Regulatory circuits mediated by lectins in immune tolerance and inflammation

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Homeostatic signals delivered in the form of immunosuppressive cytokines or inhibitory receptors are integrated into regulatory circuits that sustain peripheral tolerance mechanisms. These mechanisms serve to prevent collateral tissue damage resulting from over-exuberant immune responses to pathogens or seemingly-innocuous environmental stimuli. Conversely, their aberrant activation represents a significant hurdle for the development and perpetuation of autoimmune disease. Although the potential of therapeutic interventions is under-appreciated for many years, exciting findings underscore the essential contribution of cell-cell surface glycocalyx-mediated interactions. These findings underscore the regulatory circuits operating in immune homeostasis. Indeed, endogenous glycobinding proteins in the cell surface specifically recognize glycans on the cell surface and convey this information to functional cellular responses.  

Galectins are a family of soluble lectins characterized by a conserved carbohydrate recognition domain (CRD) that recognizes N- and O-glycans expressing the disaccharide N-acetyllactosamine (Galβ(1-4)GlcNAc or LacNAc). Secreted galectins, in contrast to cytokines or chemokines, do not have specific receptors, but can mediate cellular communication through recognition of a preferred set of cell surface glycopolymers. Galectins can affect cell function and survival through interactions with their CRD and by binding their unique CRD. 

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References