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Abundant CD4+FOXP3+ regulatory T cells fail to suppress the proliferation of T cells in patients with Turner syndrome

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Context

Why Turner syndrome (TS) patients are predisposed to autoimmune disease remains unclear.

Objective

We investigated whether the frequency, phenotype, and suppressive function of $CD4^+FOXP3^+$ regulatory T cells (Tregs) are altered in young TS patients with the 45,X karyotype compared to age-matched controls.

Methods

Peripheral blood mononuclear cells from young TS patients (n = 24, 17.4–35.9 yrs) and controls (n = 29) were stained with various Treg markers to characterize their phenotypes. Tregs sorted for $CD4^+CD25^{bright}$ were co-cultured with autologous $CD4^+CD25^-$ target cells in the presence of anti-CD3 and CD28 antibodies to assess their suppressive function.

Results

TS patients exhibited a higher frequency of $CD4^+FOXP3^+$ Tregs among their lymphocytes (mean 2.06 vs. 1.52%, P = 0.005) and $FOXP3^+$ Tregs among their $CD4^+$ T cells (7.44 vs. 4.19%, P < 0.001) compared to controls. The expression of inhibitory CTLA-4 in the Tregs of TS patients was also significantly higher (mean fluorescence intensity = 214.1 vs. 184.6, P = 0.003). The frequency of Tregs expressing $GITR^+$, $CXCR3^+$, and $CCR4^+CCR6^+$ was comparable between the two groups. However, the ability of Tregs to suppress the in vitro proliferation of autologous $CD4^+CD25^-$ T cells was significantly impaired in TS patients compared to controls (P < 0.05 at a 0.1:1 ratio of Tregs to target cells, P < 0.01 at 0.25:1, 0.5:1, and 1:1).

Conclusions

The Tregs of TS patients could not efficiently suppress the proliferation of autologous effector T cells, despite the abundance of Tregs in the peripheral circulation.

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