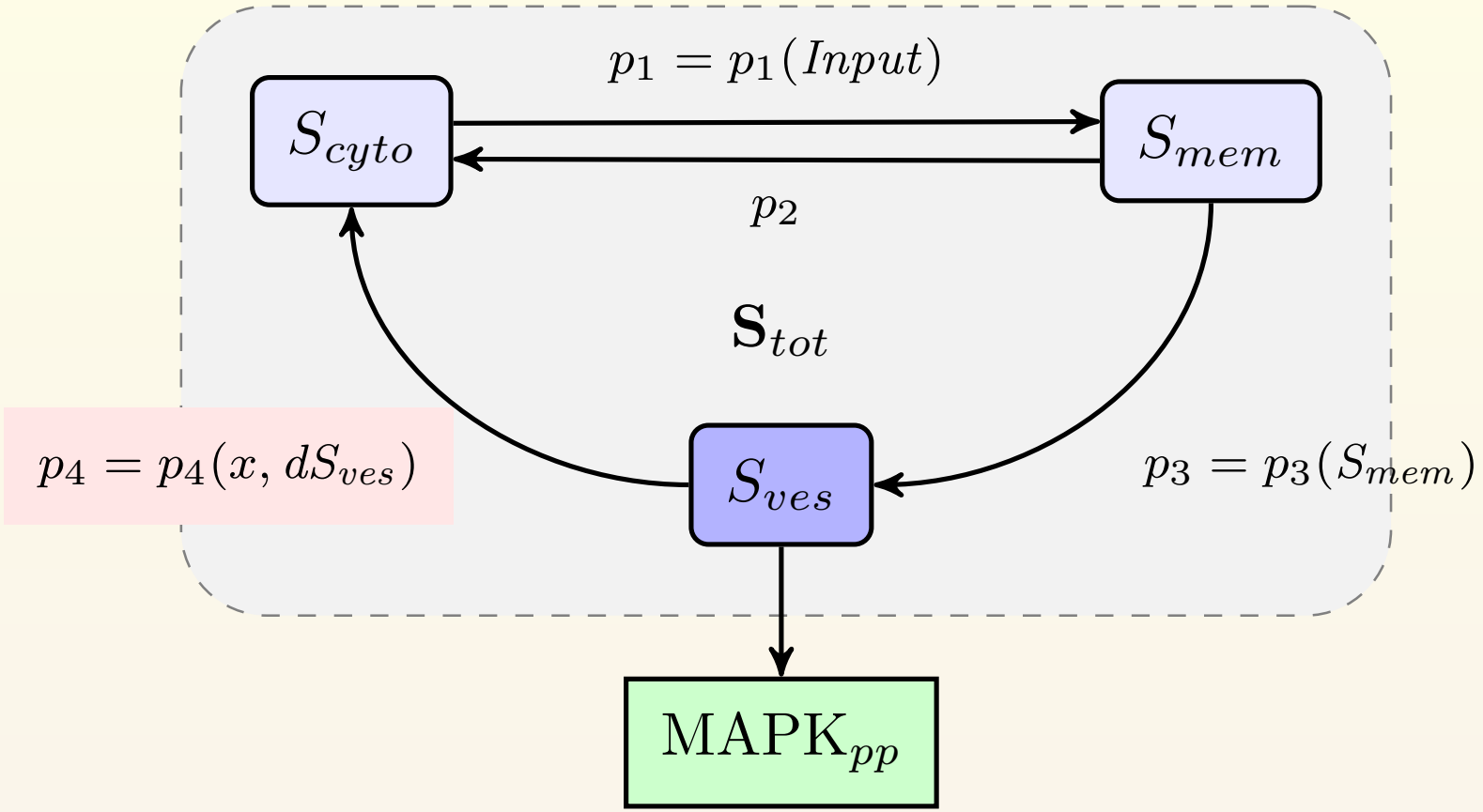
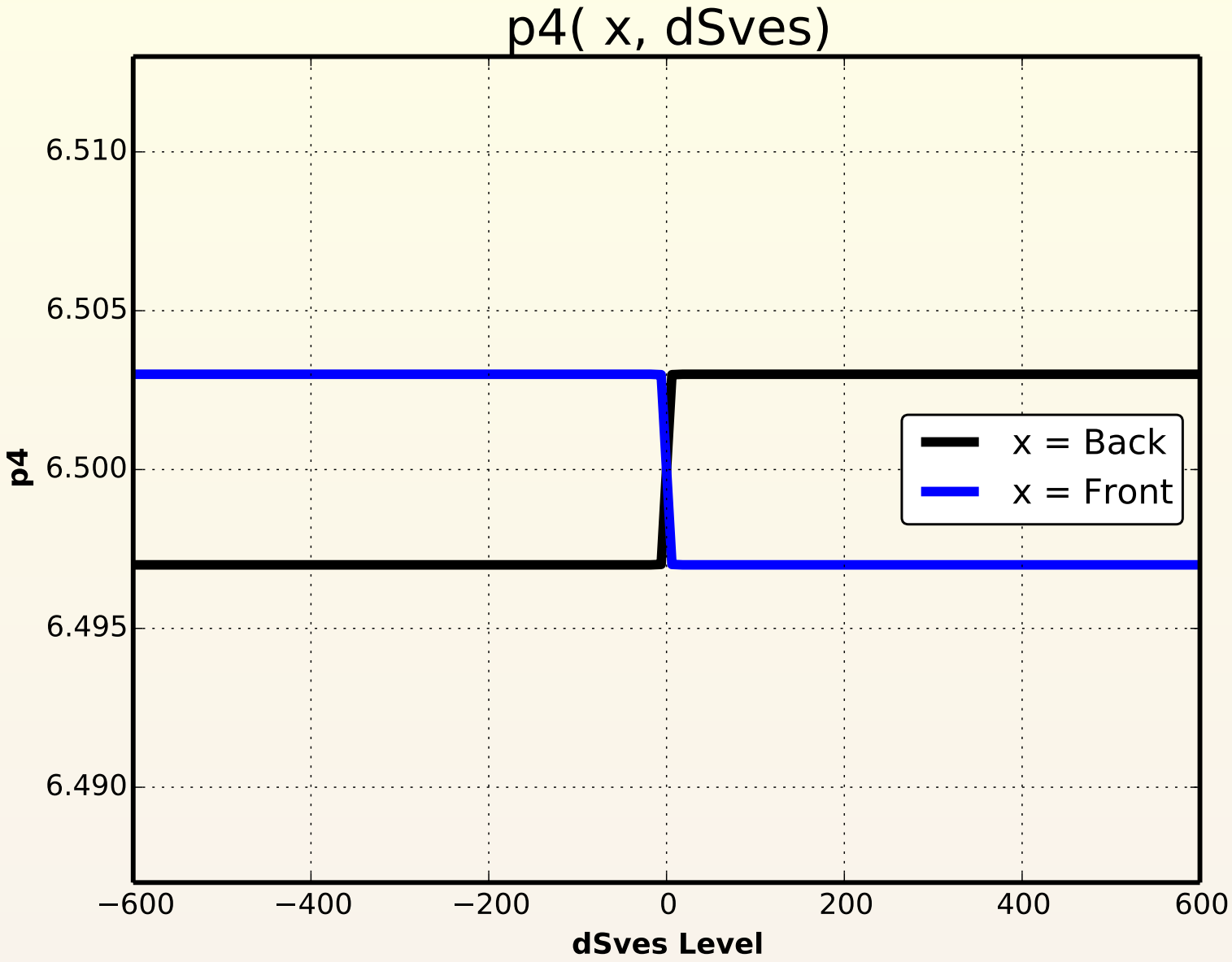
# Model Schematic



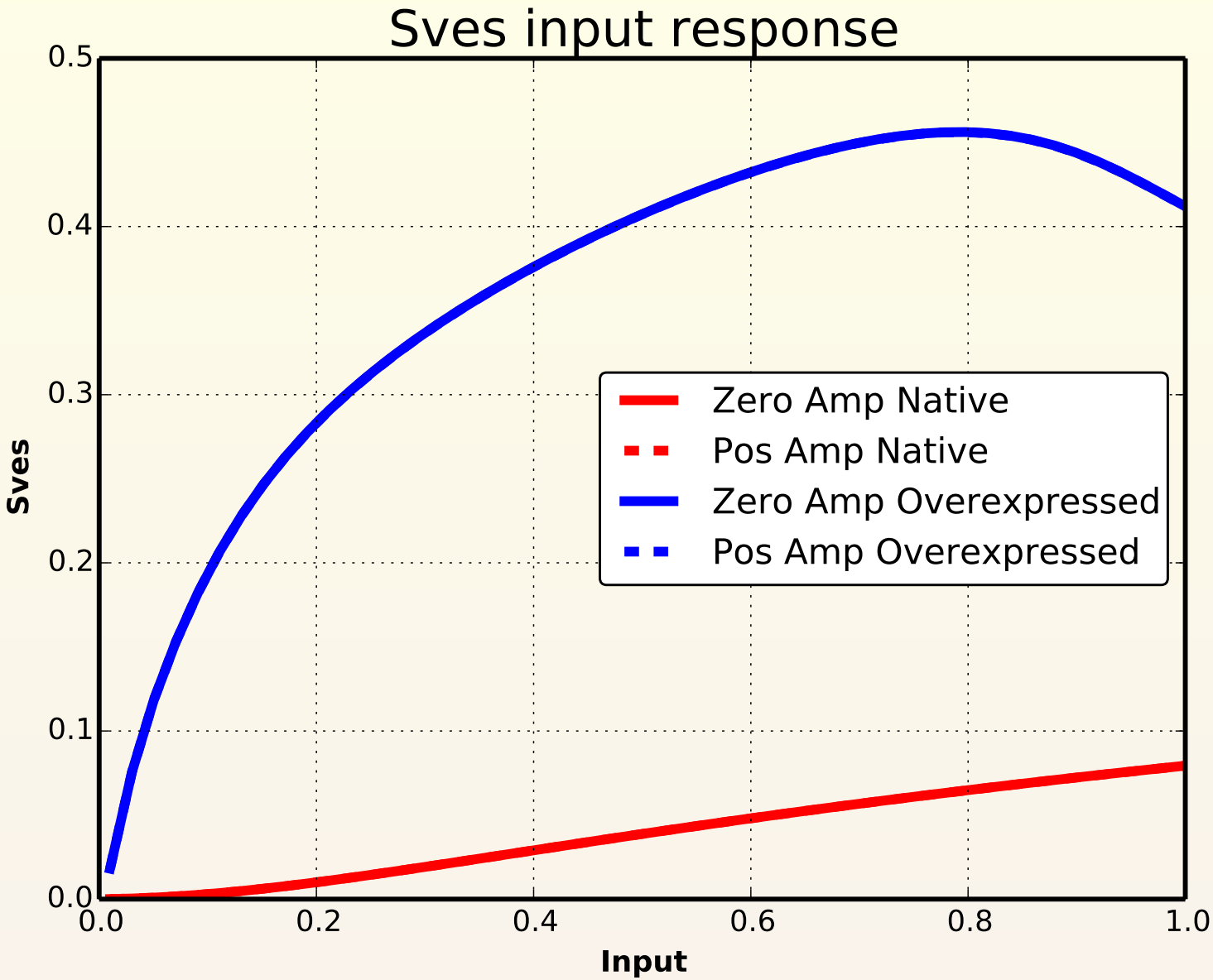
# Sves inhibits Rab11 localization

- p4(dSves) aka Rab11 