

1. The paper titled "Dendritic cells loaded with stressed tumor cells elicit long-lasting protective tumor immunity in mice depleted of CD4⁺CD25⁺ regulatory T cells" by Prasad et al. reports that stressed and gamma-irradiated syngeneic tumor cells elicit protective CD4⁺ and CD8⁺ T cell responses in the absence of dendritic cell (DC) maturation. In the absence of infectious non-self (i.e. pattern recognition receptor [PRR] agonists) do you think necrotic cells or apoptotic cells would be more effective at inducing DC activation? Explain your reasoning.

2. In the paper titled "Involvement of chemokine receptors in breast cancer metastasis" by Müller et al., migration and invasion were assayed using fibronectin- or Matrigel-coated cell culture inserts (chemotaxis or chemoinvasion assays). (A) Which steps of the process of leukocyte extravasation are modeled in this system? (B) Which steps of the process of leukocyte extravasation are **not** modeled in this system?

3. Draw an idealized classical hepatic lobule. Indicate the location of the central vein and the peripheral portal triads. What is the relationship between Rappaport's hepatic acinus and the classical hepatic lobule? (You may use an explanation and/or an illustration to answer the last part of this question).

4. "Cross-priming" refers to the activation of naive cytotoxic T lymphocytes (CTL; CD8⁺ T cells) by antigen-presenting cells (APC) that have acquired nominal antigens from another cell. Although injection of mice with MHC-mismatched cells expressing minor H antigens has been shown to prime self-MHC-restricted CTL responses specific for the minor H antigens, the biological relevance of cross-priming in protective immunity against viral infection remains controversial. Describe one experiment that could be performed to test that hypothesis that virus-specific CTL are elicited by cross-priming. Be sure to state why the experimental result supports (or refutes) the hypothesis, and how you control for direct priming.

5. Answer the following question posted on a hepatitis B virus (HBV) testing and prevention web forum.

I donated blood and the results came back positive for anti-nucleocapsid core protein (anti-HBc) only. Although the blood was trashed, I thought this might have been a false positive result. I donated blood a second time and got no notification that there were any problems with my blood, and I was asked to donate again in the future - which I did. Now the third time I donated, I was again anti-HBc positive only. Now I am told that I should never again in my entire life donate blood. First of all, why would my future donations be denied if I am not a carrier (I wasn't positive for anything else)? It seems to me that HBV isn't a death sentence for most people - greater than 90% clear the infection. Aren't the odds of transmitting HBV from a person who has cleared the infection via blood transfusion very low? Since we are desperate for donors, is this really a necessary precaution?