PI3K/PKB SINALING IN *Rhipicephalus (Boophilus) microplus* TICK EMBRYO CELL LINE BME26

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Ticks are obligatory blood-sucking arthropods and important vectors of both human and animal diseases. In order to study the insulin triggered pathway and its possible roles during embryogenesis we are using a culture of embryonic *Rhipicephalus (Boophilus) microplus* cells (BME26). 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). Exogenous insulin is able to increase the expression level of PI3K's regulatory subunity (p85), as determined by Real Time RT-PCR. In the presence of PI3K inhibitors (Wortmannin or LY294002) these effects were reversed. This correlates well with the activation of PKB by phosphorylation, as it appears to be PI3K-dependent. Additionally, PI3K inhibition increased the expression level of two insulin-regulated downstream targets from glycogen metabolism (GSK3b) and gluneogenesis (PEPCK) pathways. GSK3b inhibition by phosphorylation diminished in cells treated with PI3K inhibitors. These results strongly suggest the presence of an insulin sensitive PI3K-PKB axis in BME26 cells. The further study of PI3K and PKB activity in egg homogenates during embryogenesis may help us understand the role of ISP for *R. microplus* development.

Key words: insulin signaling, embryonic cells, tick.
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