needs to be a function of the difference between vescile scaffold in the front from the back
- $dSves = Sves_{front} - Sves_{back}$
- in other words, vescile scaffold and Rab11 are mutually inhibitory
- Since native dSves is small, need fast p4 switching to amplify native MI
- Need slow p4 switching for the OE case in order to prevent positive feedback locking, otherwise you wont see the chemorepulsion to attraction transition

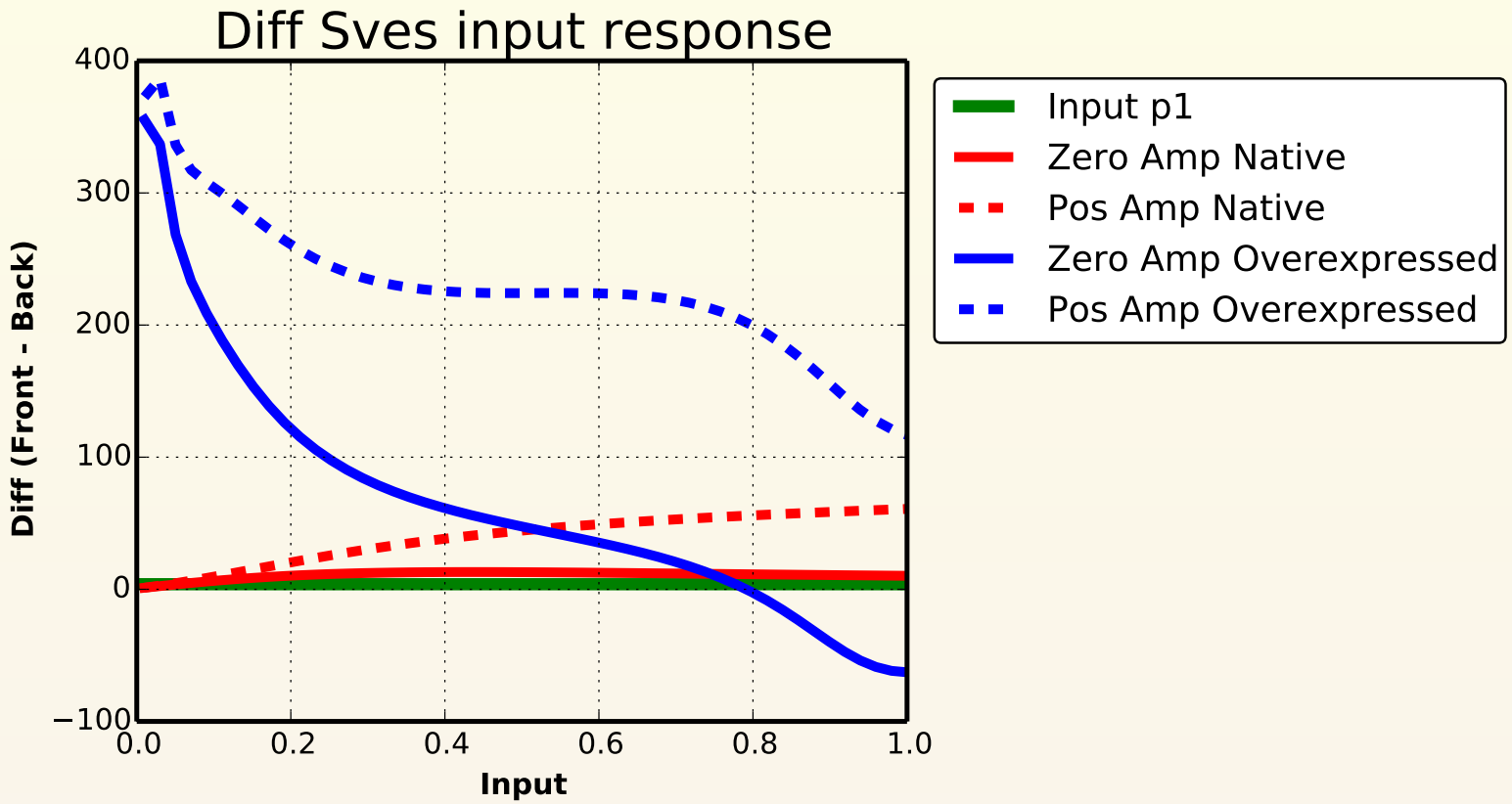
# Fast p4 switching



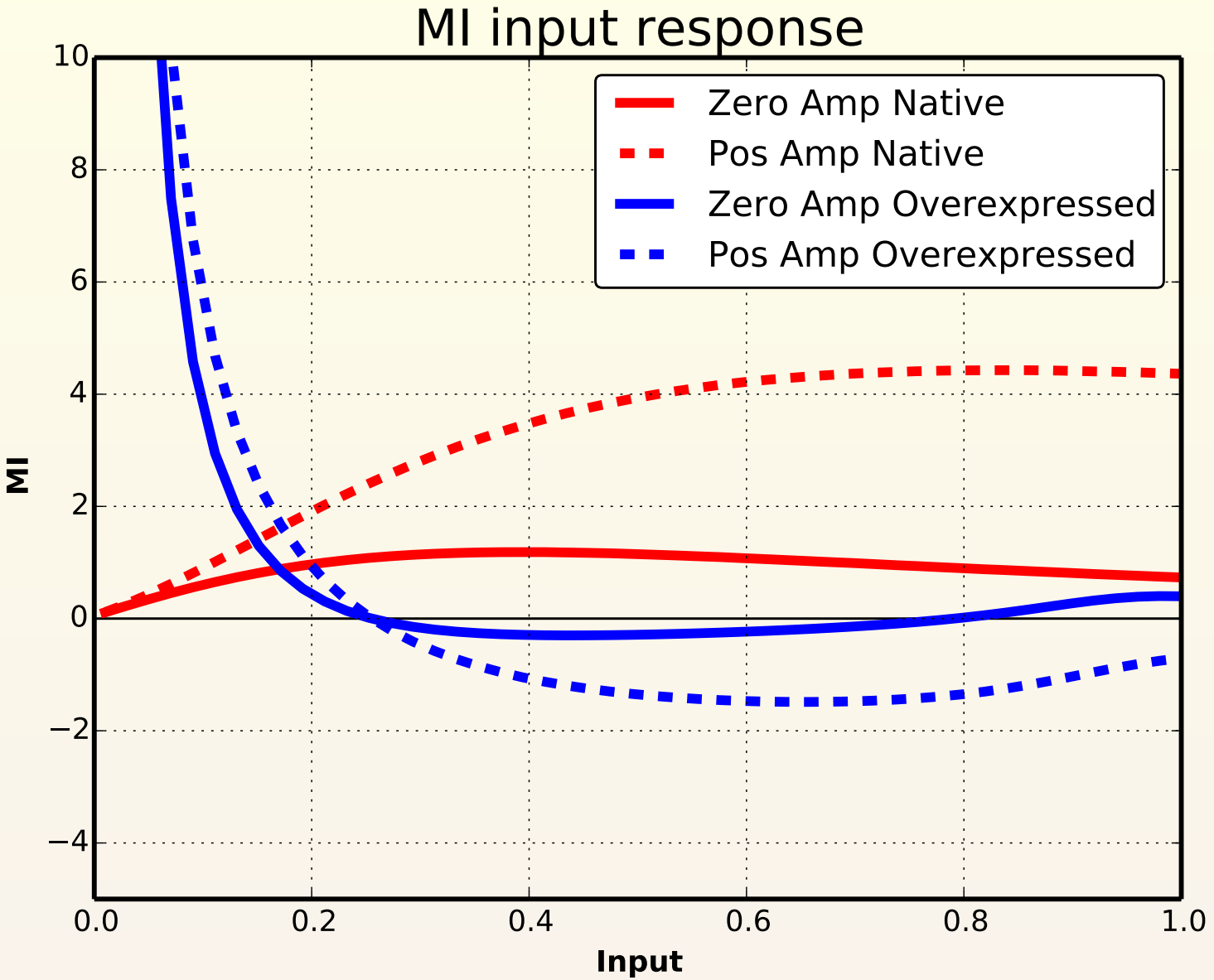
# Fast p4 switching



# Fast p4 switching



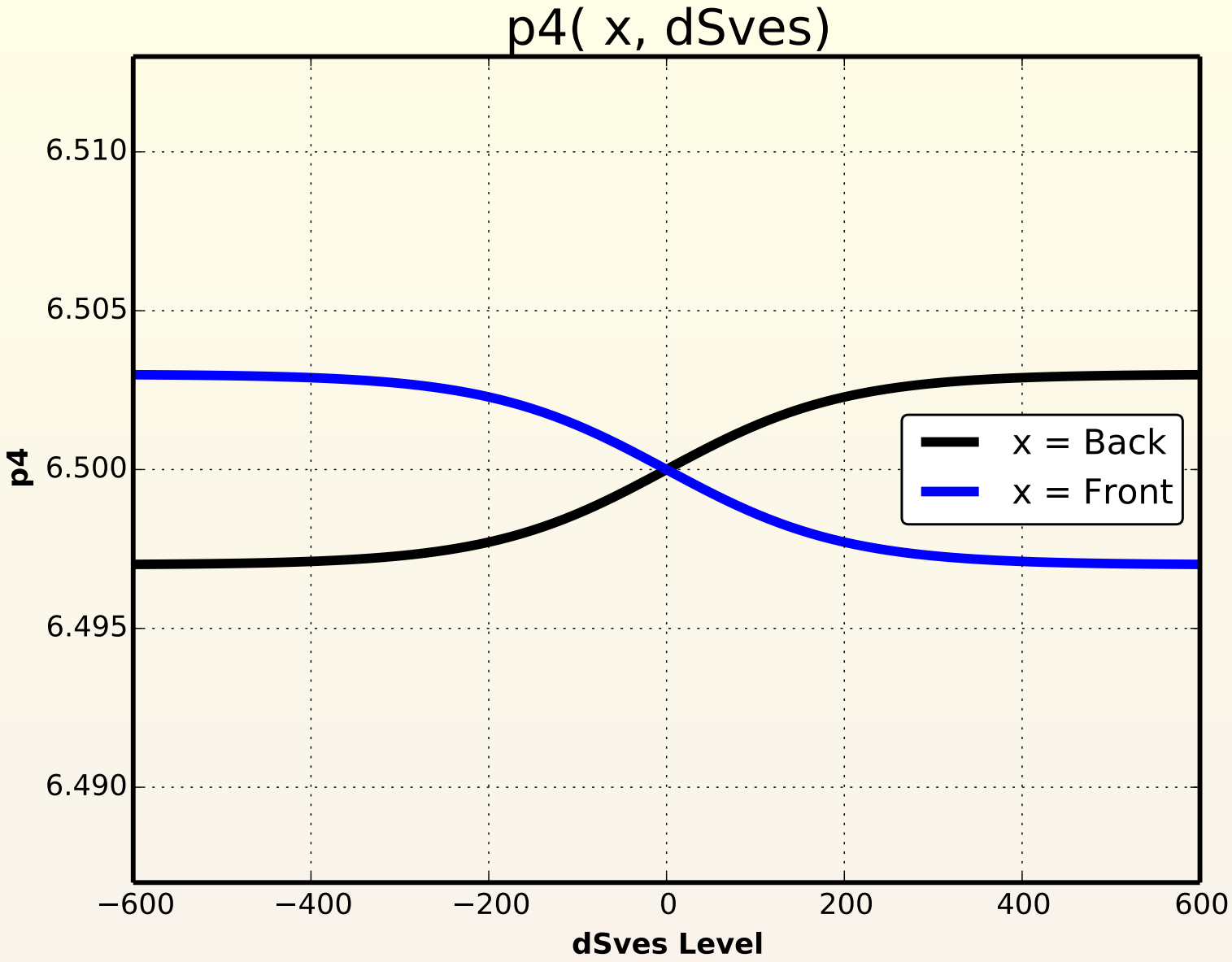
