

1. **Pick up** Name Folder

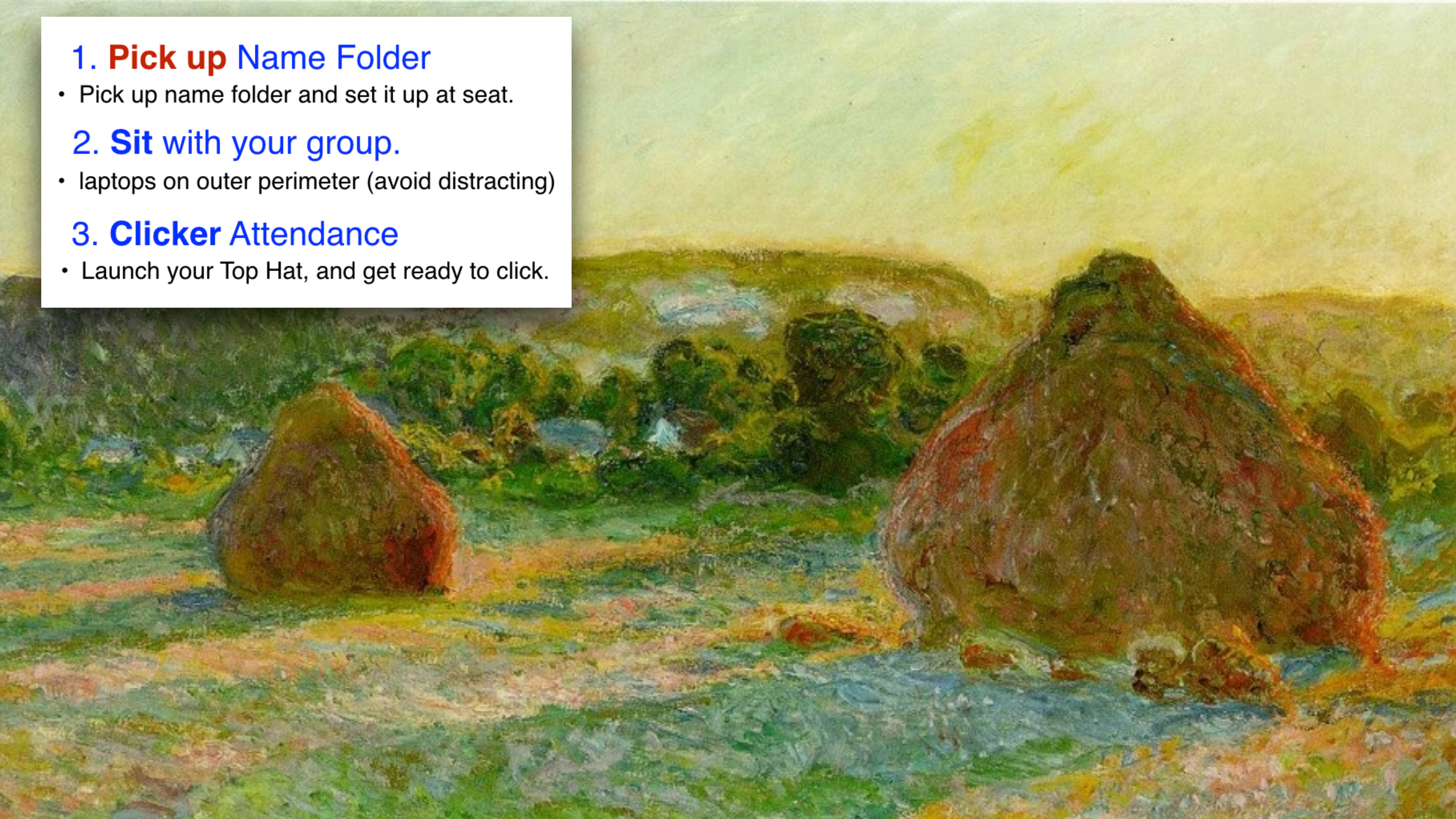
- Pick up name folder and set it up at seat.

2. **Sit** with your group.

- laptops on outer perimeter (avoid distracting)

3. **Clicker** Attendance

- Launch your Top Hat, and get ready to click.





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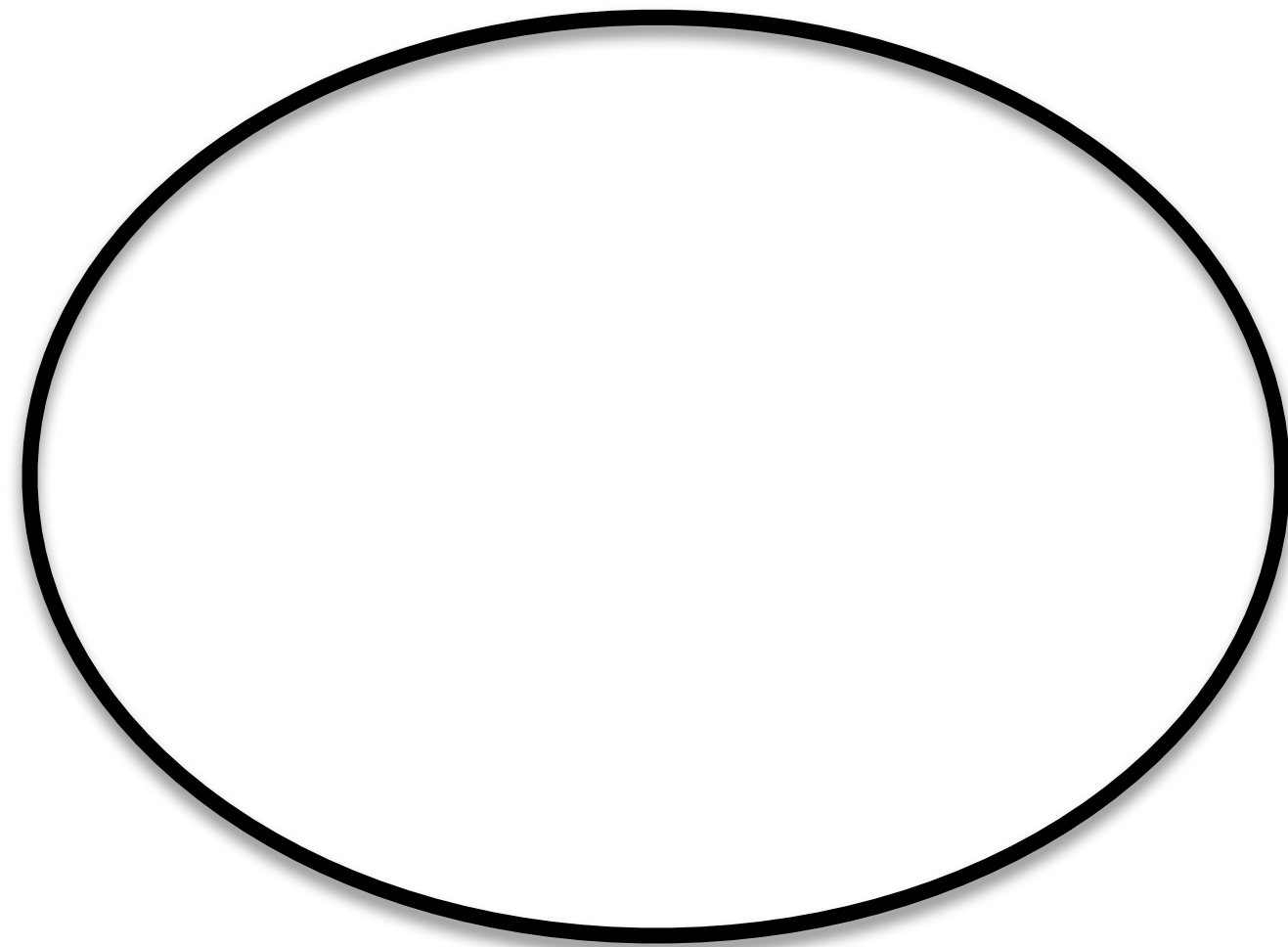
Before Spring Break:

