J Glycobiol 2017, 6:2(Suppl)
DOI: 10.4172/2168-958X-C1-009

3rd Glycobiology World Congress

June 26-28, 2017 London, UK

Proteoglycans and human mesenchymal and neural stem cells: Can we control lineage fate?

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Human neural stem cells (hNSCs) and mesenchymal stem cells (hMSCs) are now routinely used in cell culture models; however the processes and the mechanisms that regulate these cells are still largely unknown. Despite hMSC neural lineage potential, the current lack of understanding of lineage regulation limits their use in the development of human neurogenesis models as well as our understanding of how numerous neurological and brain disorders occur. The identification of the biomarkers required for maintaining neural stem cells in their undifferentiated state as well as those needed to direct lineage differentiation is central to understand neurogenesis. How these processes are regulated will help to further unravel the structural complexity of the human brain and the role of associated biological and other factors in neurogenesis. These also have important ramifications for the successful integration of newly formed neurons into existing/remaining neural circuits. The heparan sulfate (HS) and chondroitin sulfate (CS) proteoglycans (PGs) are widely distributed in the body and the nervous system, primarily in the extracellular matrix. Multiple studies have identified a role for these proteins during normal development of the nervous system as well as in the maintenance of stem cell pools in the adult. What has yet to be elucidated is how these PGs contribute to the control of neural lineage regulation, proliferation and differentiation? As NSCs have the ability to generate neurons, astrocytes and oligodendrocytes, these cells provide a promising model for understanding the process of neurogenesis. In addition, MSCs have neural lineage potential and may contribute to the localized microenvironment to mediate stemness as well as lineage specification. The identification of the factors regulating these cellular processes will complement broader research disciplines that could be applied to all fields of research and may provide new strategies for their efficient implementation in therapeutic applications.

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Is expression of blood plasma chitotriosidase O-glycans associated with type-2 diabetes?

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Chitotriosidase (CHIT1) is the first active chitinase discovered in human plasma. The exact functions of this enzyme are unexplained, but its involvement in the inflammatory processes has been suggested. Increased level of CHIT1 is also observed in type-2 diabetes (T2D). Since immunoreactivity and glycosylation profile of this protein have not been studied yet, we analyzed CHIT1 immunoblotting pattern and O-glycans expression in healthy subjects and T2D patients. CHIT1 concentration and activity were measured using immunoenzymatic and fluorometric techniques, respectively. Plasma samples from healthy and diabetic individuals were pooled and electrophoresed in SDS-polyacrylamide gel. Western blotting was used for detection of CHIT1 bands. The glycosylation was studied with lectin-ELISA with biotinylated lectins: Jacalin, *Maclura pomifera and Vicia villosa*, detects complete and truncated O-glycans, respectively. We observed significantly higher CHIT1 concentration and activity in diabetic patients than in controls ($p<0.001$). In the examined groups, bands corresponding to CHIT1 molecular mass had slightly different locations: 49 kDa and 45 kDa for healthy and T2D subjects, respectively. The expression of glycotopes reacting with applied lectins was lower in T2D and significantly different in analyzed groups ($p<0.01$). The differences in molecular masses of CHIT1 in examined groups may derive from the alterations in CHIT1 glycosylation observed by us. In diabetes, the higher CHIT1 concentration and activity was accompanied by significantly decreased CHIT1 reactivity with lectins, what may affect CHIT1 properties and have association with disease progression. Further analysis of CHIT1 glycome may be helpful in better understanding of chitotriosidase biological function in T2D pathology.

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