RETRACTION

doi:10.1038/nature09254

# Cellular APOBEC3G restricts HIV-1 infection in resting CD4$^+$ T cells

Ya-Lin Chiu, Vanessa B. Soros, Jason F. Kreisberg, Kim Stopak, Wes Yonemoto & Warner C. Greene

*Nature* 435, 108–114 (2005)

The authors wish to retract this Letter because they have been unable to reproduce the experiments demonstrating APOBEC3G functions as a post-entry restriction factor in resting CD4 T cells. Two recent studies[1,2] have also challenged this conclusion. However, the authors report that several findings described in this Letter are reproducible, including: (1) low-molecular-mass (LMM) forms of APOBEC3G predominate in resting CD4 T cells and monocytes; (2) LMM APOBEC3G is recruited in high-molecular-mass (HMM) APOBEC3G RNA–protein complexes after activation of T cells or differentiation of monocytes into macrophages; (3) HMM APOBEC3G does not exhibit enzymatic activity; (4) when HMM APOBEC3G is treated with RNase A, LMM APOBEC3G forms are generated and enzymatic activity is restored; and (5) Vif assembles with and polyubiquitylates APOBEC3G present in HMM complexes. Nevertheless, because a central finding of the paper cannot be replicated either internally or externally despite repeated attempts, the authors request that *Nature* retract the paper and regret any confusion that may have been created by the paper's publication.

1. Kamata, M., Nagaoka, Y. & Chen, I. S. Y. Reassessing the role of APOBEC3G in human immunodeficiency virus type 1 infection of quiescent CD4+ T-cells. *PLoS Pathogens* **5**, e1000342 (2009).
2. Santoni de Sio, F. R. & Trono, D. APOBEC3G-depleted resting CD4$^+$ T cells remain refractory to HIV1 infection. *PLoS ONE* **4**, e6571 (2009).