# Assessment of the contribution of HIV patient serum antibodies that recognize lymphocyte antigens to the inhibition of the HIV-1 envelope-dependent membrane fusion

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Pathogenicity of HIV-1 strains is related to their efficiency to induce fusion of CD4+ T cells. Membrane fusion depends on the interaction of the HIV-envelope (Env) with CD4 and coreceptor molecules on human T lymphocytes. In addition to virus receptors, other adhesion/signaling molecules on infected and target cells and virus particles can enhance fusion. The presence of anti-lymphocyte autoantibodies (ALAs) in HIV patients' serum suggests their possible contribution to the inhibition of Env-mediated membrane fusion. We initially determined the binding of antibodies from the sera of 38 HIV-1 infected individuals, to human CD4+ Jurkat T cells. Seventy-four and 60% of sera were positive for binding of IgG and IgM to Jurkat cells, respectively. We evaluated the contribution of ALAs to the patient's serum activity against HIV-envelope dependent cell-cell fusion. The effect of sera on the fusion of CD4+ with Env+ Jurkat cells was tested before and after adsorption on CD4- negative Jurkat cells to remove ALAs. Analysis of the effect of sera on the fusion of CD4+ with HIV-1 Env-expressing Jurkat cells showed a variable degree of inhibition by serum samples. Inhibition of fusion decreased in 58% of serum samples after adsorption, indicating that ALAs contributed to fusion inhibition in these sera. Fusion increased in 31.6% and did not change in 10.5% of other serum samples after adsorption. The extent of the contribution of ALAs to the effect of sera on fusion was highly variable, with an average of 33%. Thus, fusion inhibitory ALAs other than anti-CD4 antibodies may contribute significantly to the inhibition of Env-mediated cell-cell fusion. Only detection of fusion inhibitory ALAs, but not total ALA levels, associated with the patients' low plasma viral loads, suggesting that they may participate in virus containment during HIV-1 infection. Fusion inhibitory ALAs may be relevant to the antiviral humoral immune response in a substantial fraction of HIV-infected patients.