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Titel des Beitrags:
Adipose Recruitment and Activation of Plasmacytoid Dendritic Cells Fuel Metflammation.

Abstract:
In obese individuals, visceral adipose tissue (VAT) is the seat of chronic low-grade inflammation (metflammation), but the mechanistic link between increased adiposity and metflammation largely remains unclear. In obese individuals, deregulation of a specific adipokine, chemerin, contributes to innate initiation of metflammation by recruiting circulating plasmacytoid dendritic cells (pDCs) into VAT through chemokine-like receptor 1 (CMKLR1). Adipose tissue-derived high-mobility group B1 (HMGB1) protein activates Toll-like receptor 9 (TLR9) in the adipose-recruited pDCs by transporting extracellular DNA through receptor for advanced glycation end products (RAGE) and induces production of type I interferons (IFNs). Type I IFNs in turn help in proinflammatory polarization of adipose-resident macrophages. IFN signature gene expression in VAT correlates with both adipose tissue and systemic insulin resistance (IR) in obese individuals, which is represented by ADIPO-IR and HOMA2-IR, respectively, and defines two subgroups with different susceptibility to IR. Thus, this study
reveals a pathway that drives adipose tissue inflammation and consequent IR in obesity.

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