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AUTHOR'S CORRECTION

Regulation of the Maintenance of Peripheral T-Cell Anergy by TAB1-Mediated p38α Activation

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Volume 24, no. 16, p. 6957–6966, 2004. In this paper we identified an ~56-kDa protein expressed in anergic $CD4^+$ T cells by Western blotting using anti-TAB1 peptide antibody (N-19; Santa Cruz Biotechnology). This molecule was not present in naïve $CD4^+$ T cells, and we concluded that TAB1 is expressed in anergic and not in naïve $CD4^+$ T cells. However, our recent work indicated that this is not correct. After publication of the paper, we generated monoclonal antibodies (MAbs) specific for recombinant human TAB1 protein to confirm the expression of TAB1 in naïve and anergic $CD4^+$ T cells. Contrary to our expectations, these MAbs identified an ~70-kDa molecule in naïve $CD4^+$ T cells at levels similar to those in anergic $CD4^+$ T cells. Subsequent experiments have shown that the ~70-kDa protein is the TAB1 molecule, because these antibodies detect a molecule of the same size in COS7 cells transfected with the expression vector containing human TAB1 cDNA and not with the control vector and, furthermore, this band is specifically absent in cells lacking the TAB1 gene.

Therefore, the ~56-kDa molecule that we identified in anergic $CD4^+$ T cells by anti-TAB1 antibody (N-19) was not TAB1. The expression of TAB1 was shown in Fig. 2 of the paper, and we retract these data. We apologize for releasing and misinterpreting the immature data. While we are unable to conclude that the activation of p38α in anergic $CD4^+$ T cells is regulated by up-regulation of TAB1 protein, the main conclusions that anergic $CD4^+$ T cells show enhanced p38 mitogen-activated protein kinase activity and that signals involving TAB1 could be a critical regulatory point in maintaining $CD4^+$ T-cell anergy by activating p38 remain unchanged.