Tuesday practice exam question.

Imagine the experiment in panel below was carried out exactly the same as Figure 4A and B of Roy et al., 2011 (Adhesin Degradation Accelerates Delivery of Heat-labile Toxin by Enterotoxigenic Escherichia coli) but with very different results (shown here; note—the figure below is NOT the original figure and has been altered). Ignore figure 4C and D. Explain:

a. the <u>original</u> results of Figure 4A & B and their interpretation
b. how the results in this <u>new</u> Figure 4 are different
c. how you would now interpret the altered findings (230 words maximum)

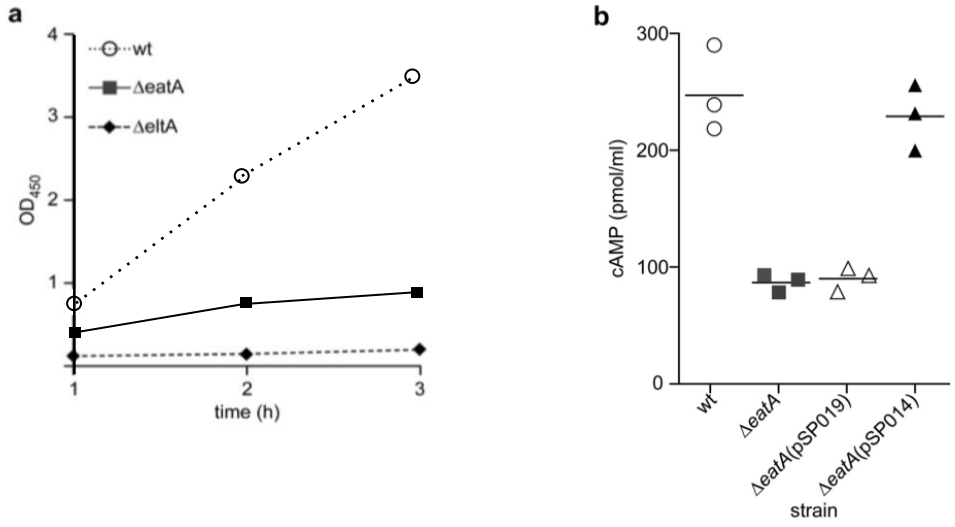

FIGURE 4. EatA accelerates delivery of heat-labile toxin. a. ganglioside-binding ELISA demonstrating production of heat-labile toxin by H10407 (wt), the *eatA* mutant, and negative control *(eltA)* strain, which contains a mutation in the genes for LT. **b.** activation of cAMP in target Caco-2 intestinal epithelial cells by WT ETEC, *eatA* strain, and *eatA* mutant complemented with plasmid expressing either the recombinant EatA protein *(pSP014)* or the mutant serine protease-deficient protein *pSP019.*

In the <u>original</u> figure 4, the Δ*eatA* mutant and the wild-type ETEC produced and secreted similar amounts of heat-labile toxin (LT). However, the delivery of that toxin to epithelial cells was impaired in the Δ*eatA* mutant, as indicated by the reduced cAMP production. The authors conclude that EatA has no affect on the production/secretion of LT, but it enhances LT delivery to target cells by modulating bacterial adhesion.

The <u>new</u> figure 4A suggests that LT production/secretion is impaired in the Δ*eatA* mutant. This could explain why less LT is delivered to target cells in figure 4B. It complicates the interpretation of the mechanism responsible for diminished delivery. It seems likely that there is less delivery because there is less LT produced/secreted. From these data, you cannot determine whether the impact of EatA on adhesion also contributes to the reduced delivery of LT.