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Innate sensors of pathogen and stress: linking inflammation to obesity.

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Abstract

Pathogen and nutrient response pathways are evolutionarily conserved and highly integrated to regulate metabolic and immune homeostasis. Excessive nutrients can be sensed by innate pattern recognition receptors as danger signals either directly or through production of endogenous ligands or modulation of intestinal microbiota. This triggers the activation of downstream inflammatory cascades involving nuclear factor κB and mitogen-activated protein kinase and ultimately induces the production of inflammatory cytokines and immune cell infiltration in various metabolic tissues. The chronic low-grade inflammation in the brain, islet, liver, muscle, and adipose tissue further promotes insulin resistance, energy imbalance, and impaired glucose/lipid metabolism, contributing to the metabolic complications of obesity, such as diabetes and atherosclerosis. In addition, innate pathogen receptors have now emerged as a critical link between the intestinal microbiota and host metabolism. In this review we summarize recent studies demonstrating the important roles of innate pathogen receptors, including Toll-like receptors, nucleotide oligomerization domain containing proteins, and inflammasomes in mediating the inflammatory response to metabolic stress in different tissues and highlight the interaction of innate pattern recognition receptors, gut microbiota, and nutrients during the development of obesity and related metabolic disorders.

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KEYWORDS: ACS; Apoptosis-associated speck-like protein containing a CARD; BM; Bone marrow; DAMP; Danger-associated molecular pattern; HFD; High-fat diet; IAPP; IKK; Islet amyloid polypeptide; IκB kinase; JNK; LDL; Low-density lipoprotein; MAPK; Mitogen-activated protein kinase; MyD88; Myeloid differentiation factor 88; NAFLD; NF-κB; NLR; NOD; Nod-like receptor; Nonalcoholic fatty liver disease; Nuclear factor κB; Nucleotide oligomerization domain; PAMP; PRR; Pathogen-associated molecular pattern; Pattern recognition receptor; T2D; TIR domain-containing adaptor-inducing IFN-β; TLR; TRIF; Toll-like receptor; Type 2 diabetes; c-Jun N-terminal kinase; metabolic inflammation; microbiota; obesity

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