1. Record Nr. UNINA9910346736903321

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Titolo	Macromolecular Structure Underlying Recognition in Innate Immunity
Pubbl/distr/stampa	Frontiers Media SA, 2018
Descrizione fisica	1 electronic resource (151 p.)
Collana	Frontiers Research Topics
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia

Sommario/riassunto

Immune molecules have evolved to distinguish “self “molecules from “non-self”, “altered self” and “danger” molecules. Recognition is mediated via interactions between pattern recognition receptor molecules (PPRs) and their ligands, which include hydrophobic and electrostatic interactions between amino acid residues on the PPRs and uncharged or charged groups on amino acid residues, sugar rings or DNA/RNA molecules. Recognition in innate immunity range from cases (C1q, mannin-binding protein etc) where recognition is orchestrated by interaction between many ligands with one receptor molecule, and density of interaction is necessary for strong specific recognition, distinct from weak non-specific binding, and cases such as TLRs and NLRs where recognition involves complexation of single receptor and ligand, followed by oligomerisation of the receptor molecule. The majority of PPR molecules bind and recognise a wide variety of ligands, e.g TLR4 recognises LPS (gram negative bacteria), Lipotechoic acid (gram positive bacteria), heat shock protein hsp60, respiratory syncytial virus fusion protein etc, molecules that are structurally dissimilar to each other. This indicates considerable flexibility in their binding domains (amino acid residue variations) and modes (hydrophobic and charged, direct or mediated via an adaptor molecule). However, in many cases there is a dearth of structural and molecular data available, required to delineate the mechanism of ligand binding underlining recognition in pathogen receptors in innate immunity. Insights into

requirements of conformation, charge, surface etc in the recognition and function of innate immunity receptors and their activation pathways, based on current data can suggest valuable avenues for future work.