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Fucose-Dependent Differentiation and Gene Expression of Common Myeloid Progenitor Cells through Notch Signaling Pathways

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Charles is currently a second year student at Case, pursuing a Bachelor of Science in biology and a Bachelor of Arts in psychology. Aside from his academics and research, he is a Resident Assistant for Juniper Residential College as well as a member of Koinonia Christian Fellowship. Charles's current career plans are to attend medical school and continue research in pathology and immunology.

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ABSTRACT

The Notch pathway is an extensively utilized, evolutionarily maintained regulatory system which mediates a wide range of fate decisions among multipotent precursor cells by inhibiting differentiation along one pathway while promoting self-renewal or differentiation along an alternative pathway. Notch signaling has been shown to affect haematopoietic stem cell (HSC) self-renewal and differentiation, T cell versus B cell fate specification, and myeloid cell differentiation. The diverse functions of Notch in vertebrates are facilitated by complex interactions between four Notch receptors and five Notch ligands, all of which are expressed by hematopoietic cells and stromal cells. Moreover, Notch signaling is modulated by genes such as *fringe* as well as two unusual types of *O*-linked glycosylation; the addition of *O*-linked glucose (*O*-glucose) and *O*-linked fucose (*O*-fucose). Our goal is to determine whether *in vitro* myeloid differentiation is regulated by Notch activation, and whether this is a fucose-dependent process. Specifically, we focused our research on common myeloid progenitor (CMP) cell differentiation and the dynamic change of Notch-targeted genes during Notch regulated myeloid differentiation that is modified by fucosylation.

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