1. **Analyze** + Defend your Gel results
-> with figures from your DRAFT2

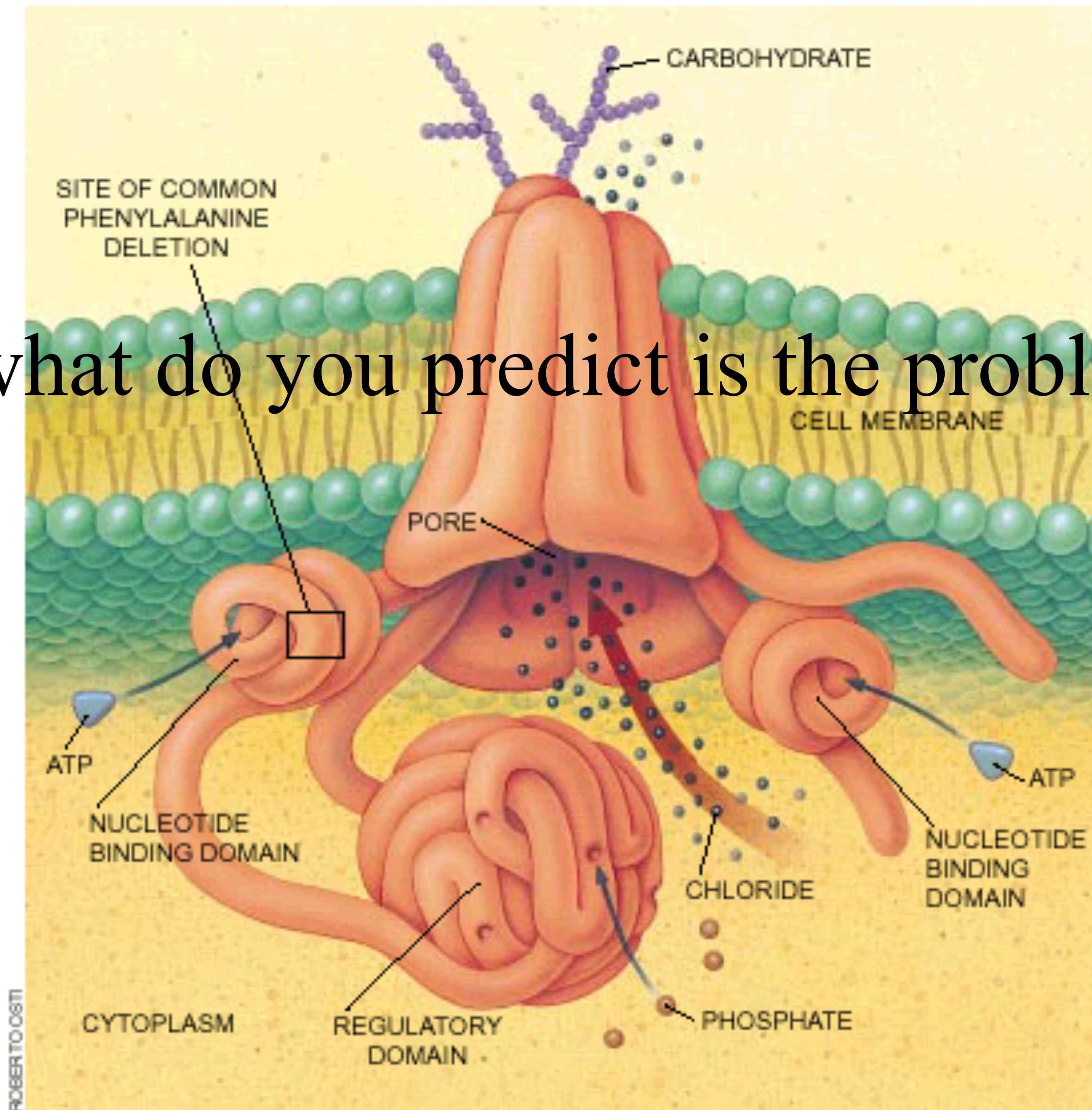
2. **Order** your “Published” Primers
-> once found where they bind on gene DNA

Art class: Biosynthesis of CFTR (central dogma + cell biology)

