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arrest and apoptosis in HeLa cells. It has been shown that two human proteins, APOBEC3G (A3G) and APOBEC3F (A3F), can block replication of human immunodeficiency virus type 1 (HIV-1) in primary human CD4+ T cells and/or monocytic cells. Here, we show that A3G- or A3F-restriction of HIV-1 depends on A3G or A3F, but not A3A. This is consistent with the failure of APOBEC-deficient mice to control HIV-1 infection. Using transiently expressed A3G or A3F, we show that both proteins induce G2/M arrest and trigger apoptosis in HeLa cells. Induction of G2/M arrest requires formation of the A3G-A3F heterodimer and does not involve activation of the proteasome. Activation of caspases by A3G or A3F does not correlate with cytochrome c release or with the loss of mitochondrial membrane potential, indicating that the mechanism of A3-dependent apoptosis is not mediated by mitochondrial release of cytochrome c. We identified a region in the C-terminal part of A3G that induces G2/M arrest and apoptosis. A3F and A3G-APOBEC3C chimera proteins were not toxic to HeLa cells. }f(x)\leq 0\quad\text{for all }x\in \mathbb{R}.\$ The function \$f(x) 82138339de

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