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Dectin-1 Receptor Ligands as Anticancer Agents

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Pattern-recognition receptors (PRRs) detect molecular signatures of microbes and initiate immune responses to infection. Immune responses generated by prototypical PRRs such as Toll-like receptors (TLRs) have been widely investigated. In contrast, the immune responses initiated by other classes of putative PRRs remain ill defined. C-type lectins are a class of PRRs that recognize carbohydrate structures which are often part of microbial pathogens. Dectin-1 is a C-type lectin receptor present on dendritic cells that recognizes fungal β-glucans. Our investigations suggest that Dectin-1 is not just an antigen uptake receptor but also a modulator or initiator of adaptive immune responses. Human dendritic cells stimulated with Curdlan, Dectin-1 agonist prime CD4 Th17 responses via IL-23 production. Furthermore, these CD4 T cells induce differentiation of B cells to secrete IgG and IgA. More importantly; these dectin-1 stimulated dendritic cells promote the expansion and differentiation of granzyme B expressing cytotoxic T lymphocyte that display high cytolytic activity against target tumor cells *in vitro*. The capacity of Curdlan-stimulated human DCs to induce differentiation of these cells makes them attractive target for manipulations in clinic against cancer.