

Oct. 16, 2008 MCB532

Class questions for "Wave-like Spread of Ebola Zaire"
" PLoS Biol. 2005 Nov;3(11):e371

What is the hypothesis?

What is the alternative hypothesis that they are trying to disprove.

What is their point about viruses with restricted diversity such as seen with Ebola Zaire as opposed to viruses with lots of diversity?

What is their point about independent outbreaks versus spatial trends of the outbreak?

What is their point about the relationship of the isolates from Yambuku? Follow-up question: what did they find, and how is it consistent with their hypothesis?

Are they arguing that there is a reservoir for Ebola, or that there isn't?

Why are they talking about dN and positive selection?

What is really cool about Figure 3?

What is their evidence that Ebola spread by serial local contacts?

What is significant about a change of direction at Boue?

Why doesn't it spread in all directions equally?

What do you think is going on?

What is great about this paper?

October 28, 2008 MCB532

Class questions for "Divergent TLR7 and TLR9 signaling and type I interferon production distinguish pathogenic and nonpathogenic AIDS virus infections"
Nature Medicine - 12, 1365 - 1371 (2006)

What is the hypothesis to be tested here?

Why is Figure 1A important to show?

Why are they interested in dendritic cells (DCs)?

Why are they specifically interested in the DCs in lymph nodes in Figure 1G?

Explain what iHIV and iSIV is regarding aldrithiol-2-inactivated virus. Not exactly the same as infectious HIV. Figure 2 they show that IFN is produced by pDCs in response to HIV or SIV (albeit inactivated), that TLR7 and/or 9 is involved and that only the DCs are responsible (i.e. not the RIG-I pathway like with influenza).

So, now they know that virus can induce IFN in pDCs, and they know that in vitro TLR7/9 is involved, they know that in vivo DCs don't get activated in SMs. How do they show directly that SMs DCs do not respond to SIV by making IFN?

How do they show that this is specific to the TLR pathway?

How do they show where the defect lies in SMs?

What is so amazing about Figure 5?

What is their model?

Why don't they think that the microbial translocation is the primary defect?

What else do they need to do to establish this model?

November 6, 2008 MCB532

Class questions for “A single mutation in Chikungunya virus affects vector specificity and epidemic potential” PLoS Pathog 3(12): e201.

What is the hypothesis to be tested here?

Why did they think this might be true?

What are they showing in Figure 1 (.i.e what do the Y and X-axis represent?)

Why is part B so important where they put they mutations into a West African strain?

Why are the competition assays so great? Follow up—What does Figure 2 add that was not shown in Figure 1

Why is Figure 3 so cool?

So, hypothesis is that transmitted better. How do they test this?
Mouse assay in Figure 5.

Why is the control of sub-cutaneous inoculation in Figure 5A so interesting?

Why do they say that this mutation did not occur before?

What is there model for why this mutation is linked to the epidemic.

Did they prove their hypothesis? What would you do next?

