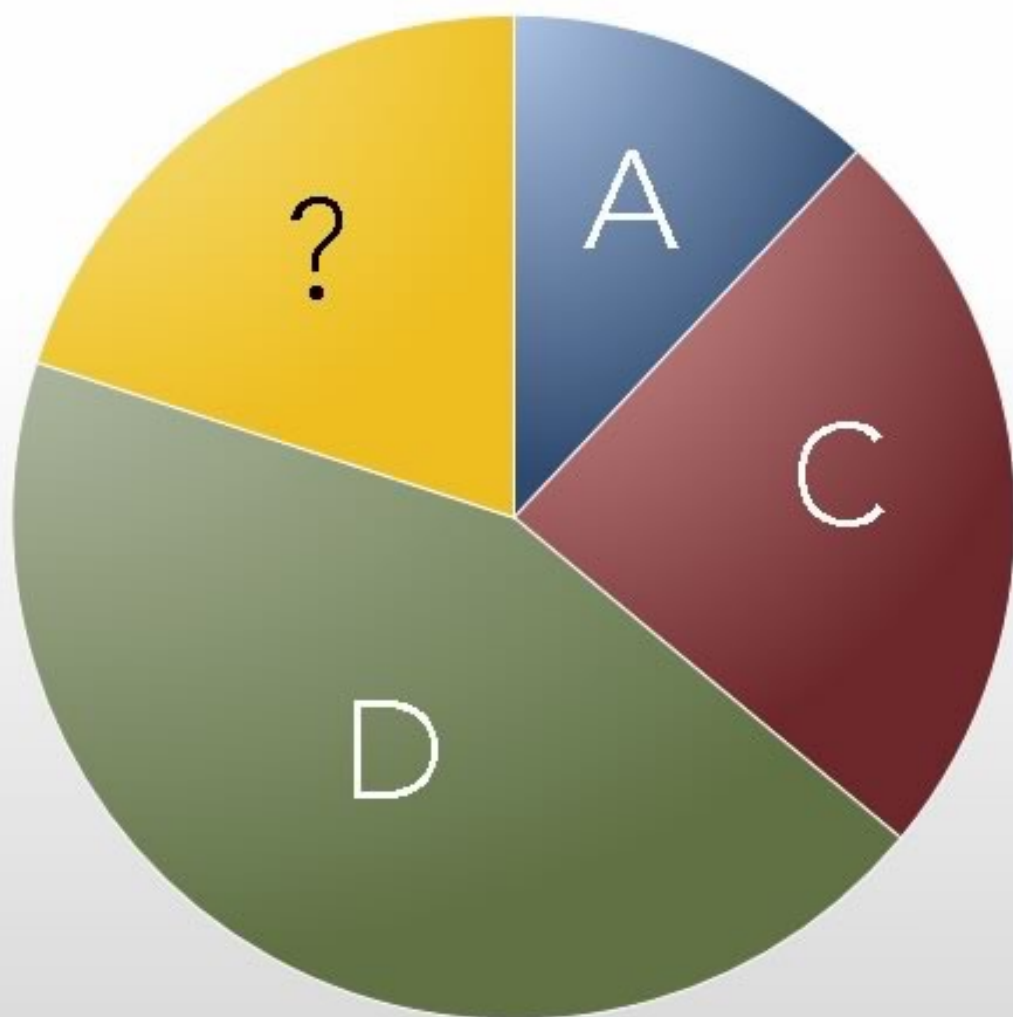


Which would be the best (rough) estimate for the average number of collisions between amino-acyl-tRNAs and an mRNA-ribosome complex before the "right" aa-tRNA binds?

