CALCIUM INVOLVEMENT IN GLUCOSE INDUCED GLUT3 EXPRESSION IN PREIMPLANTATION MOUSE EMBRYOS

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Despite their inability to utilise glucose for energy prior to compaction (E3), mouse embryos have a requirement for at least a brief glucose exposure to permit normal development. In the absence of this glucose pulse in vitro, we and others have found that embryos cleave to form morulae but fail to form blastocysts and subsequently degenerate. These embryos do not develop the capacity to utilise glucose preferentially and are unable to adapt to their nutrient environment and utilise alternate substrates (1). This inability to utilise glucose is due to failure to express GLUT3 at compaction (2). Brief glucose exposure prior to the 8-cell stage is sufficient to permit the embryo to undergo compaction, express GLUT3 and ultimately form a blastocyst, suggesting that glucose induces metabolic differentiation of the developing embryo. In this study we have explored the role of intracellular calcium in response to glucose given its central role in pancreatic glucose induced signalling events. Zygotes were cultured in the presence and absence of glucose and treated with either calcium mobilising agents, ethanol or ionomycin at 54 h post hCG or with the intracellular calcium chelator BAPTA-AM. Embryos were fixed and assayed for GLUT3 expression individually at 96 h post hCG using confocal immunofluorescence. Release of intracellular calcium by either ethanol or ionomycin, activated GLUT3 expression in a glucose like manner ($P < 0.01$) suggesting that calcium transients may be involved in glucose sensing. Moreover, buffering of calcium with the calcium chelator BAPTA-AM interfered with the ability of glucose to activate GLUT3 expression ($P < 0.05$), suggesting that glucose exposure does result in calcium transients that affect GLUT3 expression. It is unclear whether these calcium transients occur as a result of influx of extracellular calcium via voltage-gated ion channels or the release of calcium from intracellular stores via inositol triphosphate-gated calcium release channels in the endoplasmic reticulum.

(1) Martin and Leese (1995) *Mo.l Reprod. Dev.* **40**, 436–443. (2) Pantaleon *et al.* (2001) *Proc 32nd Annual SRB Conference, Gold Coast, Qld.* A42.

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