Contribution of sex steroids and prolactin to the modulation of T and B cells during autoimmunity

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Abstract

In this review we discuss how sex steroids and prolactin affect regulation and responsiveness of B and T cells. Sex hormones exert profound effects on several physiological processes of non- reproductive tissues. In the immune system, several studies with experimental models for SLE have shown a noticeable pro-inflammatory role for ERα, contributing to disease development reflected in proteinuria and renal pathology. On the other hand, ERβ appears to have an anti- inflammatory and immunosuppressive effect. Estrogen/ERα signaling induced an increase of Th17 cells in lymph nodes as well as the expression of its correspondent chemokine receptor CCR6 during collagen induced arthritis acute phase. High levels of anti- DNA antibodies and increased mortality was observed when given high E and prolactin doses to NZB/NZW mice, as compared with mice receiving low E and prolactin doses, or high E and low prolactin doses. Intracellular progesterone receptors have been detected in $TCD4^{+}$ cells but in contrast as observed with ERs, it suppresses T cell dependent responses. Progestagen administration on female NZB/NZW mice decreased anti DNA IgG, improved survival, decreased glomerulonephritis and proteinuria.

Keywords

Autoimmunity, Estrogen, Progesterone, Prolactin, T cells, B cells.