- ☐ A. 1
- ☐ B. 4
- ☐ C. 20
- ☐ D. impossible to calculate
- ☐ no idea



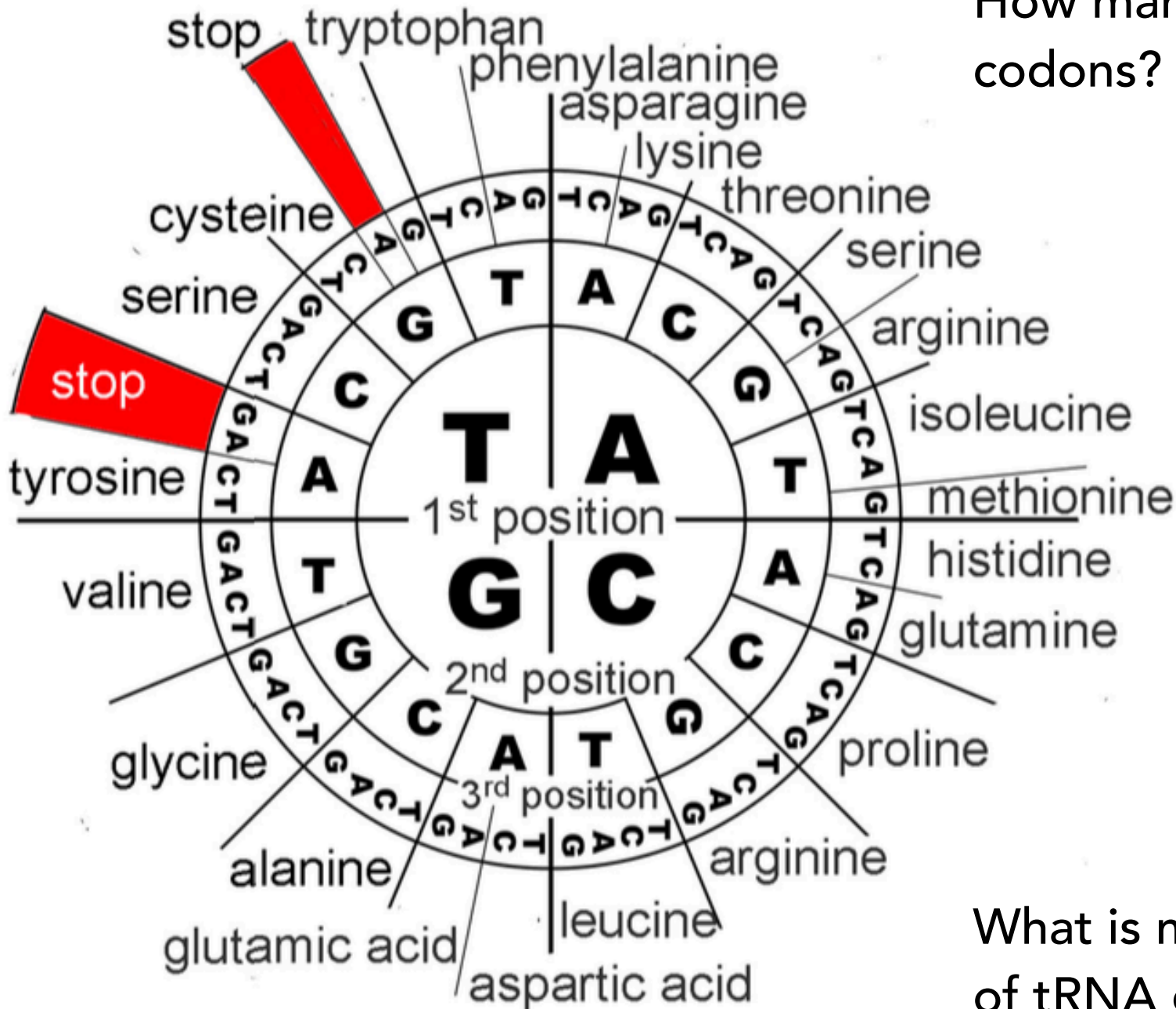
To generate a full length polypeptide of wild type length from an mRNA, which is not needed?