# Fast p4 switching



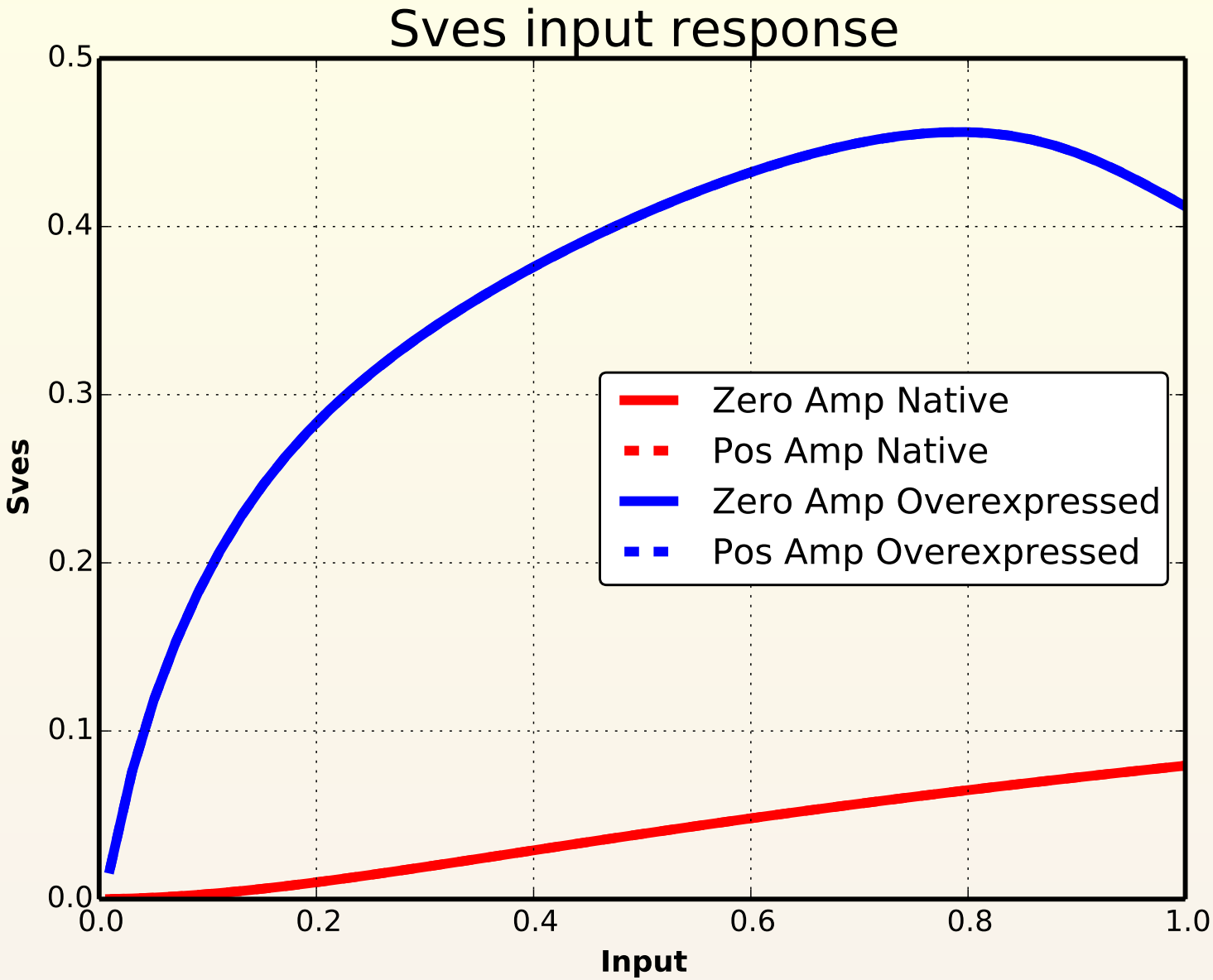
# Fast p4 switching

- we get a boost in native MI but no positive MI at high dose overexpressed cells
