

Cigarette smoke promotes HIV infection of primary bronchial epithelium and additively suppresses CFTR function.

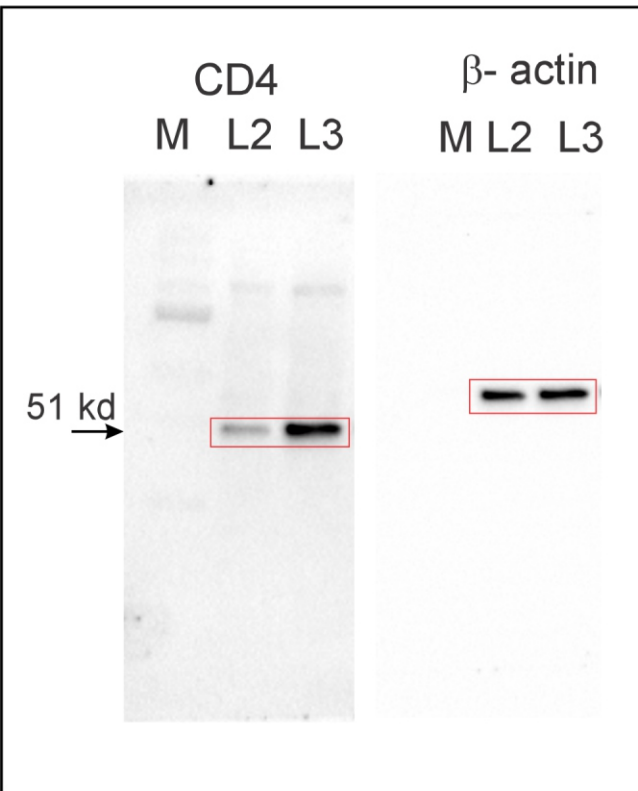
Chinnapaiyan S¹., Dutta R¹., Bala J¹., Parira T¹., Agudelo M¹., Nair M¹., and Unwalla HJ¹ *.

* To whom correspondence should be addressed.

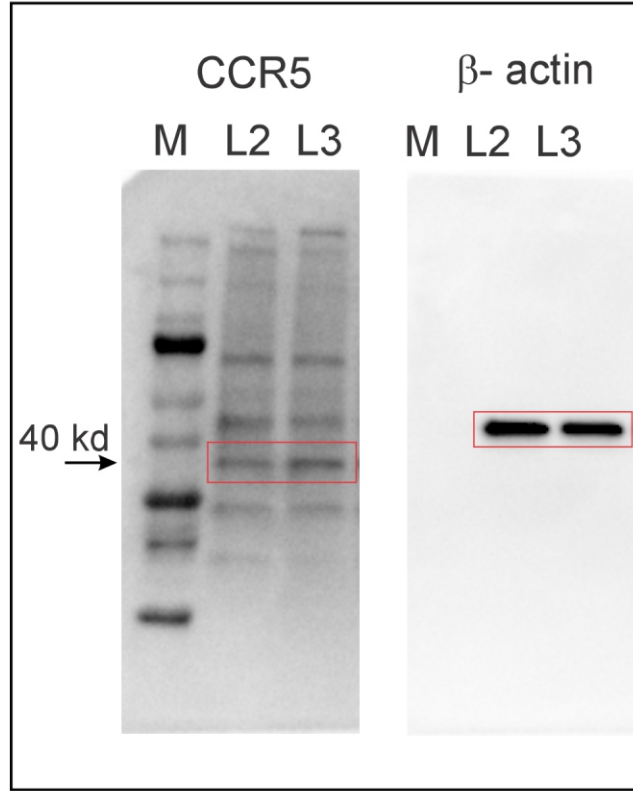
Hoshang Unwalla, PhD, Florida International University, Department of Immunology, Institute of Neuroimmune Pharmacology, 11200 SW 8th street NW 10th Ave., AHC-1 # 421, Miami, FL 33199. Tel.: 305-348-3442; Fax: 305-243-6992; email: hunwalla@fiu.edu

1. Department of Immunology, Institute of Neuroimmune Pharmacology, Herbert Wertheim College of Medicine, Florida International University. Miami, FL

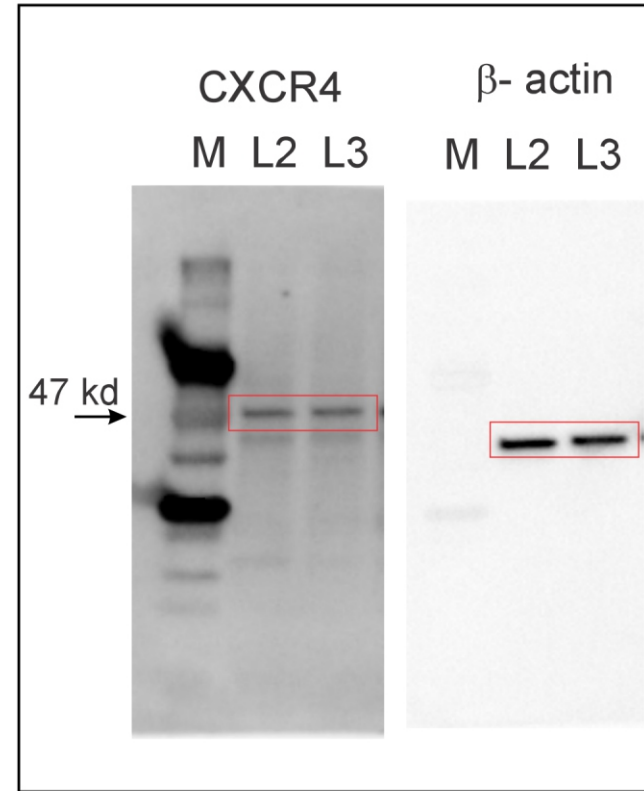
a)