- ☐ a transcription start sequence
- ☐ a stop codon
- ☐ a start codon near the 5' end of the RNA
- ☐ a promoter or enhancer sequence
- ☐ no idea

↑  
should have been  
ARE



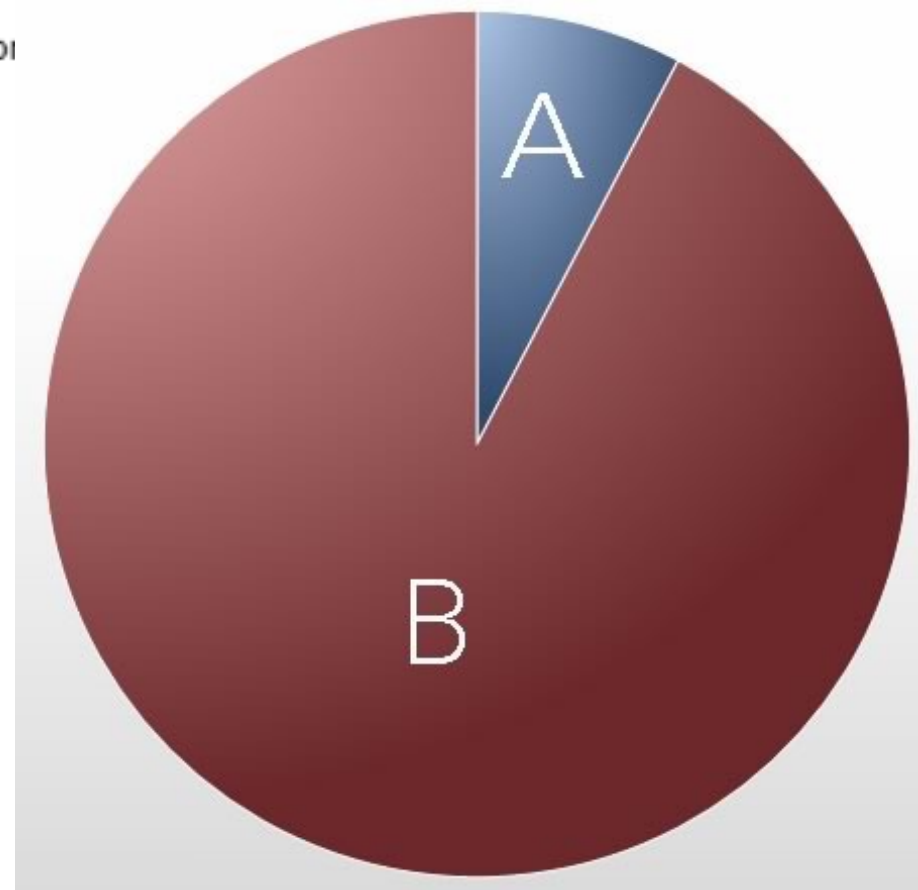
How many distinct  
codons?



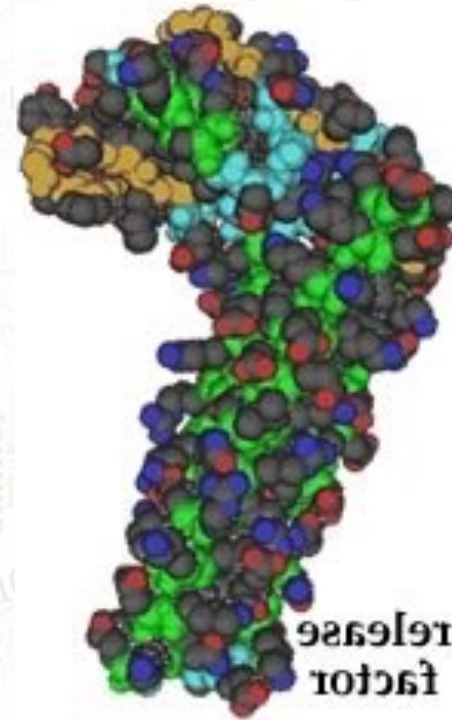
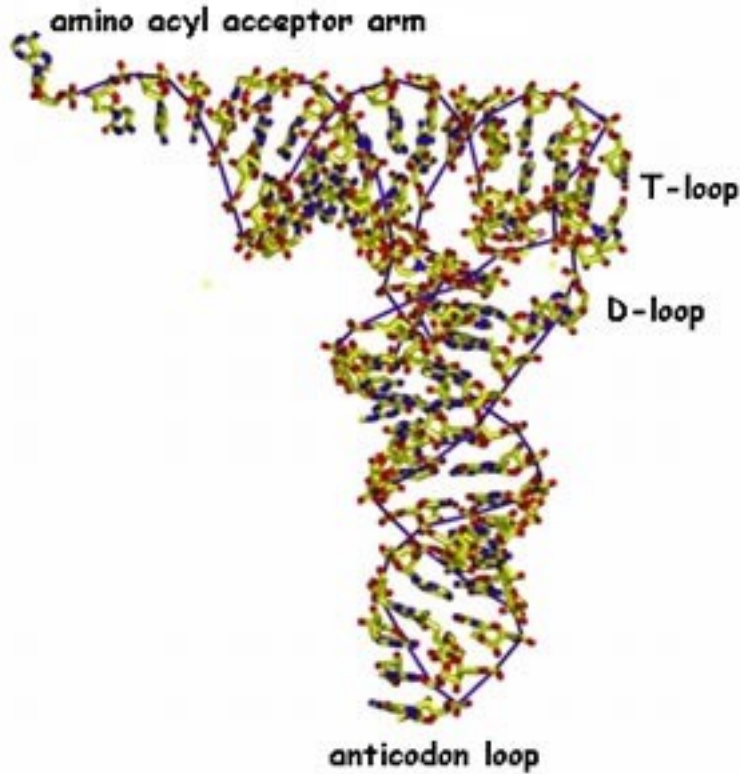
What is max. number  
of tRNA genes?

