

Effect of Glucose on the Expression of CD24 Marker in Umbilical Cord Derived Mesenchymal Stem Cells

Rithika Rajendran, Febe Renjitha Suman, Alan Mathew Punnoose, Sarah Kuruvilla and S Krishnakumar

Sri Ramachandra Medical College and Research Institute, India

Diabetes Mellitus (DM) is a chronic metabolic disease with high morbidity and significant mortality. The prevalence of DM is quite alarming with around 347 million people affected worldwide, the largest contributors to the disease load being India (65.1 million people affected in 2013) and China. Though the prevalence in India of 9.1% is only marginally higher than the worldwide prevalence of 8.3% the massive population contributes to the disease load. Though many drugs are available, insulin is the better choice to reduce blood sugar levels. Insulin administered through syringes and pumps is discharged at doses adjusted according to blood sugar levels estimated either once daily or once in two weeks. However, there occur periods of variation in glucose level at the cellular level which is responsible for long term complications. The only definitive treatment option is a source of islet cells which discharges human insulin according to the changing milieu in the body to maintain sugar levels. The difficulties encountered with islet allograft transplant are the availability of cadaver donor, need for immune suppressants, graft cell loss and autoimmunity. Hence it is the need of the hour to manufacture islet cells in vitro to have a constant source of supply replacing the currently available therapeutic measures. The development of in vitro islets requires a viable source of stem cells. The perinatal tissues which include umbilical cord and placenta are wasted every day in tons. They are rich sources of MSCs which are immunologically naive and obtaining consent is relatively easy, as these tissues are only discarded otherwise. At the initial stage of the process, the MSCs have to be isolated. Though there are different protocols defined by researchers to isolate MSCs from the umbilical cord, no standardized protocol is available yet. Currently, islets cultured from human stem cells are quite fragile and have a limited shelf life

of only 72 hours. Also, the characterization, culture, and storage protocols have to be standardized. By studying pancreatic progenitor cells and development pathways we may find methods to improve our culture and storage techniques. Novel markers identify pancreatic precursor cells. Based on the observations made in the present study and review of literature about other studies done so far, it seems that MSCs express CD24. These cells may develop into a clone of pancreatic progenitors, neural stem cells or cancer stem cells. This development depends on the environmental conditions and differentiating factors available. It is known that elevated glucose concentrations impair cellular functions and induce apoptosis and is supposed to be the cause of further degeneration of pancreatic beta cells and all the long term complications in DM. It is hypothesized that high blood glucose levels also affect the body's native stem cells through mechanisms still under study. Controversial reports on the toxic effects of glucose on MSCs both in vivo and in vitro are available. It had been reported by Stolzing, et al. that high glucose is toxic to MSCs derived from rats while cell proliferation increased and the rate of apoptosis decreased when glucose concentration was reduced. Li, et al. cultured bone marrow-derived human MSCs in media containing 5, 6, 11, 25 and 40 mmol concentration of glucose and measured the rate of proliferation and apoptosis. The inhibitory effect on cell apoptosis was noticed on short term exposure to high glucose medium but there was no effect on cell proliferation and apoptosis in long term cultures. Also Dhanasekaran, et al. reported retention of MSC surface marker characteristics, proliferation, and differentiation potential in bone marrow and adipose-derived MSCs cultured at 25 mmol glucose concentration.

johnjennerresearch@gmail.com