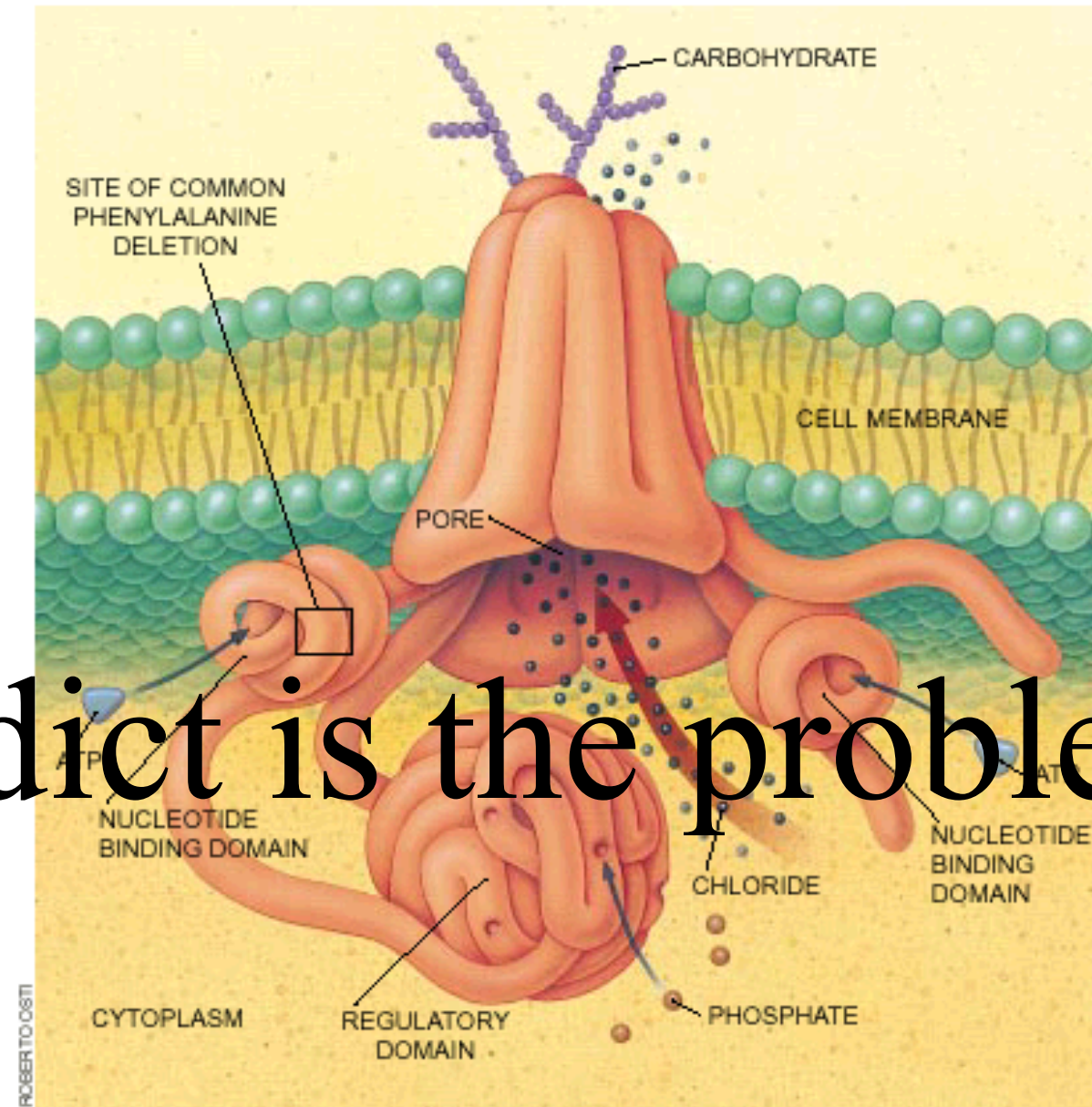
DRAW IT



So what do you predict is the problem?



So what do you predict is the problem?



- A. The $\Delta F508$ deletion alters gating, thus blocking the CFTR.
- B. The deletion alters ATP binding, thus stopping CFTR.
- C. The deletion alters the folding, but CFTR still works.
- D. None of the above cause the disease.

