Regulation of Allergic Contact Sensitivity in Mice: Soluble $\alpha\beta$ T Cell Receptors, Derived via cDNA Transfection and Enzymatic Cleavage from the Surface of T Cells, Protect Contact Sensitivity Effector T Cells from Active T Cell Suppression

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Key Words

Suppressor T cells
Contrasuppressor factor
Soluble TCR
TCR cDNA Transfection

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Using a system in which suppressor T cells actively suppressed contact sensitivity effector T cell adoptive cell transfers, in vitro pre-treatment of the effector cells with soluble T cell receptor (sTCRs) protected the contact sensitivity effector T cells from the active T cell suppression. sTCRs were derived from BWE5147 thymoma cells that were previously transfected with chimeric cDNAs encoding the extracellular domains of the α and β chains of conalbumin-specific TCR from the Th2 DIO CD4⁺ T cell clone that were spliced to the glycosyl-phosphatidylinositol (GPI) membrane anchor sequences of the PI-linked molecule, Thy-1. Treatment of TCR $\alpha\beta$ -transfected cells with PI-specific phospholipase C (PI-PLC) caused release of the transfected TCR in soluble form from the cell surface. sTCR protection from suppression of contact sensitivity effector T cells was not due to PI-PLC contaminating the sTCRs, since treatment of DIO TCR $\alpha\beta$ -expressing BW5147 cells with PI-PLC alone had no effect, and sTCR purified further on anti-TCR β (H57) monoclonal antibody columns preserved contact-sensitivity-assisting activity. Only TCR-Thy-1-cDNA-transfected cells, but not several other cell lines, including cells transfected with the DIO TCR $\alpha\beta$ cDNA, but with the normal transmembrane portion, produced contact-sensitivity-protecting activity. Specific antigen (conalbumin) of the DIO TCR did not interfere with the protecting activity of DIO sTCR. The contact-sensitivity-protecting activity of sTCR was shown to adhere to and be subsequently eluted from anti-TCR α (H28) and anti-TCR β (H57) monoclonal antibody columns. Reduced and separated TCR $\alpha\beta$ chains of DIO sTCR lacked contact-sensitivity-protective activity, but subsequent recombination by covalent joining of both chains and heterodimer formation, via the oxido-shuffling reaction, restored activity.

We concluded that DIO sTCR, derived via cDNA transfection of chimeric TCR Thy-1 constructs, and PI-PLC release from cell surfaces, could protect contact sensitivity effector $\alpha\beta$ T cells from T cell suppression, and thus was contrasuppressive. The contrasuppressive activity was not antigen specific and required both chains of TCR $\alpha\beta$, and their disulfide linkage, to express this activity.

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