

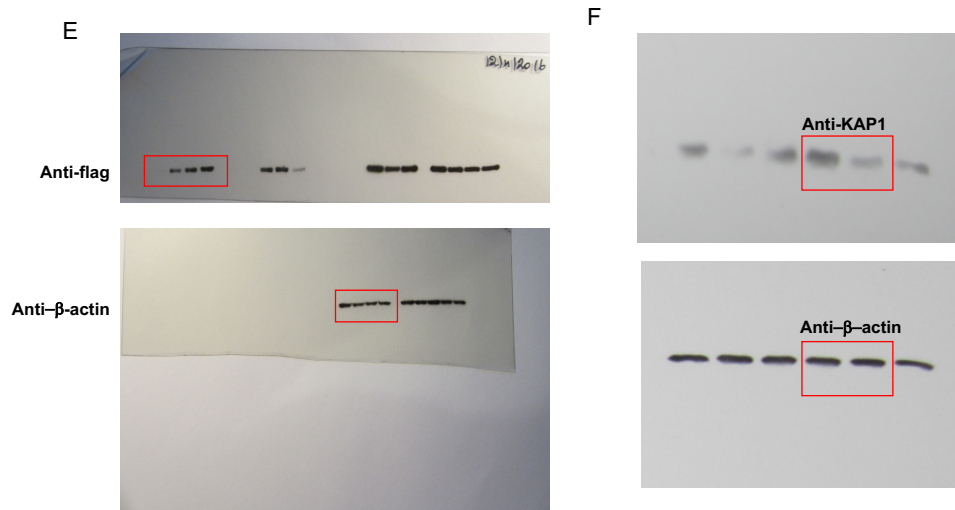
Inhibition of HIV-1 gene transcription by KAP1 in myeloid lineage

Amina AIT AMMAR^{1,2,3}, Maxime BELLEFROID³, Fadoua DAOUAD¹, Valérie MARTINELLI⁴, Jeanne VAN ASSCHE¹, Clémentine WALLET¹, Anthony RODARI³, Marco DE ROVERE¹, Birthe FAHRENKROG⁴, Christian SCHWARTZ¹, Carine VAN LINT^{3*}, Virginie GAUTIER^{2*} and Olivier ROHR^{1*}

1. Université de Strasbourg, UR 7292 DHPI, FMTS, IUT Louis Pasteur, 1 Allée d'Athènes 67300 Schiltigheim, France
2. Center for Research in Infectious Diseases (CRID), School of Medicine and Medical Science (SMMS), University College Dublin (UCD), Dublin, Ireland
3. Service of Molecular Virology, Institute for Molecular Biology and Medicine (IBMM), Université Libre de Bruxelles (ULB), Gosselies, Belgium
4. Laboratory Biology of the Nucleus, Institute for Molecular Biology and Medicine, Université Libre de Bruxelles, Charleroi 6041