Why is the genetic code considered redundant?

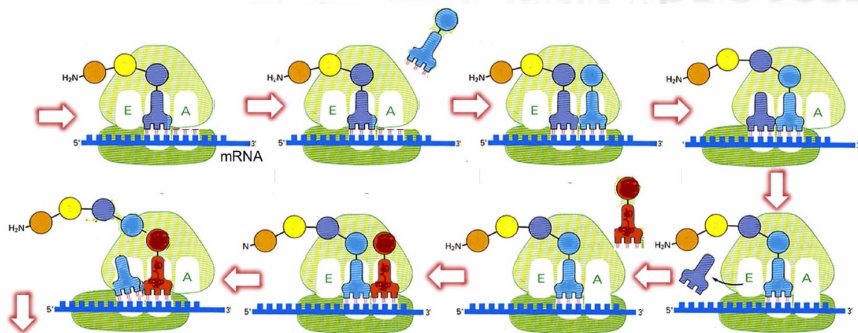
- ☐ there is only a single stop codon
- ☐ each codon encodes a unique amino acid.
- ☐ multiple codons encode the same amino acid
- ☐ an mRNA can have multiple start codon
- ☐ no idea



# Start codon (initiator tRNA (methionine))



stop codon  
release factor





organisms

selection

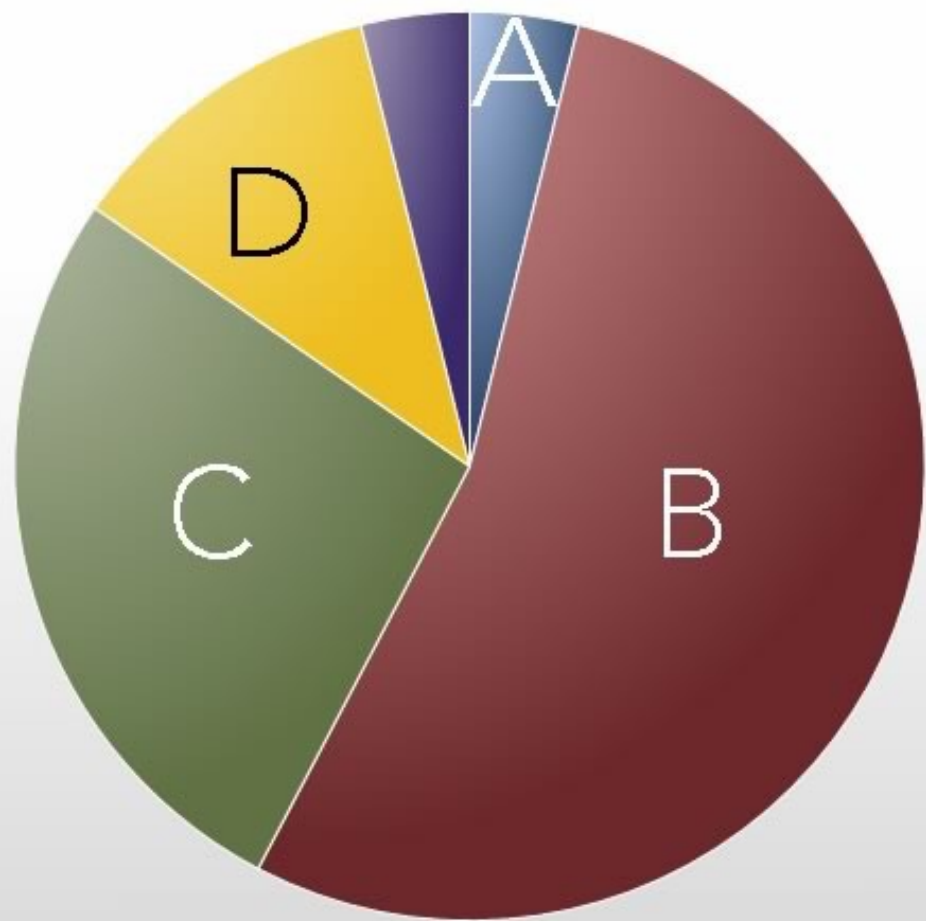
trait  
distribution  
traits

regulatory