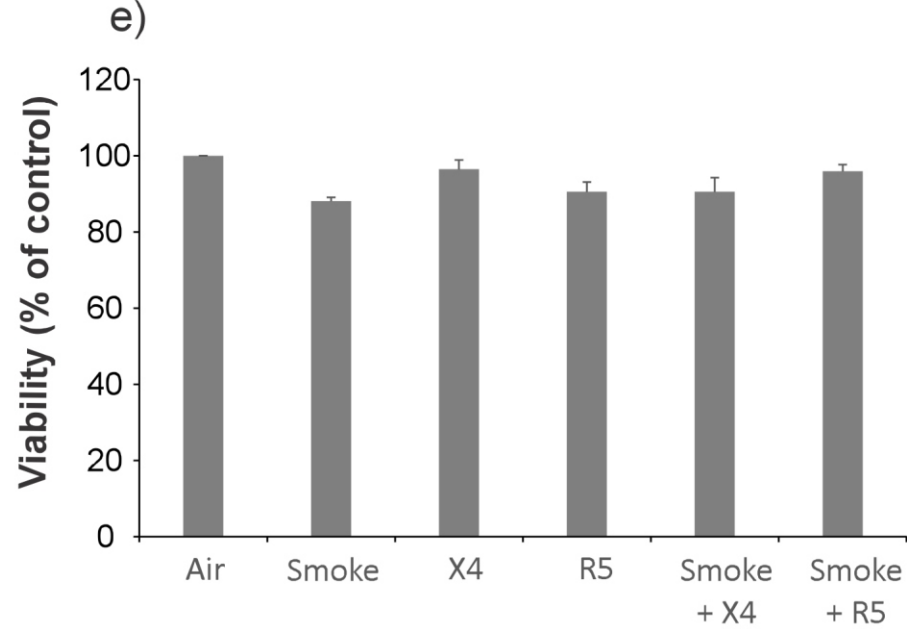
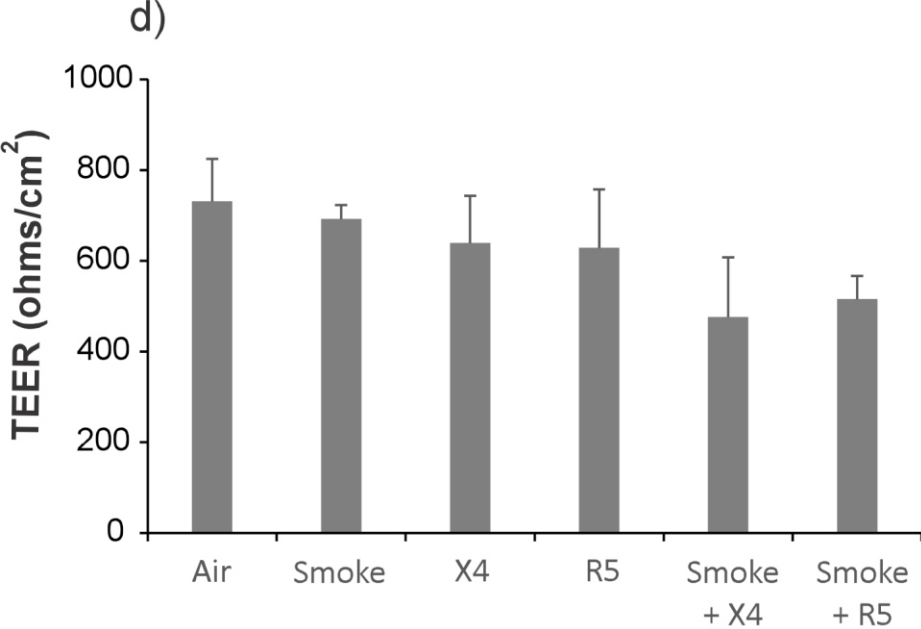
b)



c)



Full length western blot images. Panel a) CD4; Panel b) CCR5, Panel c) CXCR4.
 M: Protein marker; Lane 2: Air; Lane 3: Smoke; Red color highlighted bands were used for our manuscript.



Panel d: Transepithelial electrical resistance (TEER) was measured following chronic exposure to HIV and/or CS. NHBE ALI cultures exposed to HIV and CS did show a trend towards a decreased TEER. Panel e: Cell viability assay demonstrates no significant change in viability in HIV or smoke exposed cells from Air exposed controls with the smoke and HIV p24 dose used.