

Bi 1 Extra Credit Assignment:

For a maximum of 3 extra points on your overall grade.

For this assignment, you are asked to watch webcast lectures from the Conference on Retroviruses and Opportunistic Infections (CROI). You will need a computer with sound and Adobe Flash plug-in to view these presentations. Question 1 is based on a talk from the 2008 CROI meeting [www.retroconference.org/2008] while questions 2 and 3 are from the 2009 CROI meeting [www.retroconference.org/2009].

1. Watch the plenary talk by **Paul Bieniasz** from the 2008 CROI conference in Boston, MA. The talk is entitled ***New Insights into Retrovirus-Host-cell Interactions*** (Wednesday, February 6th, 2008) and can be found at the following website:
<http://app2.capitalreach.com/esp1204/servlet/tc?c=10164&cn=retro&e=8064&m=1&s=20376&&espmt=2&mp3file=8064&m4bfile=8064&br=80&audio=false>
 - a. Name the three retrovirus replication inhibitors (restriction factors) found in host cells, as presented in the talk.
 - b. One year after this talk was given, Klaus Strebel at the NIH established that tetherin is not present on the envelope surface of HIV(del Vpu) virions budding from so-called "non-permissive" cells. Considering that tetherin was shown to inhibit HIV, Ebola and Herpes Simplex Virus (HSV) budding, propose a mechanism explaining how tetherin functions to tether viral particles together.
 - c. Describe a novel therapeutic strategy/approach that could be used towards curing HIV using the information presented in this talk and in class.

2. Watch the plenary talk by **Daniel Douek** from the 2009 CROI conference in Montréal, Canada. The talk is entitled ***Immune Effects at HIV-infected Mucosal Surfaces*** (Monday, February 9th, 2009) and can be found at the following website:
<http://app2.capitalreach.com/esp1204/servlet/tc?c=10164&cn=retro&e=10647&m=1&s=20415&&espmt=2&mp3file=10647&m4bfile=10647&br=80&audio=false>
 - a. How does HIV "create" its own target cell-- the activated CD4+ T cell-- in the context of the talk?
 - b. Why don't SIV infections in Sooty Mabebeys or African Green Monkeys progress to AIDS? Why do they have the same viral loads to pathogenic SIV/HIV infections? (SIV, or Simian Immunodeficiency Virus, is the monkey equivalent of HIV)
 - c. Describe a novel therapeutic strategy/approach that could be used towards curing HIV using the information presented in this talk and

in class.

3. Watch the plenary talk by **Michael Worobey** from the 2009 CROI conference in Montréal, Canada. The talk is entitled ***Local Origin and Global Spread of Immunodeficiency Viruses*** (*Wednesday, February 11th, 2009*) and can be found at the following website:
<http://app2.capitalreach.com/esp1204/servlet/tc?c=10164&cn=retro&e=10667&m=1&s=20415&&espmt=2&mp3file=10667&m4bfile=10667&br=80&audio=false>
- a. According to the talk, when did HIV-1 group M emerge? From which West-African country does HIV-1 group M originate? In which West-African country did the first cross-species transmission occur?
- b. In epidemiological terms, what is the average number of secondary infections relative to primary infections if $R_0 = 25$?
- c. How will we (scientists; society; humanity) drive $R_0 < 1$ for HIV? Describe 3 feasible public health policies, research goals, or other organizational measures that could be implemented to end the current global HIV pandemic.