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THERAPY

TNF inhibition suppresses angiogenic mechanisms implicated in RA

"TNF blockade may be an important mechanism in combating rheumatoid arthritis (RA) angiogenesis" states Alisa Koch, author of a new study published in *Arthritis Research & Therapy*. The *in vitro* research suggests that preventing TNF-induced angiogenesis might underpin some of the therapeutic benefit of anti-TNF agents in patients with RA.

Incubation of human dermal micro-vasculature endothelial cells (HMVECS) with TNF induced cell migration and upregulated the expression of proangiogenic chemokines. These effects were blocked by co-incubation with the anti-TNF agent certolizumab pegol, in a dose-dependent manner. Furthermore, "we found that TNF increases the expression of a number of angiogenic adhesion molecules in HMVECs," says Koch; "again, their expression was blocked by certolizumab pegol." Thus, myeloid cell adhesion (critical in RA inflammation) to HMVECs or synovial tissue vasculature from patients with RA was prevented.

"Certolizumab pegol ... effectively reverses much of the activity that TNF exerts upon HMVECs" explains Koch. "TNF is very active on HMVECs at very low concentrations, and it was at these levels of TNF that certolizumab pegol was most effective at abrogating TNF activity."

The investigators conclude that anti-TNF agents could block TNF-dependent endothelial cell migration and angiogenesis in RA. In future studies, they plan to use *in vivo* animal models to study the effects of TNF inhibition on blood vessel growth.

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Original article Shu, Q. *et al.* Suppression of endothelial cell activity by inhibition of tumor necrosis factor-α *Arthritis Res. Ther.* doi:10.1186/ar3812