Explain Aloud

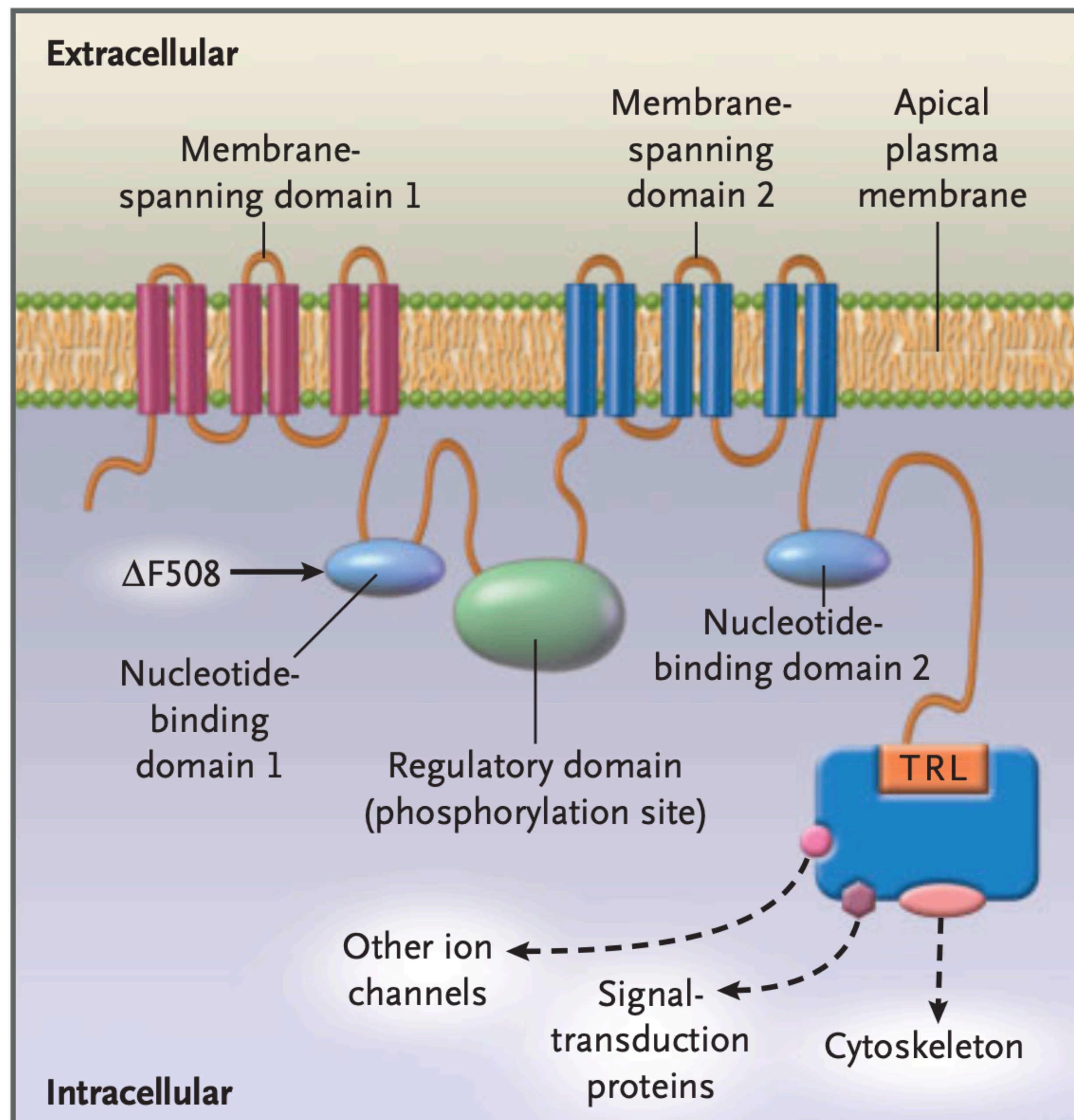
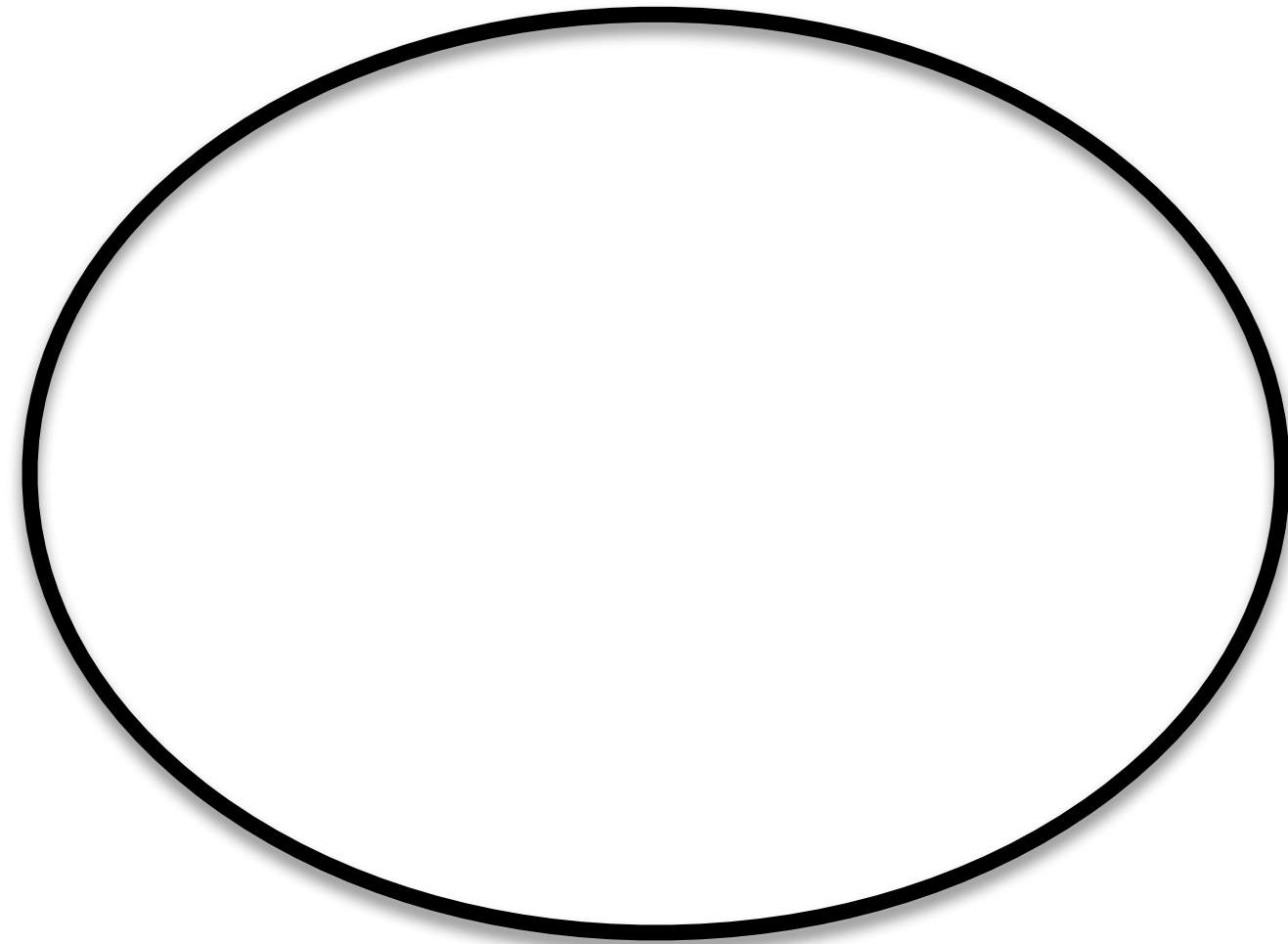


Figure 1. Hypothesized Structure of CFTR.

