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DECREASED OOCYTE-GRANULOSA CELL GAP JUNCTION COMMUNICATION AND CONNEXIN EXPRESSION IN A TYPE I DIABETIC MOUSE MODEL

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Women with Type I and Type II diabetes are two to eight times more likely to experience poor pregnancy outcomes, including fetal abnormalities and miscarriage, than nondiabetic women. Evidence of impaired oocyte meiotic maturation, abnormal oocyte metabolism, and increased granulosa cell apoptosis in murine models of Type I or insulin-dependent diabetes suggests developmental disturbances in the preovulatory oocyte. Normal folliculogenesis is mediated by gap junction communication between the oocyte and surrounding cumulus cells. This interdependent relationship between somatic and germ cells is necessary to produce an oocyte capable of fertilization and proper embryo development. The objective of this study was to investigate differential levels of communication in a streptozotocin-induced Type I diabetic B6SJL/F1 mouse model as compared to nondiabetic mice. We compared the expression of a select group of connexins, the transmembrane proteins that comprise the gap junction, that are thought to play significant roles in ovarian function. Real-time RT-PCR analyses revealed comparable mRNA expression of Cx26 and Cx57 in diabetic and nondiabetic mice. We observed a significant decrease in Cx37 expression in diabetic mice as compared to nondiabetic mice. Western analyses detected protein expression of Cx26 in cumulus-enclosed oocytes (CEOs) but not denuded oocytes (DOs) and of Cx37 in both CEOs and DOs. Protein levels of both connexins were lower in diabetic mice compared to nondiabetic mice. Fluorescence recovery after photobleaching (FRAP) analyses showed a 60% decrease in oocyte-granulosa cell communication in diabetic mice compared to nondiabetic mice. This decrease in connexin expression and impaired gap junction communication may be responsible for the adverse effects observed in diabetic pregnancies.