

Claudia Traidl-Hoffmann, Valentina Mariani, Hubertus Hochrein, Kathrin Karg, Hermann Wagner, Johannes Ring, Martin J. Mueller, Thilo Jakob, and Heidrun Behrendt
 Vol. 201, No. 4, February 21, 2005. Pages 627–636.

The authors regret that an inaccurate version of Fig. 4 appears in the original article. The correct version of Fig. 4 and its legend appear below.

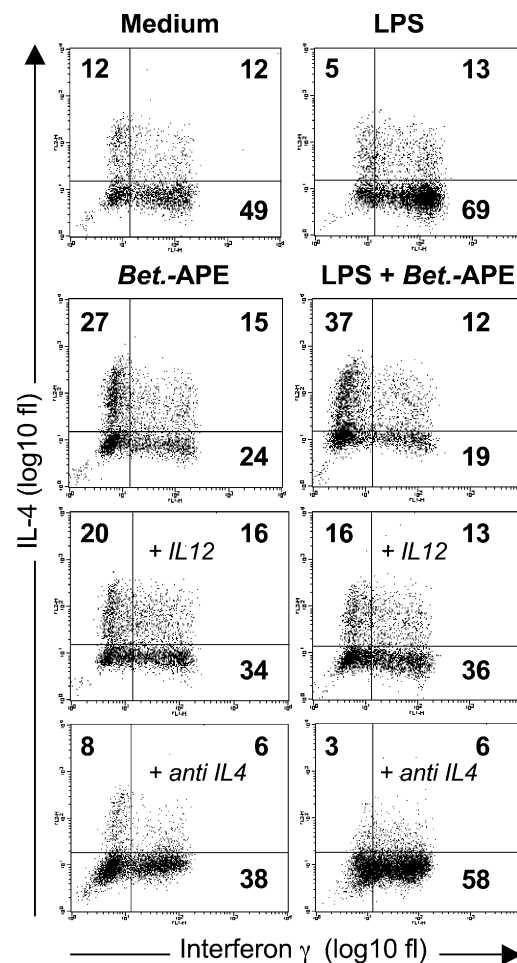


Figure 4. DCs matured in the presence of *Bet.-APE* display reduced Th1- and increased Th2-polarizing capacity. DCs were left untreated or stimulated with *Bet.-APE* (3 mg/ml) in the presence or absence of LPS (100 ng/ml). After 24 h DCs were washed and cocultured with CD4⁺CD45RA⁺ allogenic T cells (DC/T cell ratio 1:4) that were expanded for 12 d in the presence of IL-2. T cell polarization was determined by analyzing intracellular IFN- γ and IL-4 accumulation via flow cytometry after restimulation with PMA and ionomycin in the presence of brefeldin A during the last

2 h of stimulation. To address, if the *Bet.-APE*-dependent Th2 polarization could be reverted by exogenous IL-12, hrIL-12 (10 ng/ml) was added at the beginning of the coculture of *Bet.-APE*/LPS-treated DCs and T cells. Representative experiment of $n = 3-6$ (compare Table I). To explore the role of IL-4 in the Th2 polarization induced by *Bet.-APE*-treated DCs, IL-4-neutralizing antibodies (10 μ g/ml) were added at the beginning of the DC/T cell coculture. Representative experiment of $n = 3$.