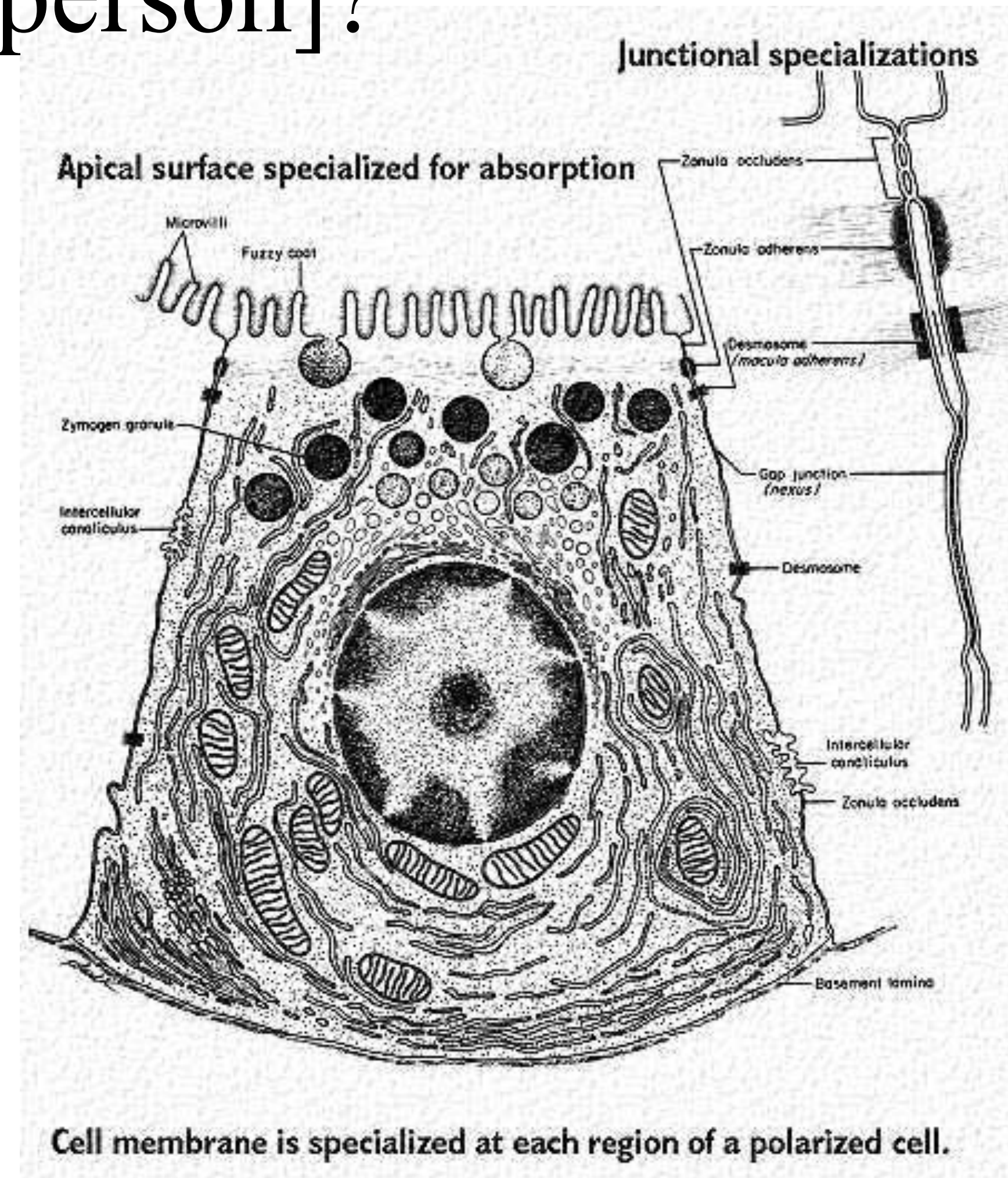
Art class: Biosynthesis of CFTR (central dogma + cell biology)

DRAW IT



Where are the CFTR channels found normally [in a healthy person]?

- A. the apical surface ->
- B. inside the ER
- C. the lateral surface ->
- D. in the lysosome
- E. the basal surface ->



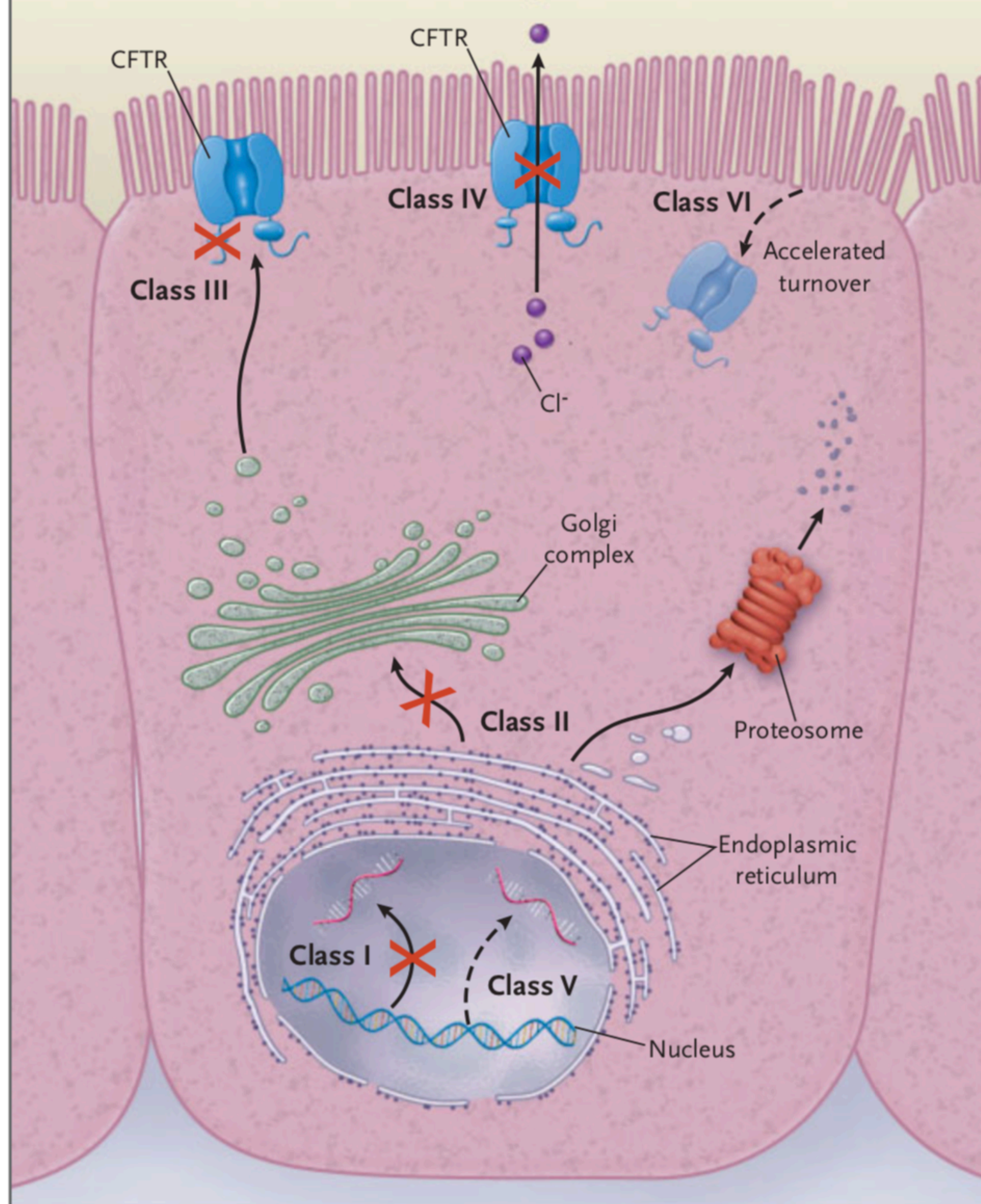
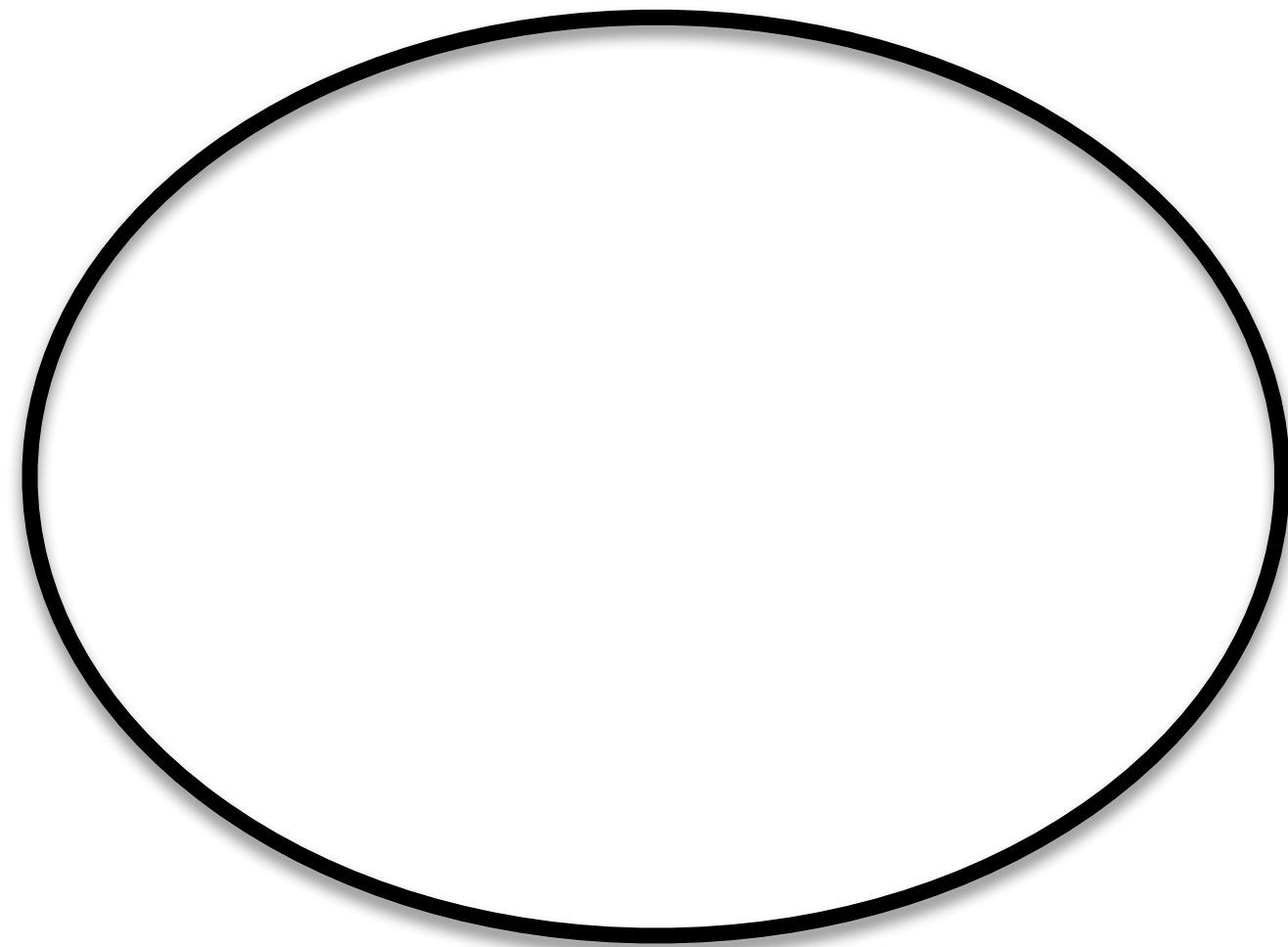


