The outcome of granulomatous experimental autoimmune thyroiditis is not associated with the chemokine profile

Severe granulomatous experimental autoimmune thyroiditis (G-EAT) develops in two wild type (WT), DBA-1 and CBA-J mice, and in DBA-1 IFNγ-/- mice. Over time, G-EAT can either progress to fibrosis or spontaneously resolve. To further define the role of IFNγ and chemokines in regulating disease outcome, G-EAT was induced by adoptive transfer of splenocytes activated in vitro with mouse thyroglobulin (MTg) and IL-12, and the outcome was compared among the different strains of mice. Severe G-EAT lesions in IFNγ-/- mice spontaneously resolved by day 35-45. Conversely, G-EAT in WT DBA-1 progressed to atrophy and fibrosis. One group of WT CBA-J mice progressed to resolution the other group of WT CBA-J mice progressed to fibrosis. Expression of cytokines and chemokines was determined by immunohistochemistry and RT-PCR. Thyroids of IFNγ-/- mice expressed no IFNγ, minimal iNOS, TNFα, IP-10, Mig, MCP-1 and CXCR3, but high levels of IL-4 and IL-10. DBA-1WT thyroids expressed high levels of the cytokines IFNγ, iNOS, TNFα and TGFβ, and higher levels of the chemokines IP-10, Mig, MCP-1, and CXCR3, but lower CCR3 than IFNγ-/- mice. Both groups of WT CBA-J mice expressed similarly high levels of IFNγ, IP-10, Mig, MCP-1 and CXCR3. These results suggest that the difference in outcome of G-EAT is not due to the chemokine profile. To account for the difference in outcomes between the two different WT strains, more data should be gathered about the difference between each strain’s cytokine profile.