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EXPERIMENTAL CHRONIC INFLAMMATION OF LYMPH NODES INDUCES THE FORMATION OF MORE ADIPOCYTES IN CONTIGUOUS ADIPOSE TISSUE

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OBJECTIVES: Most of the adipose depots that hypertrophy in HIV-associated adipose redistribution syndrome (HARS), including the mesentery and omentum, contain embedded lymphoid tissue. The hypothesis that signals from inflamed lymphoid cells induce the formation of additional mature adipocytes in adjacent adipose tissue was tested by chronically activating a single peripheral lymph node with mild immune stimulants that caused only local inflammation, avoiding fever, anorexia and other systemic responses.

METHODS: One popliteal lymph node of large male rats was stimulated by local subcutaneous injection of 10 μ g or 20 μ g lipopolysaccharide three times/week for 6 weeks. Adipocyte volumes in sites defined by their anatomical relations to the stimulated and homologous unstimulated popliteal lymph nodes were measured, plus adipocyte complement of the popliteal depot, and the lipid and protein content of adipocytes and adipose stroma.

RESULTS: The higher dose of lipopolysaccharide doubled the mass of the locally-stimulated lymph node and the surrounding adipose tissue enlarged by the appearance of additional mature adipocytes. Similar but smaller changes were observed in the popliteal adipose depot of the unstimulated leg and in a nodeless depot. The lipid content of adipocytes decreased and that of the stroma increased dose-dependently in all samples measured but the changes were consistently greater in the depot surrounding the stimulated lymph node. These findings are consistent with the observation that newly formed lymphoid cells in an immune-stimulated lymph node preferentially incorporate

fatty acids derived from the adjacent adipocytes. The protein content of adipocytes and stroma increased in samples surrounding the stimulated node, as expected from the increased numbers of dendritic cells that permeate the perinodal adipose tissue and may mediate the transfer of lipids from adipocytes to lymphoid cells.

CONCLUSION: We conclude that chronic immune stimulation of lymphoid tissues induces lipolysis and the formation of more adipocytes in the adjacent adipose tissue. These findings are consistent with the hypothesis that perinodal adipocytes interact locally with lymphoid tissues, and suggest mechanisms for the selective hypertrophy of lymphoid-containing adipose depots in HARS. The formation of additional adipocytes may explain why HARS reverses very slowly during interruptions to antiviral therapy, often not at all.

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