A mutation in the DNA binding domain of chicken STAT5 inhibits leptin dependent signal transduction *in vitro*

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Whereas STAT5 as well as STAT3 is phosphorylated by leptin, how STAT5 contribute on avian leptin system is not fully understood. Regarding this issue, we cloned chicken STAT5 cDNA and found a mutant STAT5 possesses 4 amino acid substitutions. Therefore, we characterized property of the mutant chicken STAT5 (chSTAT5M) on leptin dependent signaling via chicken leptin receptor (chLEPR). In chLEPR expressing CHO-K1 cells, overexpressed chSTAT5M was phosphorylated by leptin. Furthermore, leptin-induced phosphorylation of STAT3 was not affected in chSTAT5M expressing cells. However, luciferase assay showed that leptin induced transcription was inhibited by chSTAT5M, and which may indicate that chSTAT5M interferes transcriptional activity of STAT3. Characteristic mutations were observed in DNA binding domain (R430C) and transcriptional activation domain (F757S) in the chSTAT5M. Then, we further analyzed that effect of the two amino acid substitutions found in the chSTAT5M on signal transduction of leptin. chSTAT5/R430C inhibited leptin induced activation of reporter gene, but chSTAT5/F757L didn't. Taken together, a mutation in the DNA binding domain of chSTAT5M might be responsible to show its inhibitory effect. Present study may suggest that activated chSTAT5M formed heterodimer with STAT3 and which inhibited signal transduction by preventing interaction with target gene.