librium  
object

How would a mutation in the gene that encodes release factor influence the cell?

- ☐ A. there would be no observable effect
- ☐ B. it would cause polypeptide synthesis to terminate prematurely
- ☐ C. it would lead to C-terminal extension to many (most) proteins
- ☐ D. it would lead to aberrant translation start sites
- ☐ no idea



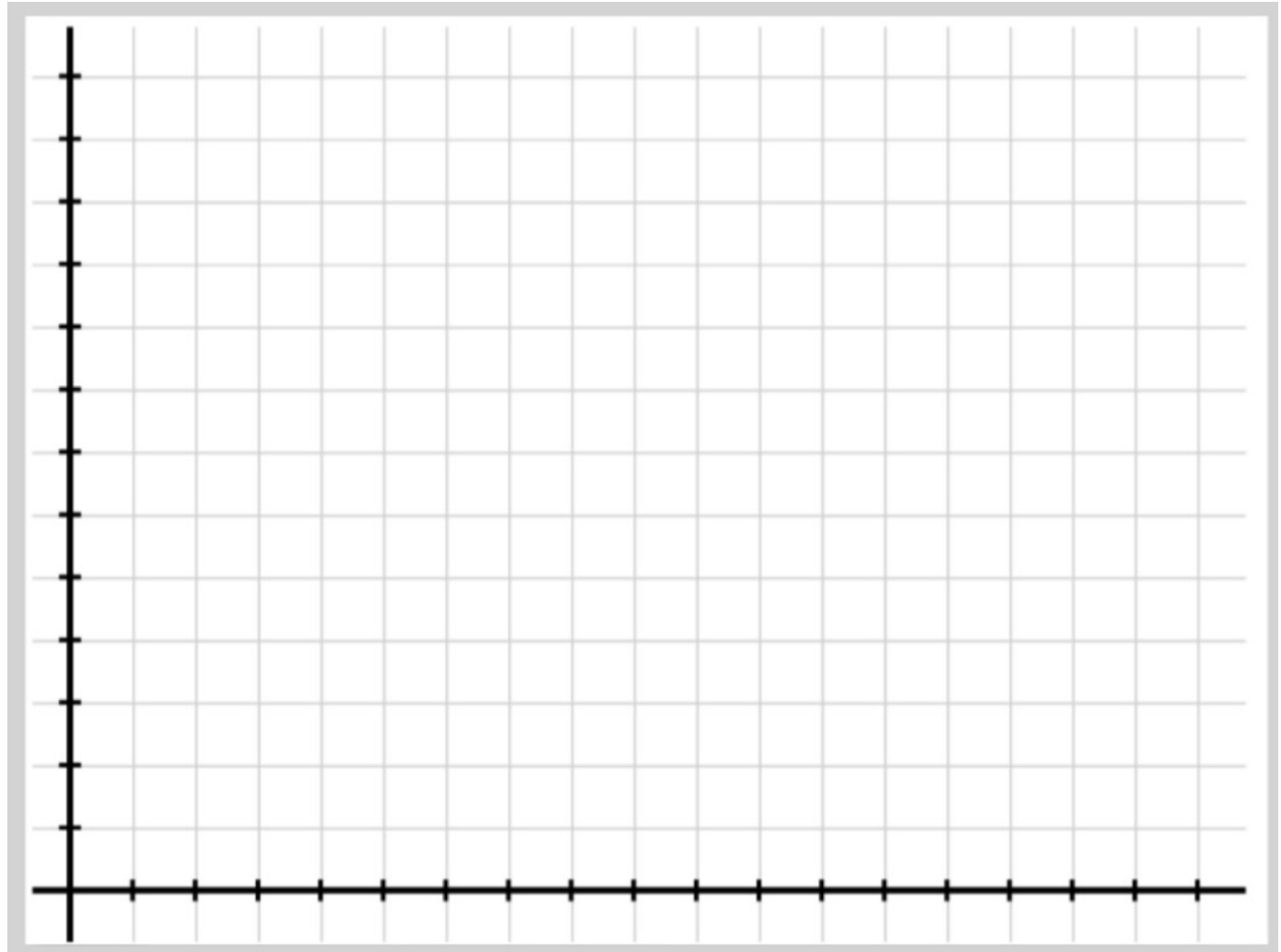
How could a mutation in a cell act to suppress a non-sense mutation?