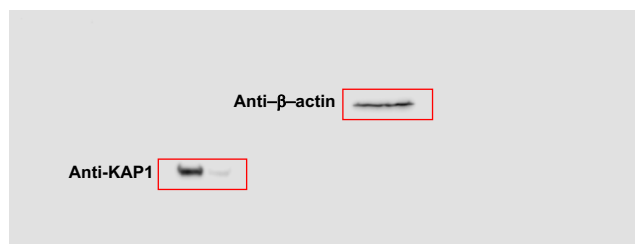
Supplementary figure 1

Figure 1



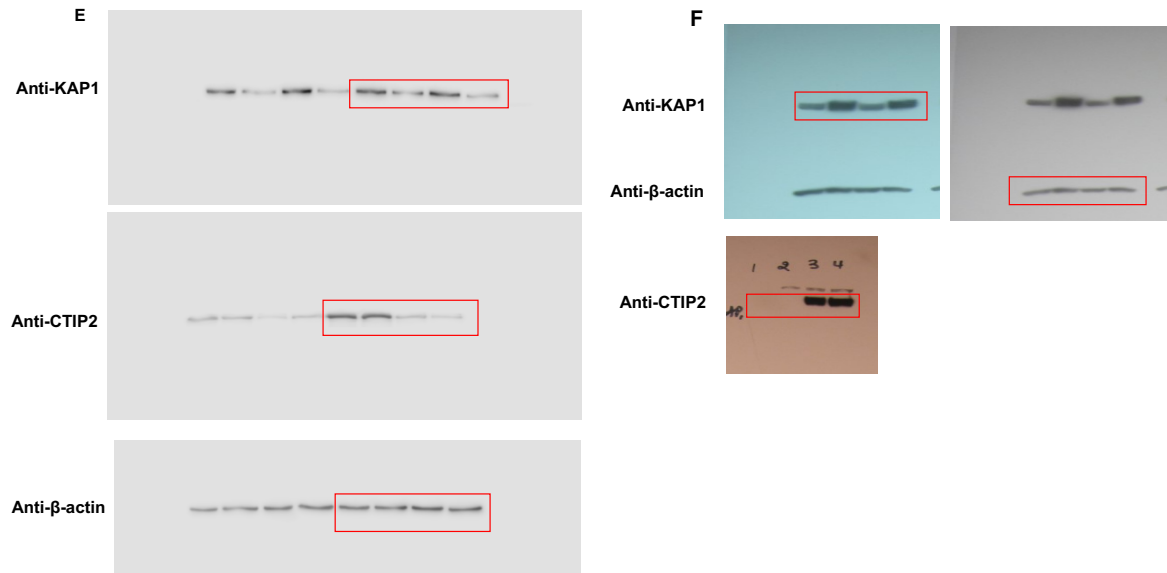
Supplementary figure 2

Figure 3A

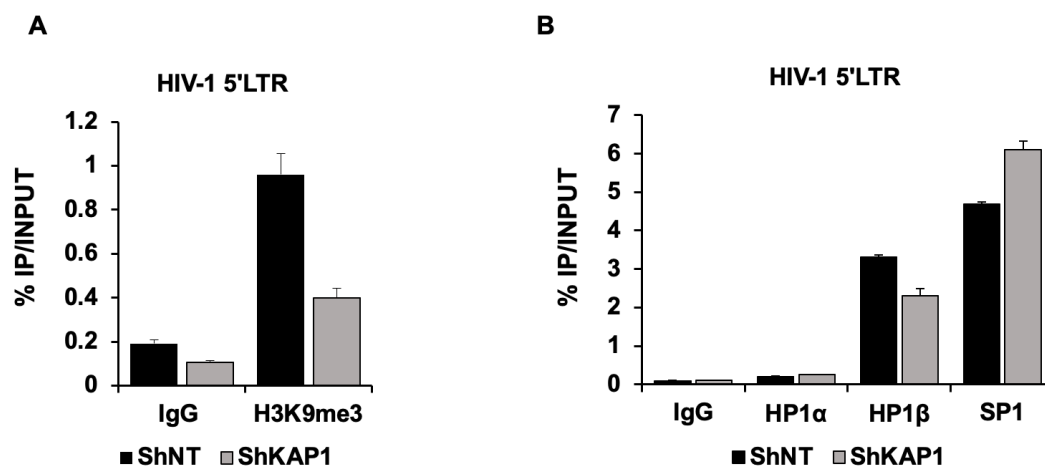


Supplementary figure 3

Figure 4



Supplementary figure 4

**KAP1 depletion leads to reduced heterochromatin marks at the HIV-1 promoter**

(A, B) The HIV-1 infected monocytic THP89 cells stably transduced either with non-targeting shRNA (indicated as shNT) or shKAP1 were subjected to ChIP-qPCR experiments using the indicated antibodies and the primers targeting the HIV-1 5' LTR. Values are presented as percentages of immunoprecipitated DNA compared to the input DNA (% IP/INPUT) and are representative of biological duplicates.

