Evidences of the Conserved Insulin Signaling Pathway Machinery and its Involviment in Glycogen Metabolism on *Rhipicephalus microplus* development

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Besides its metabolic role, insulin signaling pathway (ISP) is widely described as crucial for vertebrate and invertebrate embryogenesis and development. In such cascade Phosphatidylinositol 3-OH Kinase (PI3K) is hierarchically located upstream Protein Kinase B (PKB). Recent discoveries from our group point that the insulin signaling pathway (ISP) is present and plays important roles on *Rhipicephalus microplus* ticks, embryos and BME26 embryo cell line. Exogenous insulin increased glycogen content in a dose-dependent manner on BME26 cells. Moreover, the expression level of PI3K's regulatory subunity (p85) increased in the presence of insulin, as determined by Real Time RT-PCR. When PI3K inhibitors (Wortmannin or LY294002) were added these effects were reversed. Additionally, PI3K inhibition increased the expression level of two insulin-regulated downstream targets from glycogen metabolism (GSK3b) and gluneogenesis (PEPCK) pathways. GSK3b expression was increased in ovaries and especially high in partially engorged females. Though p85 and GSK3b presented different expression profiles during embryogenesis, GSK3b activity showed strong correlation with glycogen content during the same period. Interestingly, the GSK3b specific inhibitor SB216763 was able to inhibit GSK3b enzymatic activity on egg homogenates. Further studies on the correlations of these ISP components are on the way and point to the presence of a PKB kinase, as a partial sequence was obtained using degenerate primers and touchdown PCR. Our results support the presence of an insulin responsive enzymatic machinery closely related with glycogen metabolism on *R. microplus* embryogenesis.

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