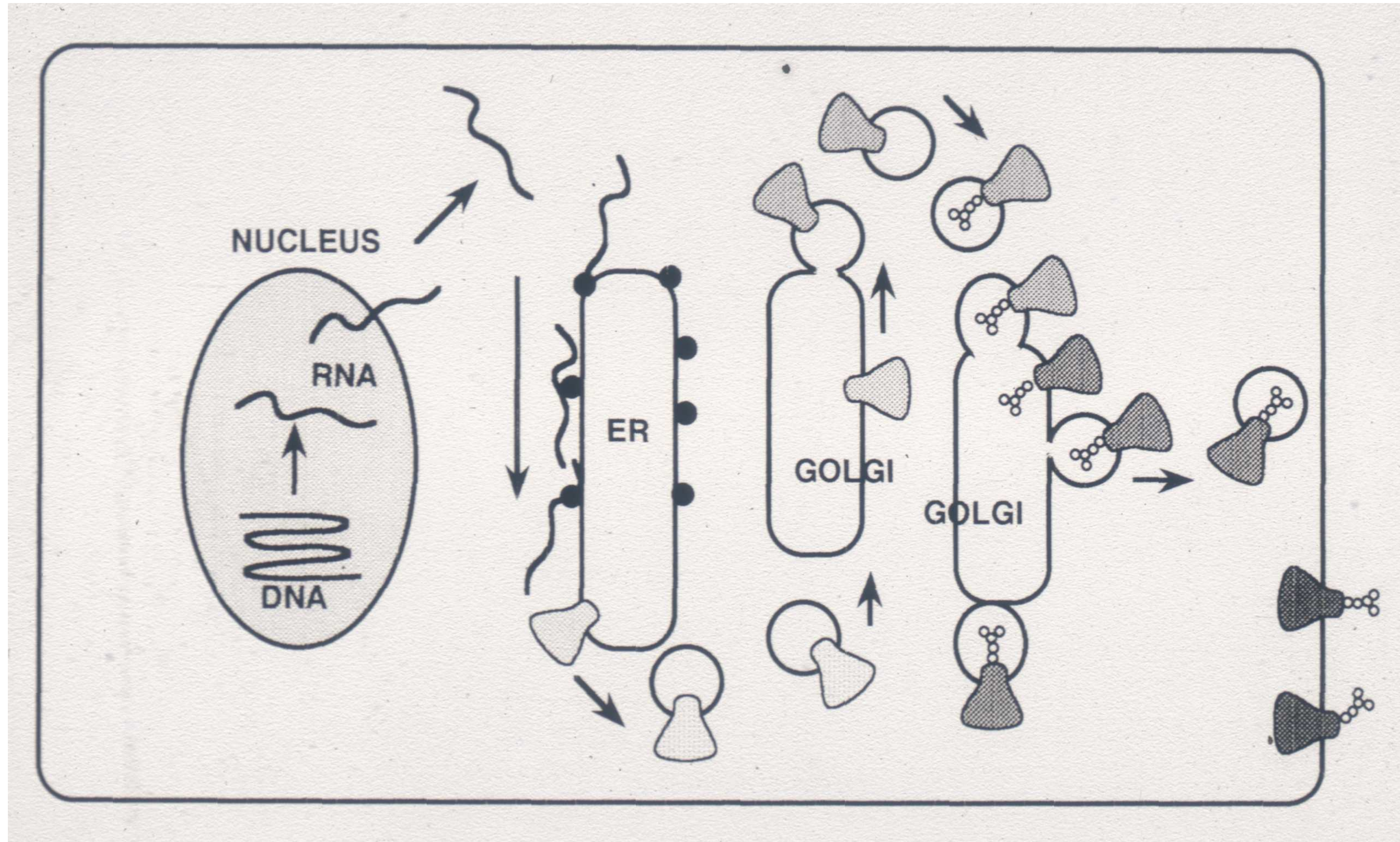
Figure 5. Categories of CFTR Mutations.

