

ADIPONECTIN LEVELS AND TNF BLOCKADE

In patients with inflammatory arthritis, the pathways through which treatment with tumor necrosis factor (TNF) inhibitors might influence cardiovascular risk are unclear. According to new research, however, this effect of anti-TNF therapy is unlikely to be attributable to the agents' effects on circulating levels of the adipocyte-derived hormone adiponectin.

"Adiponectin is of course linked to insulin sensitivity and lower diabetes risk, but it is also perceived to have anti-inflammatory effects and an inverse interaction with inflammation parameters," says the study's lead investigator, Mike Peters. To date, evidence of whether attenuation of inflammation leads to a rise in circulating levels of adiponectin in patients with inflammatory arthritis has been inconclusive.

To address this question the researchers analyzed data from a placebo-controlled trial of oncept in 126 patients with psoriatic arthritis, as well as a prospective trial of 171 consecutive patients with rheumatoid arthritis treated with adalimumab. Baseline correlations between levels of adiponectin and those of several metabolic markers of cardiovascular risk were as expected.

Changes from baseline adiponectin levels were determined after 12 weeks of oncept therapy or 16 weeks of adalimumab therapy. "In both trials," says Peters, "although inflammation markers and disease activity were substantially suppressed, we observed no change in adiponectin levels in the circulation." In addition, no correlation was observed between changes in levels of adiponectin and C-reactive protein. The results do not support a major role for adiponectin in the inflammatory state.

"These data are by far the best-quality data to address this question and suggest that adiponectin levels are not modulated by systemic inflammation," says Peters.

Sarah Price

Original article Peters, M. J. L. *et al.* Lack of effect of TNF α blockade therapy on circulating adiponectin levels in patients with autoimmune disease: results from two independent prospective studies. *Ann. Rheum. Dis.* doi:10.1136/ard.2009.114207