- but if we reduce the sigmoidal transition w.r.t. dSves, we can amplify at OE levels at the expense of the native case

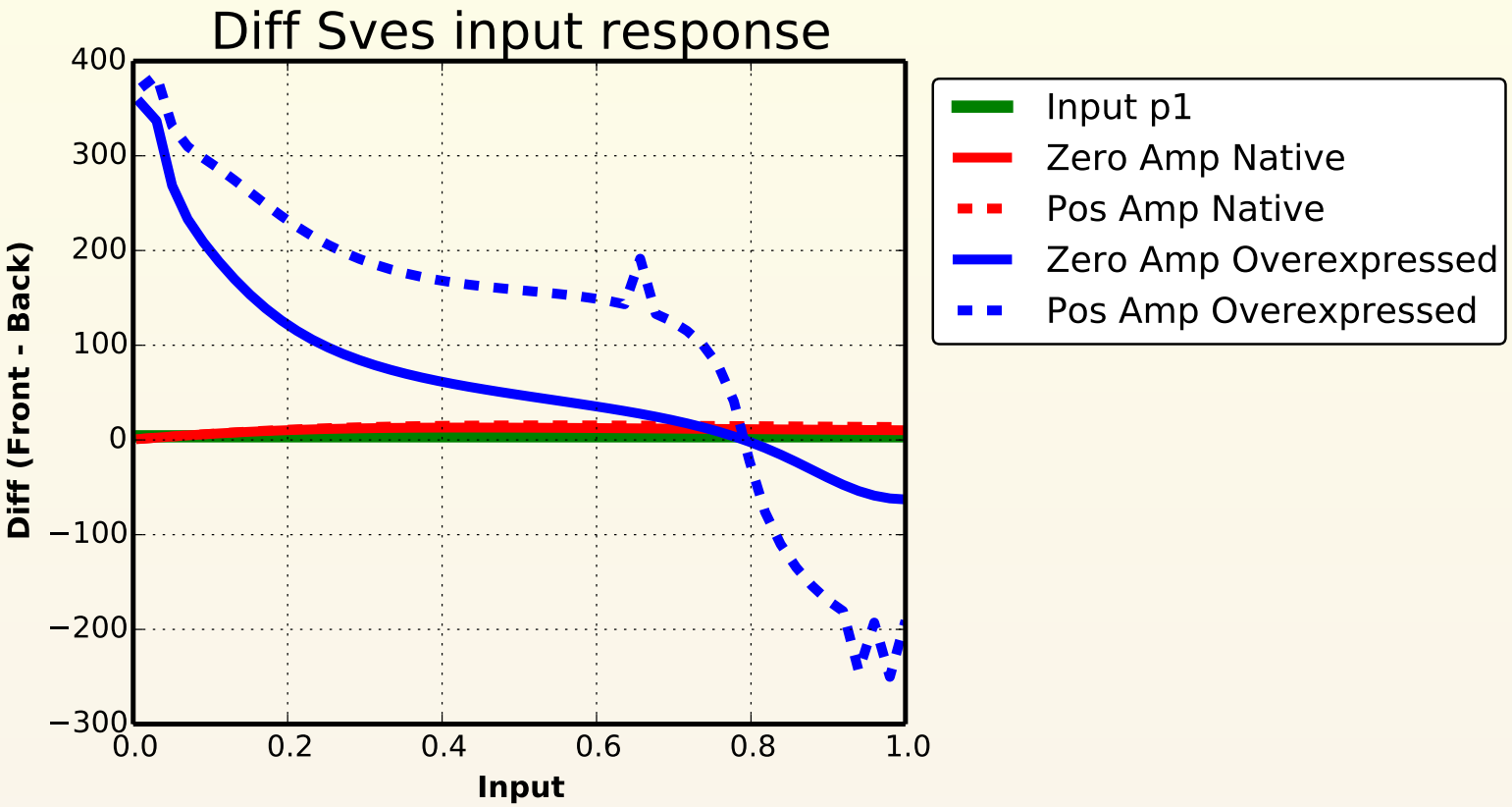
# Slow p4 switching



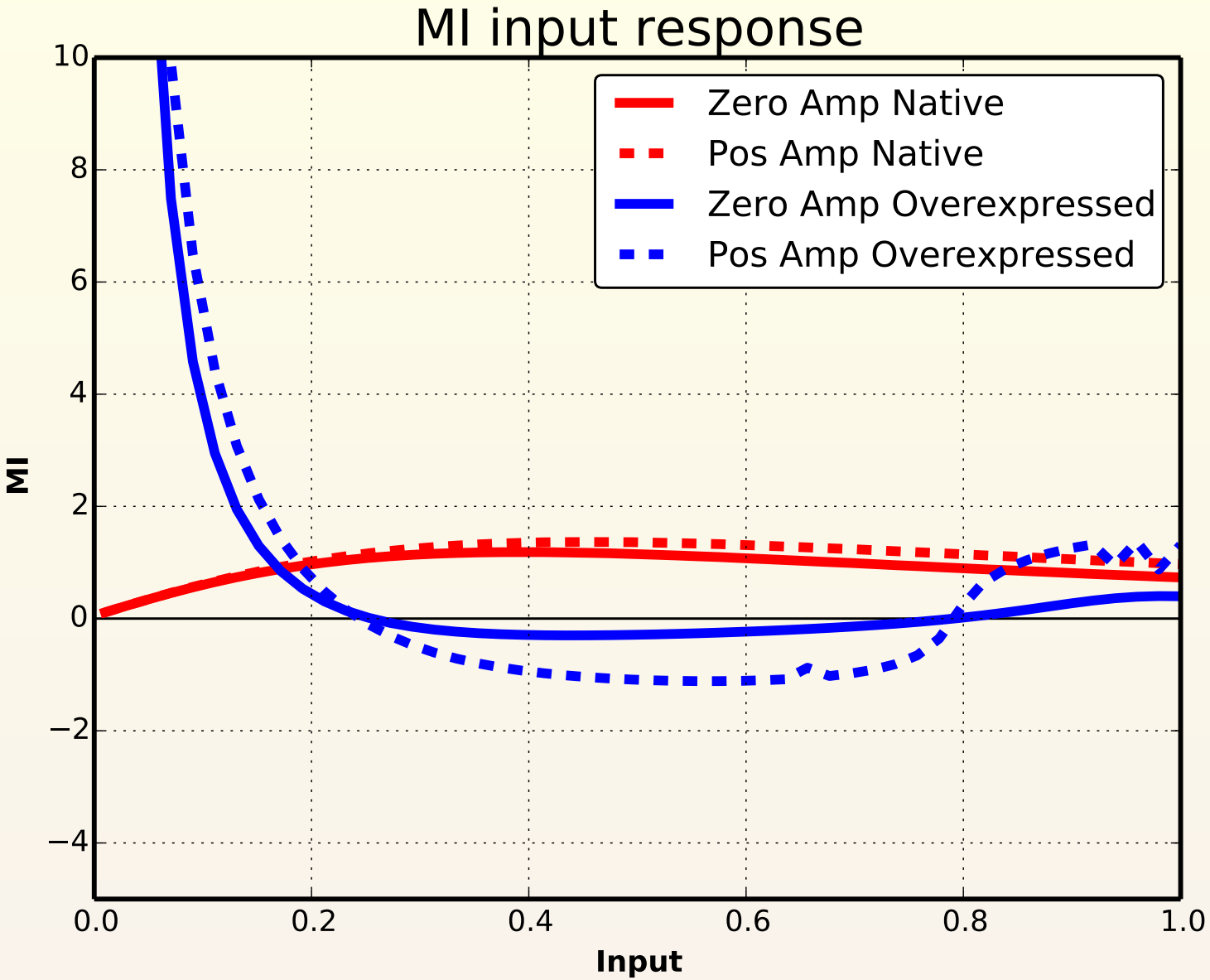
# Slow p4 switching



# Slow p4 switching



# Slow p4 switching



# Sves inhibits Rab11 localization

- I feel like we can almost get both cases to work
- We have a single model that can amplify in the native case and in the OE case, just with just a signle parameter change
- Another way to interpret this is that vesicle scaffold inhibits Rab11 translocation AND reduces its kinetics