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# The enhancement of HIV-1 infectivity by Nef depends on dynamin 2

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Nef is a virulence factor of HIV-1 and other primate lentiviruses that is crucial for rapid progression to AIDS. Nef modulates the activation state of infected T cells and macrophages, and induces the downregulation of the viral receptor CD4 and of MHC class I molecules. Additionally, Nef increases the intrinsic infectivity of HIV-1 progeny virions by an unknown mechanism. We now show that dynamin 2 (Dyn2), a key regulator of vesicular trafficking, is a binding partner of Nef that is required for its ability to increase viral infectivity. Dominant-negative Dyn2 or the depletion of Dyn2 by small interfering RNA potently inhibited the effect of Nef on HIV-1 infectivity. In Dyn2-depleted cells, this function of Nef could be rescued by ectopically expressed Dyn2 but not by Dyn1, a closely related isoform that does not bind Nef. In contrast, Dyn2 is not specifically required for the downregulation of CD4 or MHC class I molecules by Nef. These findings suggest a functional link between the infectivity enhancement activity of Nef and dynamin-dependent membrane trafficking events.