Supplementary figure 5

Figure 5B



Figure 5C

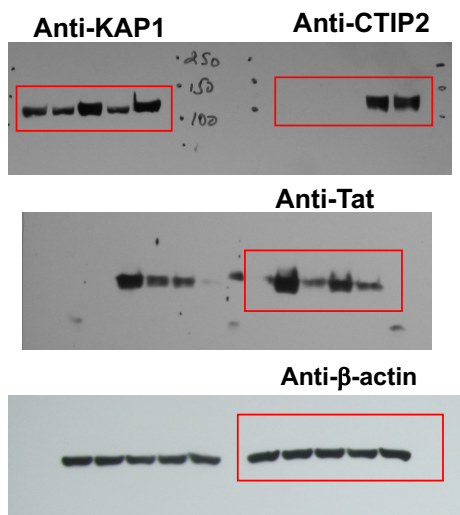
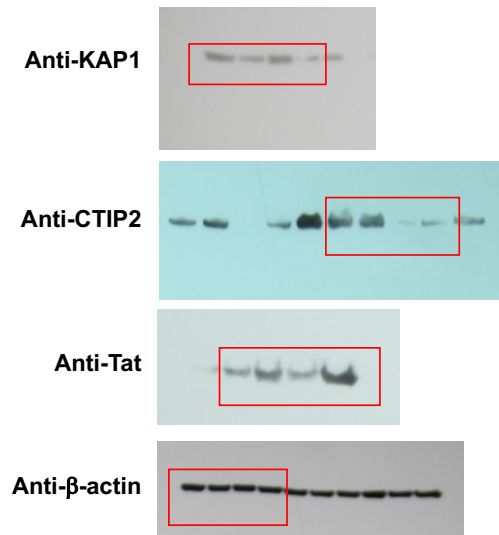


Figure 5D



Supplementary figure 6

Figure 6A

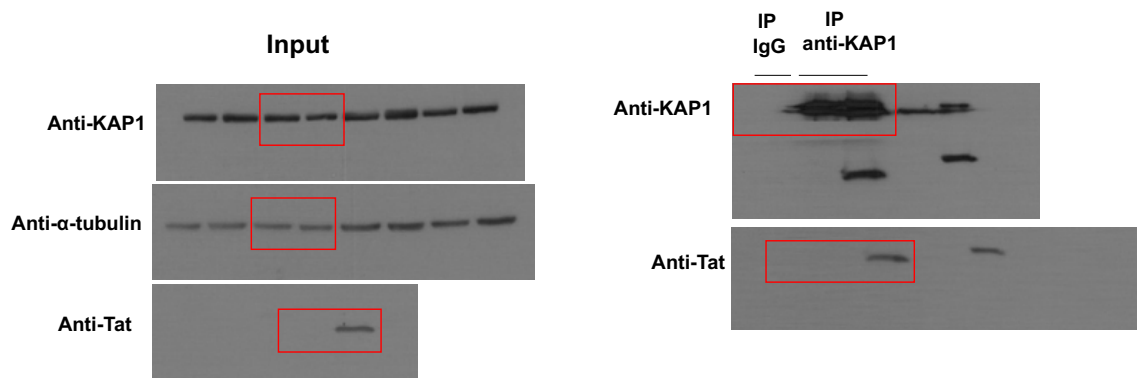
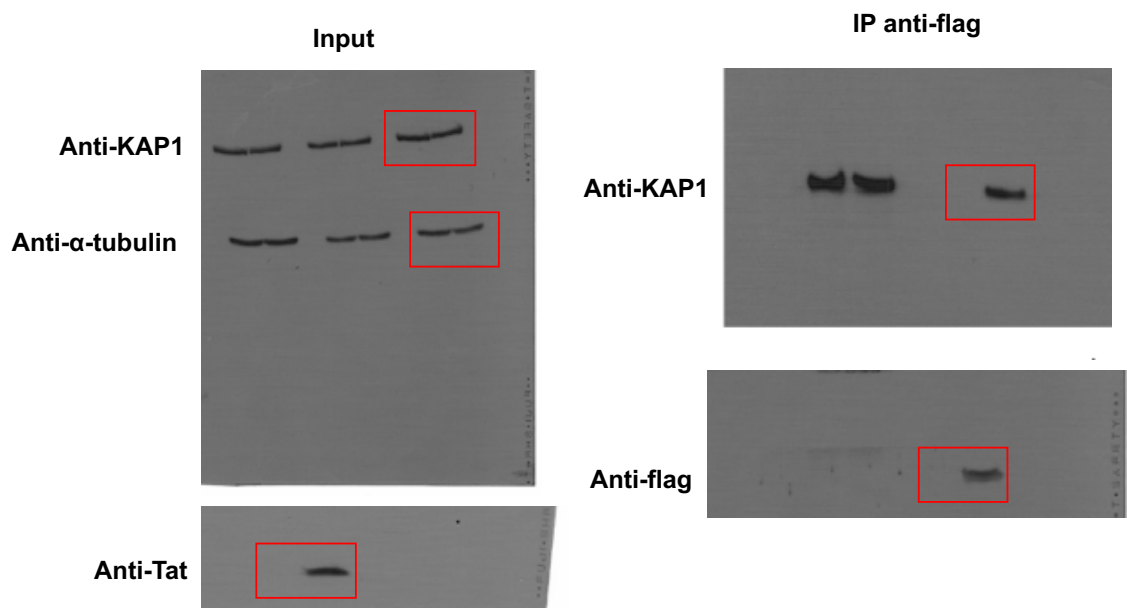


