Angiotensin II Stimulates Glucose Transport via PKC in Mouse Embryonic Stem Cell

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Background : ANG II was initially described as being a primary vasoconstrictor peptide. However, recent studies demonstrate that ANG II has growth factor- and cytokine-like properties. However, little is known regarding whether ANG II regulates glucose transport in mouse embryonic stem cells (ES). Thus, in present study, glucose transport in response to ANG II was assessed by measuring the uptake of [3H]-2-deoxyglucose, a radiolabeled non-metabolizable glucose analogue, and its related signal pathway in mouse ES cells. Methods. Mouse ES cells were grown in D-MEM (Dulbecco's Modified Eagle Medium) containing 15 % FBS supplemented with 1000 unit Leukemia Inhibitory Factor (LIF). ES cells were exposed to ANG II (10-7M) for 24 hr and [3H]-2-deoxyglucose uptake, western blotting analysis were performed. Results. ANG II significantly increased [3H]-2-deoxyglucose uptake in time- and concentration-dependent manner (>24 hr, >10-7M) and increased expression of glucose transporters (GLUTs) by 63±2.6% (GLUT1) and 144±5.2% (GLUT2) expression, respectively compared to control. ANG II-induced increase of [3H]-2-deoxyglucose uptake was blocked by losartan [an ANG II type 1 (AT1) receptor blocker], but not by PD 123319, [an ANG II type 2 (AT2) receptor blocker]. In addition, ANG II-induced stimulation of [3H]-2-deoxyglucose uptake was completely prevented by staurosporine, bisindolylmalemide I, and H-7, protein kinase C (PKC) inhibitors,. Indeed, ANG II activated a PKC translocation from the cytosolic to membrane fraction, suggesting a role of PKC. Conclusions. ANG II increases glucose transporter by PKC activation via AT1 receptor in mouse embryonic stem cells.