



Differential modulation of human lactoferrin activity against both R5 and X4-HIV-1 adsorption on epithelial cells and dendritic cells by natural antibodies. ✓

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In **Results**, in sentence 16 under the heading *A Cullin-based ubiquitin ligase pathway is involved in host cell-mediated IRF-3 degradation following SeV infection*, reference to CREB coactivator is incorrect. In sentence ten, under the heading *Degradation of IRF-3 is dependent of the TBK1/IKKi-signaling pathway*; reference to RNA interference silencing technology is incorrect. The corrected sentences are shown below.

Interestingly, this increase in the stability of the hyperphosphorylated forms of IRF-3 was also associated with a sustained activation of IRF-3 as verified by the presence of dimers or its association to CREB binding protein (CBP) coactivator after infection with SeV (Fig. 3E).

We next directly examined the contribution of the IKK-related kinases in IRF-3 degradation by first using RNA interference (RNAi) technology.

Saidi, H., J. Eslaphazir, C. Carbonneil, L. Carthagena, M. Requena, N. Nassreddine, and L. Belec. 2006. Differential modulation of human lactoferrin activity against both R5 and X4-HIV-1 adsorption on epithelial cells and dendritic cells by natural antibodies. *J. Immunol.* 177: 5540–5549.

The second author's last name is misspelled. The correct name is Jobin Eslaphazir.