Art class: Biosynthesis of CFTR (central dogma + cell biology)

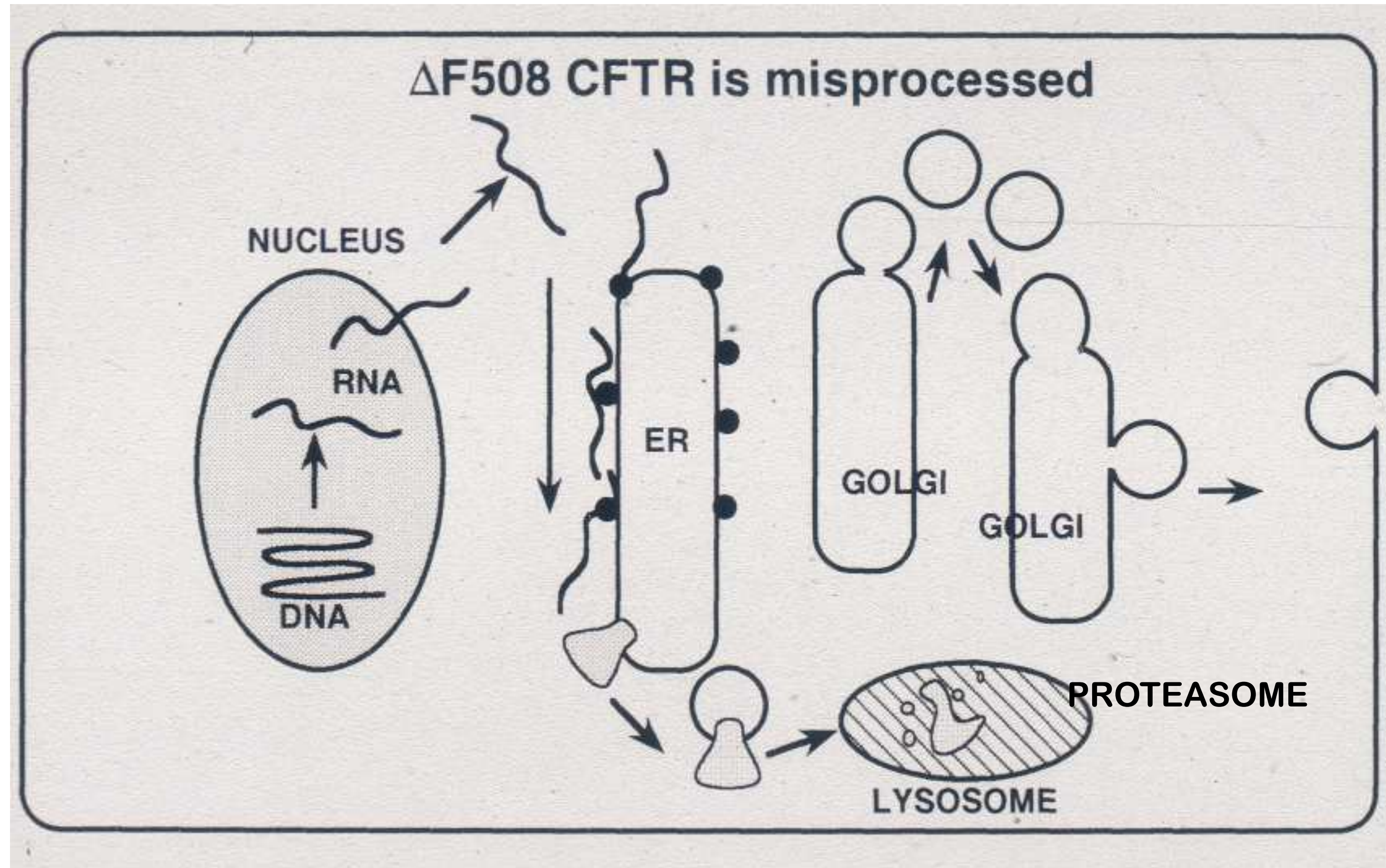
DRAW IT



biosynthesis of normal wild-type CFTR

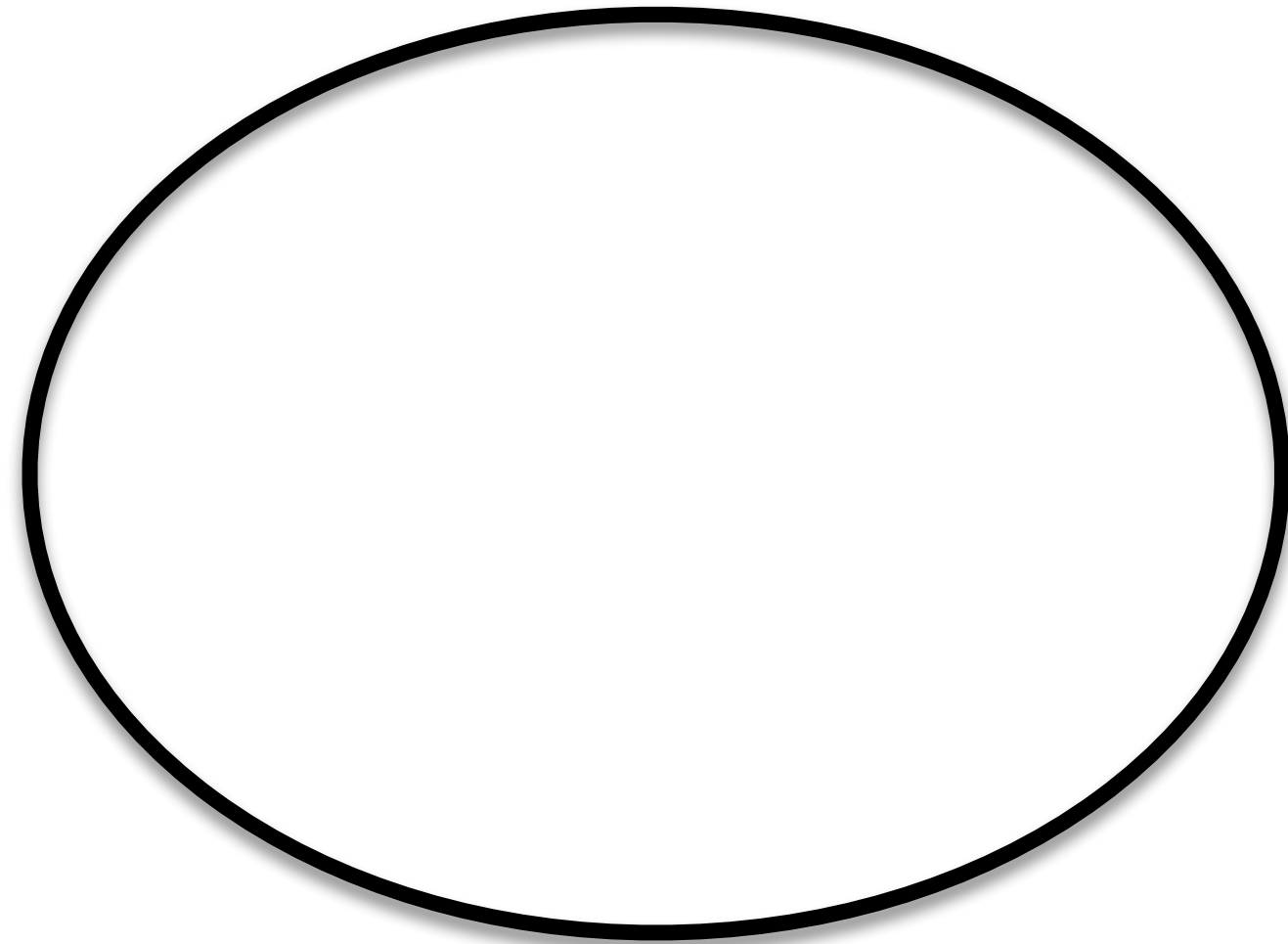