mutation that change anti-codon of a specific tRNA  
multiple genes encoding tRNAs for that amino acid

Other factors regulate translation of RNA and its stability (half-life): endo-/exo-nucleases)

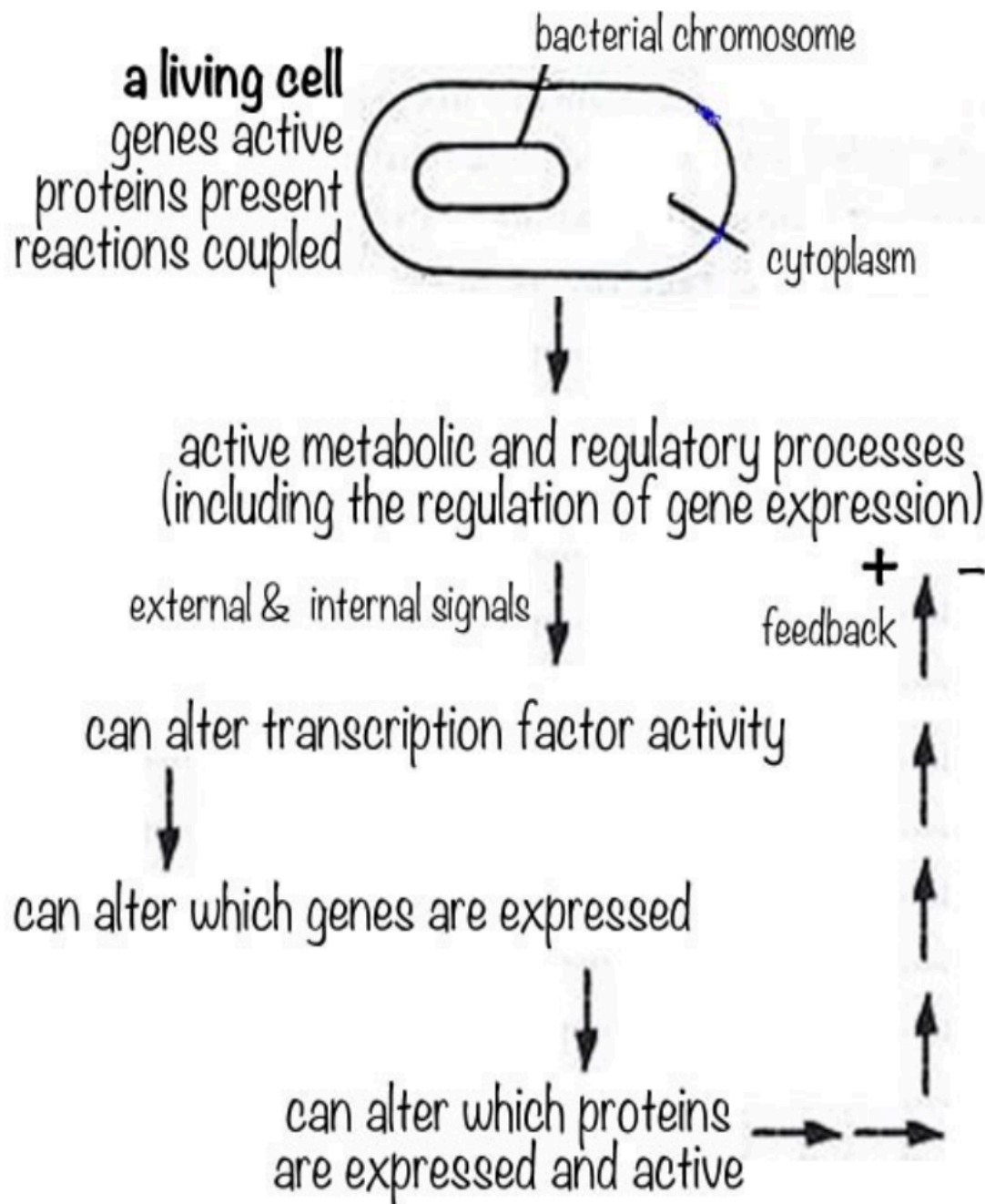
Similar factors/signals regulate polypeptide stability.

