hens. Endotoxic shock was induced by a single intravenous injection of 107 Escherichia coli (E. coli,) colony/hen. In the present study, 240 forty-week-old laying hens (H&N) were randomly assigned into 2 groups with 3 replicates of 40 birds each. Hens were reared in battery cages with wire floors in an open-sided housing system under natural conditions. Housing and general management practices were similar for all groups. At 42-wk of age, 45 hens from the first group (15 replicate) were infected with E. coli, while the same number of hens from the second group was injected with saline and served as a control. Heat shock protein-70 (HSP-70) expression, plasma corticosterone concentration, body temperature, and the gene expression of bax, caspase-3 activity, P38, Interlukin-1 $\beta$  (IL-1 $\beta$ ), and tumor necrosis factor alpha (TNF- $\alpha$ ) genes and DNA damage in the brain and liver were measured. Hens treated with E. coli showed significant ( $P \leq 0.05$ ) increase of body temperature by 1.2 °C and plasma corticosterone by 3 folds compared to the controls. Further, hens injected with E.Coli showed markedly over-expression of HSP-70 and increase DNA damage in brain and liver. These results were synchronized with activating cell death program since our data showed significant ( $P \leq 0.05$ ) high expression of bax and caspase-3 activity genes in the brain and liver. These results were related to remarkable over-inflammation gene expression of P38, IL-1 $\beta$ , and TNF- $\alpha$  in brain and liver. In conclusion, our results indicate that endotoxic shock induces inflammatory physiological response and triggers cell death program by promoting P38, IL-1 $\beta$ , and TNF- $\alpha$  gene expression in the brain and liver.

**Keywords :** chicken, DNA damage, Escherichia coli, gene expression, inflammation

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