Figure 6B



Supplementary figure 7

Figure 7A

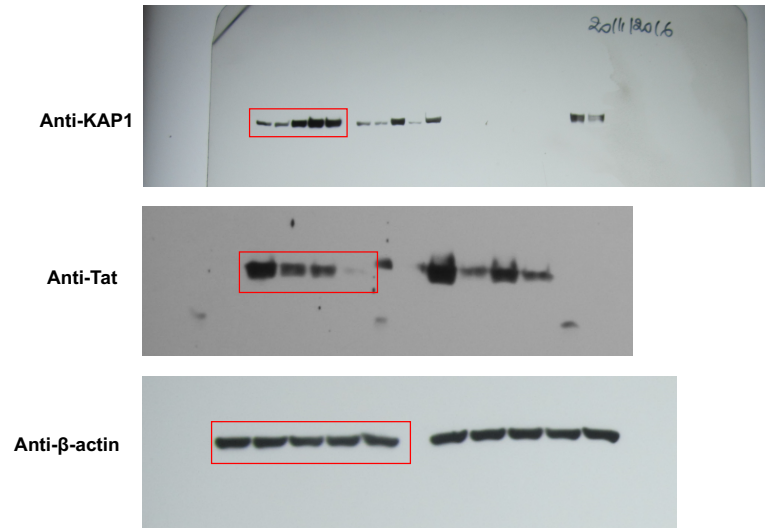


Figure 7B

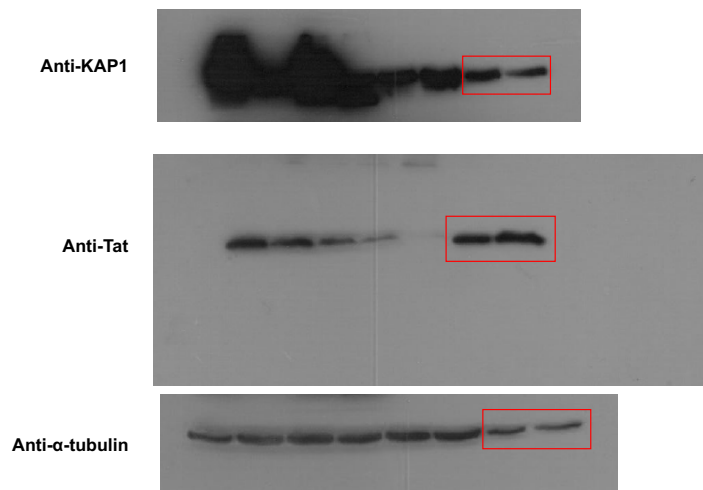


Figure 7D

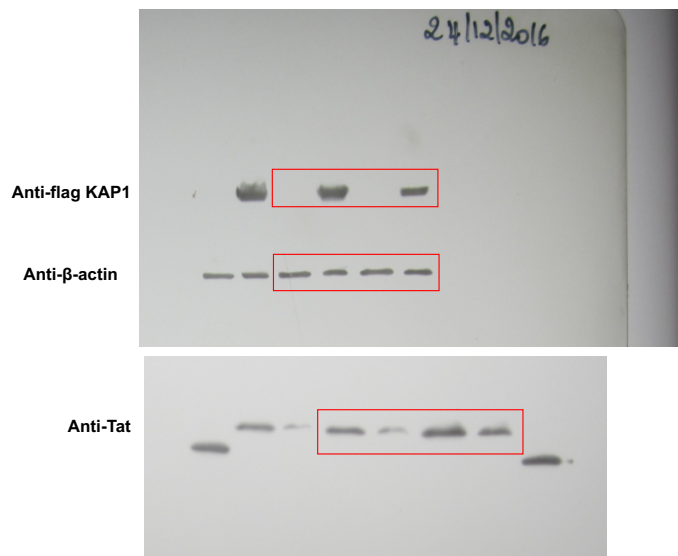
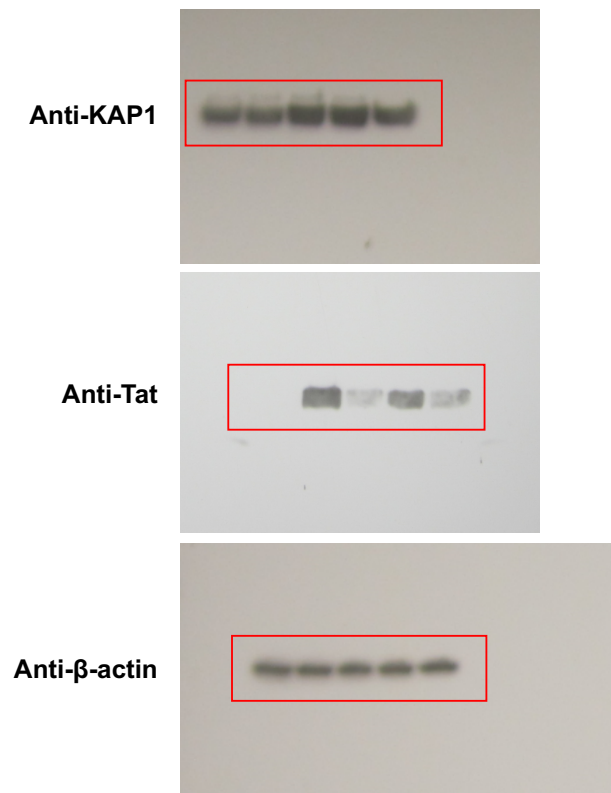
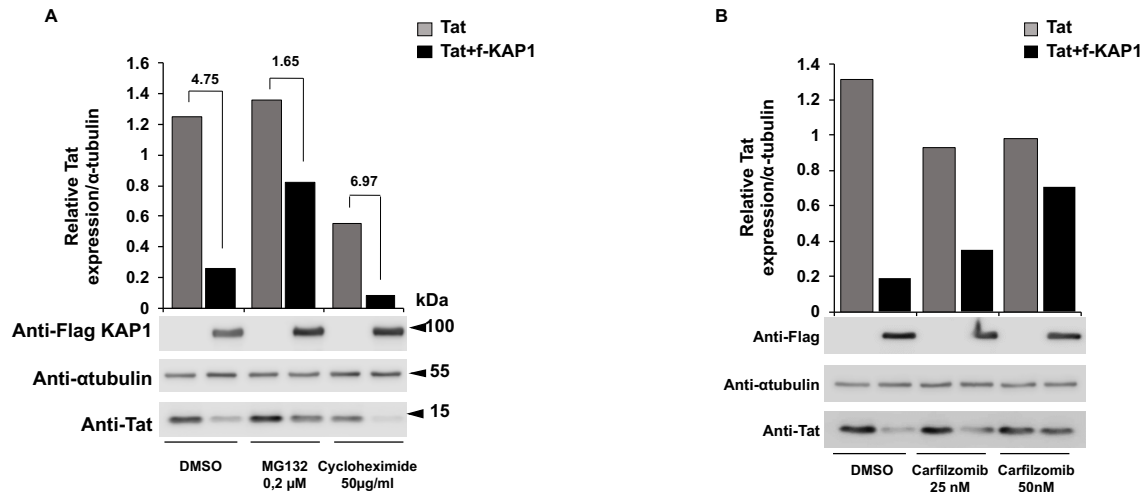


Figure 7F



Supplementary figure 8

**Inhibition of protein synthesis does not impact proteasomal degradation of Tat by KAP1**

HEK cells cultured in a 6-well plate were transfected with expression vectors encoding Tat alone or in combination with flag-KAP1 (A, B). 4h post-transfection, the cells were treated with 0.2 μ M of MG132 for 20 hours or 50 μ g/ml cycloheximide for 4h (A). 4h post-transfection the cells were treated with either 25 nM or 50nM of carfilzomib for 20 hours (B). DMSO treated cells were used as control (A, B). 24h post-transfection cells were lysed, and total protein extracts were analyzed by western blot for the presence of Tat and KAP1. Tat expression levels were normalized to α -tubulin expression, using image J software. The results are representative of two independent experiments.