What does the term “half-life” mean?





Why is a RNA's or protein's life span stochastic (how is it like radioactive decay)?

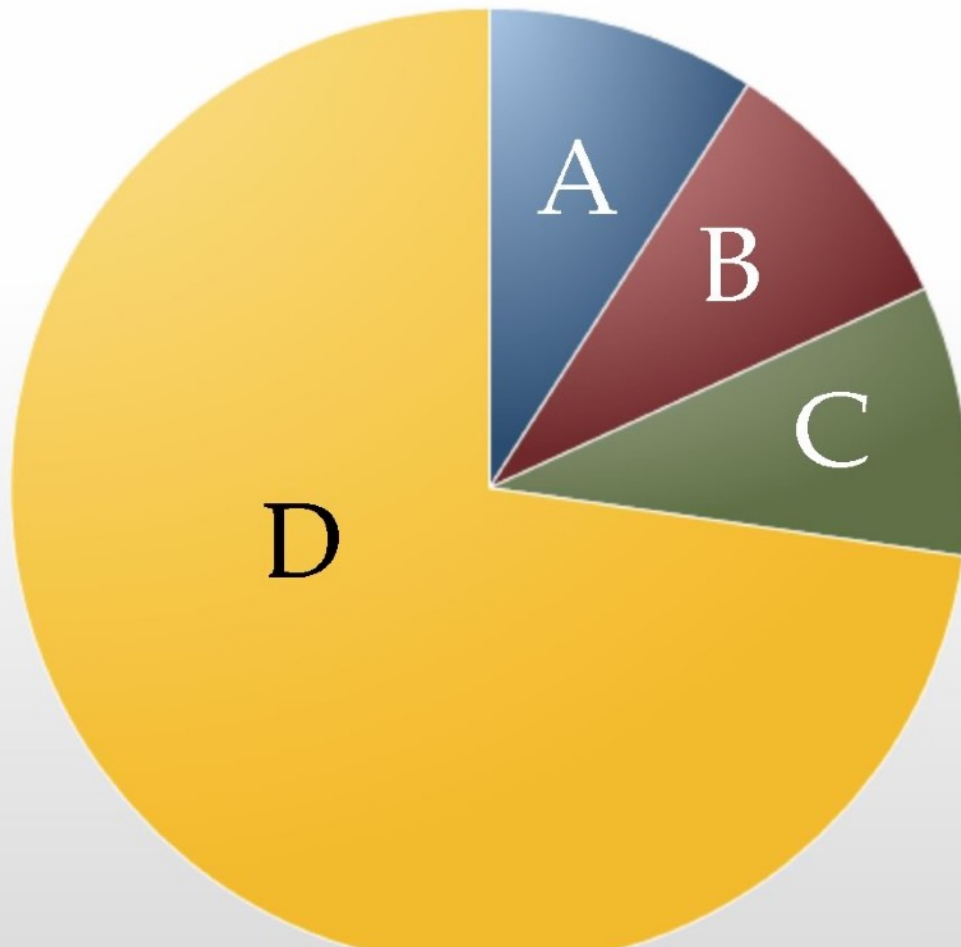


**tricky question to answer:**

There are a number of cases when either transcription and translation occur in bursts, that is, a number of RNAs or polypeptides are synthesized in a short time, followed by a quiet period with no synthesis, and then another burst. What kind of plausible mechanism can you propose (given what you know about transcription and translation, and molecular level behaviors) for this bursting phenomena?

**8.3 read p. 187-192.**

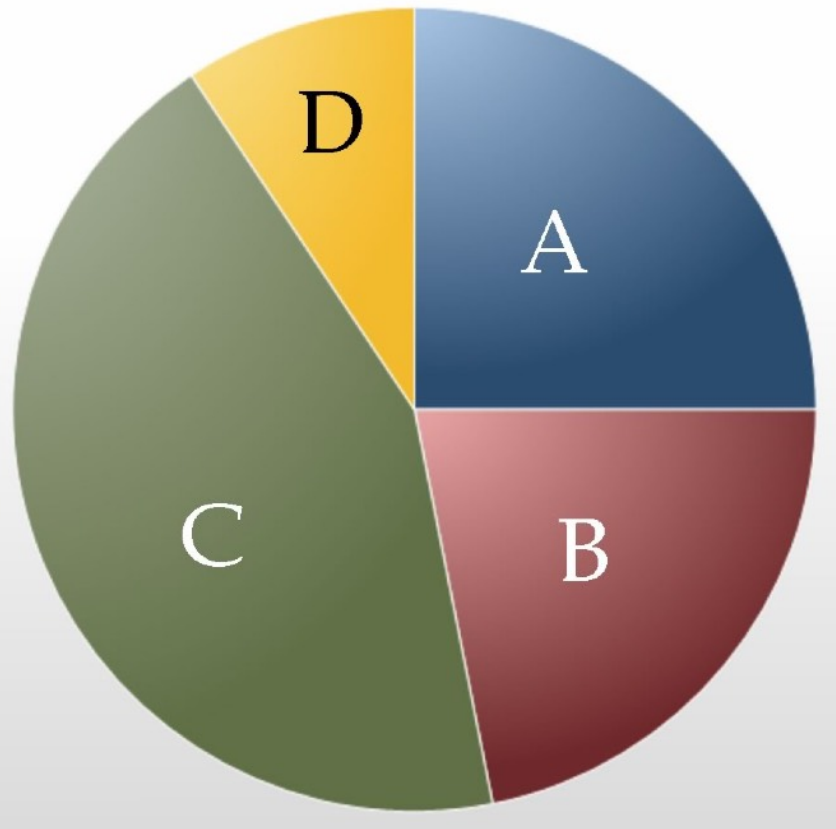
Eukaryotes differ from prokaryotes (eubacteria and archaea) by the presence of a nucleus. Within the nucleus, newly synthesized RNA molecules are modified before they are transported to the cytoplasm.



- ☐ the nucleus
- ☐ the cytoplasmic face of the plasma membrane
- ☐ the extracellular face of the plasma membrane
- ☐ throughout the cell
- ☒ throughout the cytoplasmic, but not within the nucleus
- ☐ no idea



As a polypeptide is synthesized it is initially inhibited from folding because it has to pass through a narrow channel in the ribosome. What do you think is pushing it through this channel?



- ☐ A. diffusion
- ☐ B. molecular motors within the channel
- ☐ C. the process of polypeptide synthesis
- ