

Regulation of Acyl-CoA-binding Protein Gene Expression by Hormones in the
Insect *Rhodnius prolixus*

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Acyl-CoA esters have many functions in cell metabolism, such as energy production and cell signaling. Acyl-CoA-binding protein (ACBP), a highly conserved 10 kDa intracellular protein, binds long-chain acyl-CoA esters with very high affinity, directing them to specific metabolic routes and protecting them from hydrolysis. Using RT-PCR, ACBP gene expression was detected in anterior and posterior midgut, fat body, ovary, flight muscle and salivary glands of *Rhodnius prolixus*, and it was highest (~ 5-fold) in posterior midgut. Expression analysis of ACBP gene in the posterior midgut by Real-Time PCR showed a great increase after blood meal. It was very high (~ 10-fold increase) on first day after feeding and then decreased. The injection of 4 pmol of 5-hydroxytryptamine (serotonin) to unfed females produced a similar effect, inducing an increase of approximately 5-fold in ACBP gene expression of posterior midgut. The same increase was also observed in the presence of Cholera Toxin, a G protein activator, and Dibutyryl-cAMP, a cyclic AMP analogue. The effect caused by injection of serotonin was inhibited by Spiperone, a specific antagonist of serotonin receptors type 2A. Moreover, the injection of 4 ng of 20-hydroxyecdysone into unfed females inhibited the expression of this gene in approximately 30%. These results indicate the presence of a mechanism of control of the ACBP gene expression in the posterior midgut, where a cascade of cellular responses triggered by serotonin acts inducing this gene. After feeding, 20-hydroxyecdysone would be responsible for restoring the basal expression level at the days following this event. Supported by CNPq, PIBIC/UFRJ, Faperj and CAPES.

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