Supplementary figure 9

Figure 8A

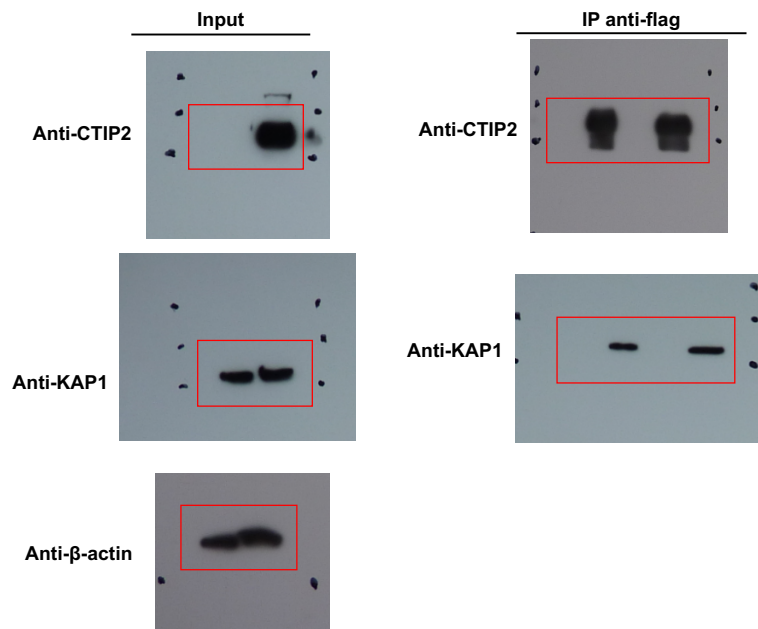


Figure 8B

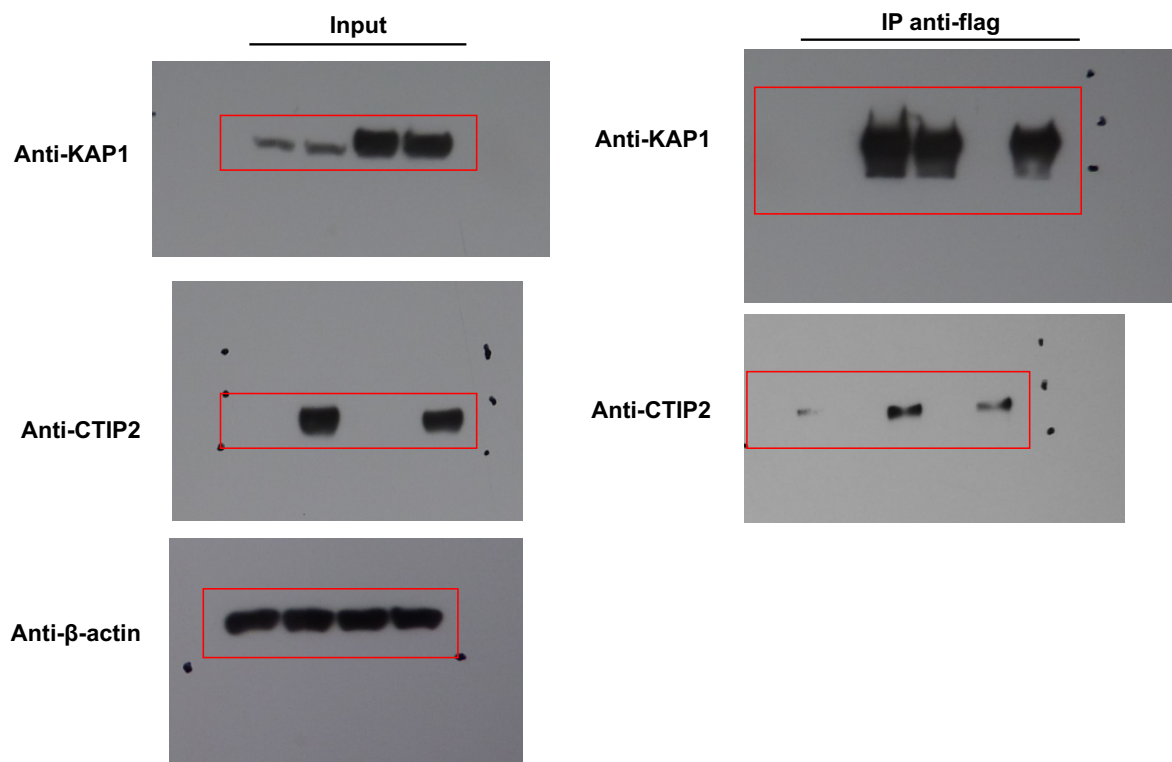


Figure 8C

