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Presentation Abstract

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Title:	Characterization of a neuronal-like GABAergic system in human lymphocytes
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Authors:	***M. J. DE ROSA**, L. R. DIONISIO, C. BOUZAT, M. C. ESANDI; Inst. De Investigaciones Bioquimicas Bahia Blanca, Bahia Blanca, Argentina
Abstract:	GABA is a major neurotransmitter in the central nervous system. Here we identified components of the GABAergic system in human lymphocytes and determined their functional role. By RT-PCR we first detected mRNA of different components of this system in resting and mitogen-activated lymphocytes: i) GAD67, an isoform of the enzyme that synthetizes GABA; ii) VIAAT, the vesicular protein involved in GABA store; iii) GABA transporter (GAT1-2); iv) GABA-T, the enzyme that catabolizes GABA; and v) alpha1 and rho2 subunits that conform ionotropic GABA receptors. In addition we performed immunocytochemistry to detect VIAAT protein and real time PCR to quantify mRNA levels of GABA-T. We observed upregulation of VIAAT and GABA-T upon mitogen stimulation. We also measured the functionality of GABA transporters by measuring uptake of radioactive GABA. The results demonstrate that GABA uptake is significantly increased in activated lymphocytes. To determine if GABA subunits assemble into functional channels, we performed whole-cell recordings in activated lymphocytes. GABA and muscimol, a specific agonist for ionotropic GABA receptors, elicit currents in about 10-15% of the cells. These experiments show that lymphocytes express functional GABA-activated channels. Finally, using [3H]thymidine incorporation, we established that GABA is able to modulate negatively lymphocyte proliferation. Muscimol, similarly to GABA, inhibits lymphocyte proliferation, thus suggesting that this action is mediated by ionotropic GABA receptors. Taken together, our results reveal that lymphocytes have most of the essential components needed to constitute a neuronal-like GABAergic system. Pharmacological modulation of this system may provide new approaches for regulation of T cell response.
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