Figure S1. Time course of gp120 effects on acetylcholine-stimulated currents in SH-SY5Y cells. (A) Whole-cell representative current traces recorded from either control SH-SY5Y cells or cells treated with 1500 nM gp120 for 1, 24, or 48 hours. (B) The average current density for control SH-SY5Y cells was 19.7 ± 2.8 pA/pF (*n* = 14). The average current density for cells exposed to gp120 for 1, 24, and 48 hours was 31.9 ± 5.5 pA/pF (*n* = 10), 39.3 ± 8.6 pA/pF (*n* = 14), and 35.6 ± 6.9 pA/pF (*n* = 9), respectively. Currents were recorded as described for Figure 1. *, p-value < 0.05

Figure S2. Up-regulation of the α7-nAchR requires activation of MEK. (A) Whole-cell representative current traces recorded from SH-SY5Y cells, cells treated with SDF1, or cells treated with SDF1 and PD98059. (B) The average current density for SDF1-treated cells was 107.1 ± 22.6 pA/pF (n = 12) and was 50.1 ± 13.1 pA/pF (n = 13) for cells treated with SDF1 and SDF1 + PD98059.

Figure S3. Real-time PCR and Western blot analysis of α7-nAChR in hippocampus of gp120-transgenic mice. No difference in α7-nAChR mRNA (A) or protein levels (B) was detected in the hippocampus of gp120 transgenic mice as compared with WT (n = 3).

Supplemental videos. Ca^{2+} mobilization in SH-SY5Y cells. HIV-gp120 treated SH-SY5Y cells stimulated with ACh (VS2) present larger calcium mobilization when compared to control cells (VS1). The specific α7-nAchR antagonist MLA inhibits Ca^{2+} mobilization in response to ACh (VS3 and VS4), showing that the change in intracellular [Ca^{2+}] requires this receptor.