

Galectin-1-matured human monocyte-derived dendritic cells have enhanced migration through extracellular matrix

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Dendritic cells (DCs) are potent mediators of the immune response, and can be activated by a variety of signals, primarily exogenous pathogen components. Galectin-1 is a member of the highly conserved β -galactoside-binding lectin family which binds galactoside residues on cell-surface glycoconjugates. Galectin-1 is known to play a role in immune regulation via action on multiple immune cells including T cells and B cells. Here we show that galectin-1 induces a phenotypic and functional maturation in human monocyte-derived dendritic cells (MDDCs) similar to but distinct from the activity of the exogenous pathogen stimuli, lipopolysaccharide (LPS). Immature human MDDCs exposed to galectin-1 upregulated cell surface expression of CD40, CD83, CD86, and HLA-DR, a characteristic phenotype of mature DCs, and galectin-1 treated immature MDDCs secrete high levels of IL-6 and TNF- α , stimulate T cell proliferation, and show reduced endocytic capacity, similar to LPS-matured MDDCs. However, unlike LPS-matured DCs, galectin-1-treated MDDCs did not produce the Th1-polarizing cytokine IL-12. Microarray analysis revealed that while galectin-1 generally modulated many of the same DC maturation genes as LPS, galectin-1 also uniquely upregulated a significant subset of genes related to cell migration through extracellular matrix. Indeed, compared to LPS, galectin-1-treated human MDDCs exhibited significantly better chemotactic migration through Matrigel, an *in vitro* extracellular matrix model. Our findings show that galectin-1 is a novel endogenous activator of human DCs that upregulates a significant subset of genes distinct from those regulated by a model exogenous stimuli (LPS). One unique effect of galectin-1 is to increase DC migration through ECM, suggesting that galectin-1 may be an important component in initiating an immune response.