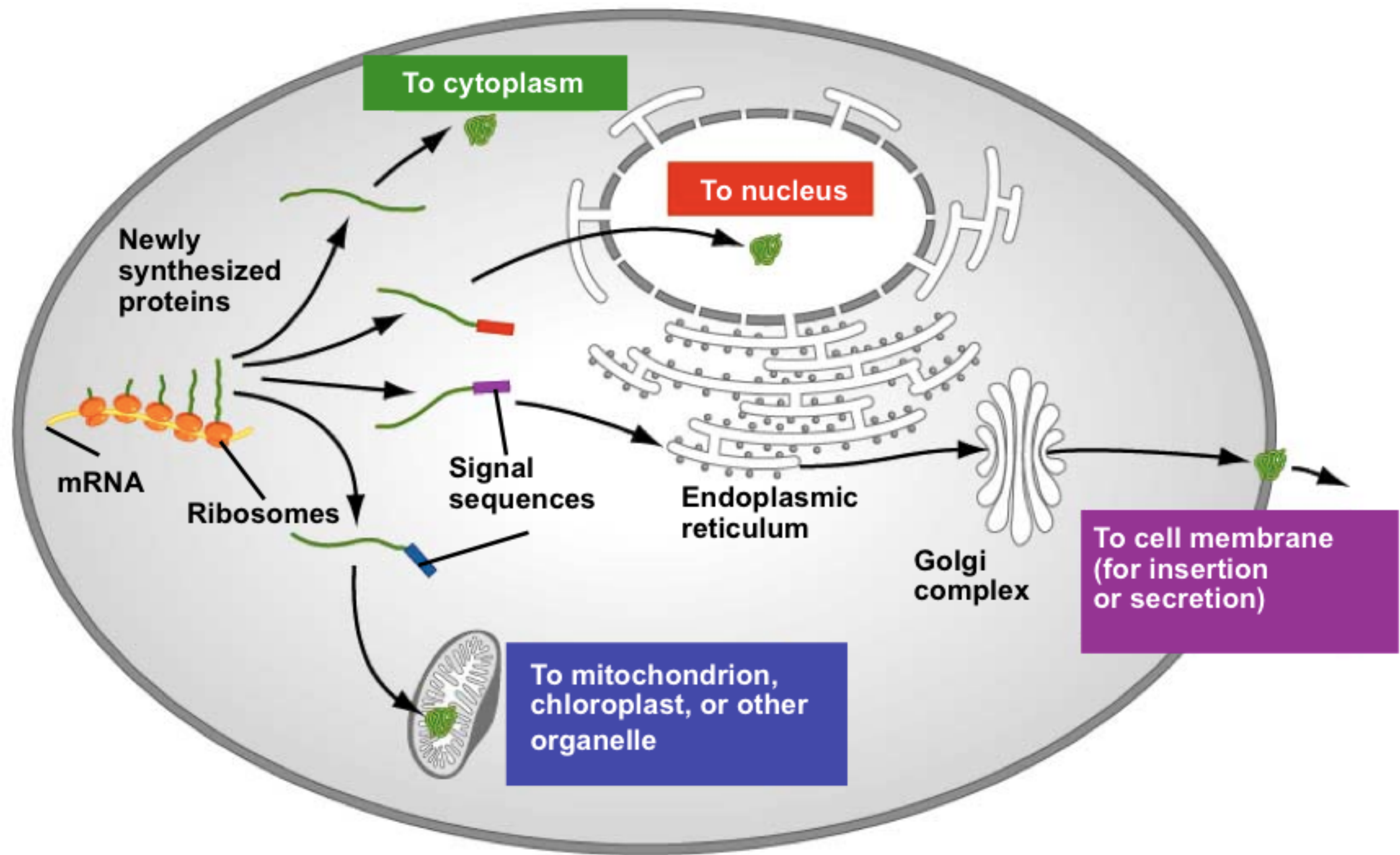
biosynthesis of mutant CFTR



Art class: Biosynthesis of CFTR (central dogma + cell biology)

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- 1.
- 2.
- 3.
- 4.
- 5.

