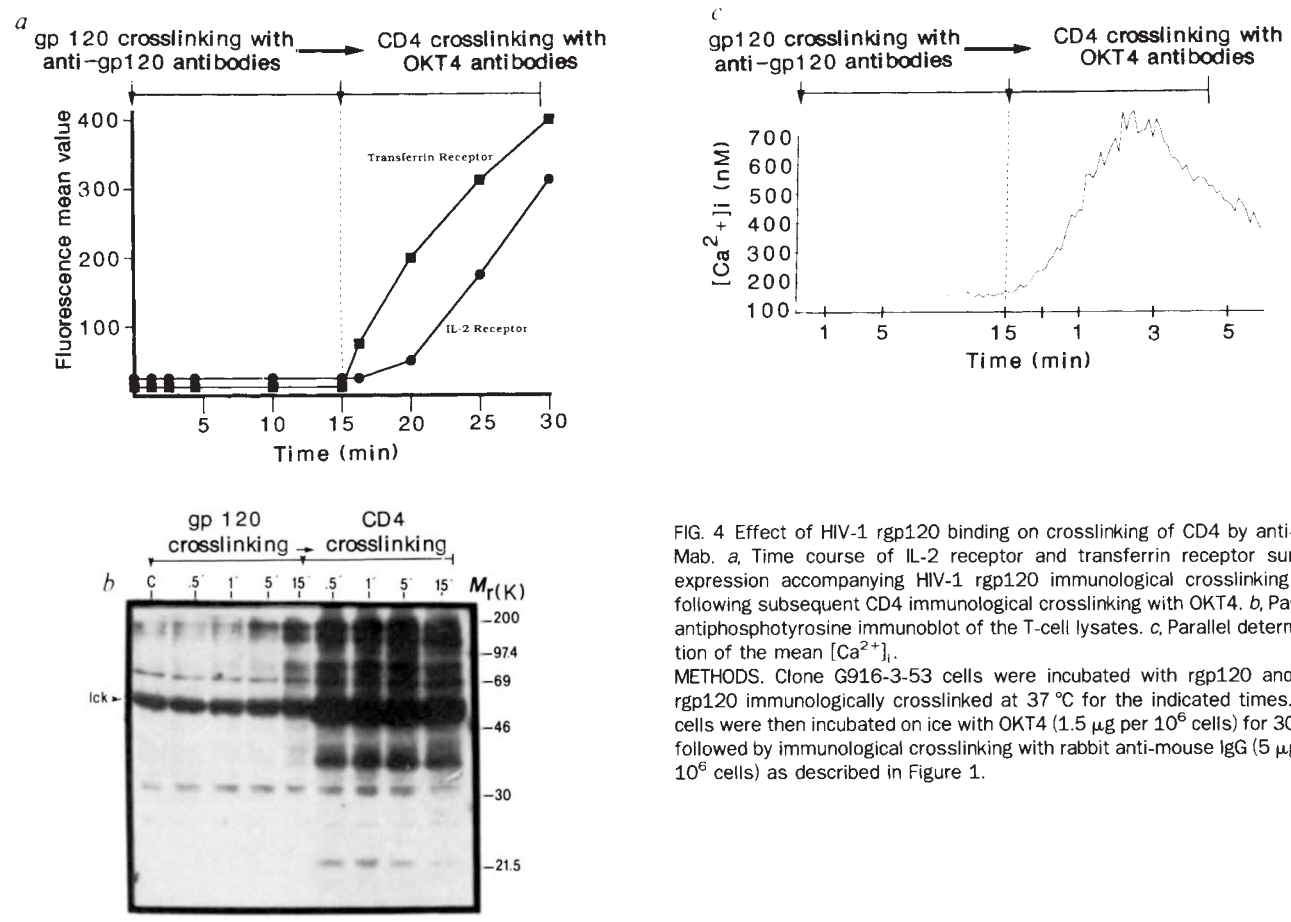

FIG. 4 Effect of HIV-1 rgp120 binding on crosslinking of CD4 by anti-CD4 Mab. *a*, Time course of IL-2 receptor and transferrin receptor surface expression accompanying HIV-1 rgp120 immunological crosslinking and following subsequent CD4 immunological crosslinking with OKT4. *b*, Parallel antiphosphotyrosine immunoblot of the T-cell lysates. *c*, Parallel determination of the mean $[Ca^{2+}]_i$.
METHODS. Clone G916-3-53 cells were incubated with rgp120 and the rgp120 immunologically crosslinked at 37 °C for the indicated times. The cells were then incubated on ice with OKT4 (1.5 μg per 10^6 cells) for 30 min followed by immunological crosslinking with rabbit anti-mouse IgG (5 μg per 10^6 cells) as described in Figure 1.

not inhibit CD4-mediated signal transduction events stimulated following CD4–CD4 interactions.

Our results show that absorption of HIV-1 or binding and crosslinking of HIV-1 gp120 does not stimulate the human $CD4^+$ T lymphocyte signal transduction pathways that are triggered upon surface CD4 engagement mediated by anti-CD4 antibodies. These results support the finding that non-proliferating T cells can be infected with HIV[8–11] and indicate that early HIV-1 interactions with quiescent T cells may not generate the types of stimulatory signals that have been established as requirements for virus production. Although in agreement with the data of Mittler and Hoffman[12], our results differ from other reports demonstrating that CD4-dependent gp120 binding to T cells[13,14] results in increased Ca^{2+} mobilization and stimulation of other intracellular activation signals. Increased Ca^{2+} mobilization has also been reported following CD4-independent gp120 binding to cultured rodent retinal ganglion cell neurons[15]. An explanation for the discrepancies is not clear at present. □

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ERRATUM

Targets of homeotic gene control in *Drosophila*

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IN the above article, the first line of the immunopurification strategy shown in Fig. 1 should have read: Nuclei from 3–15 h embryos.