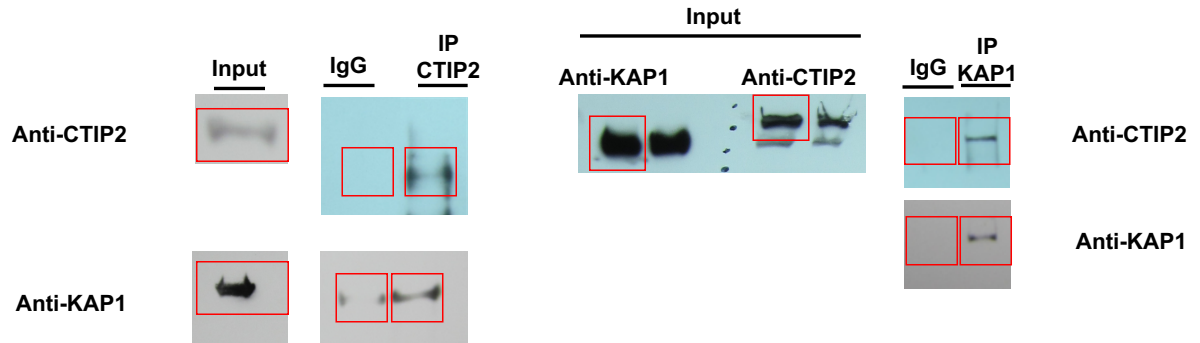


Figure 8D

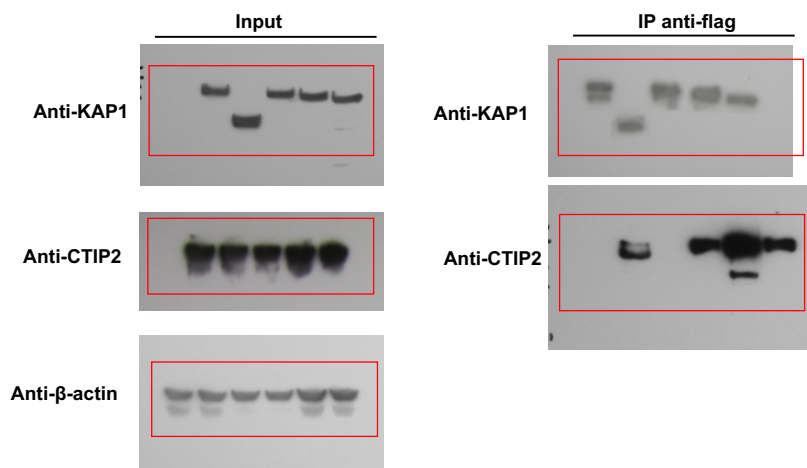


Figure 8E

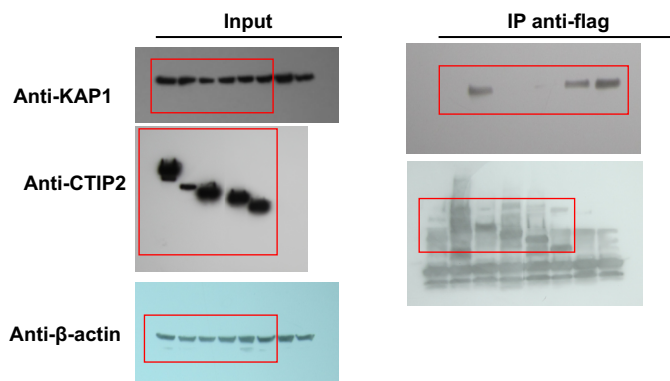


Figure 8G

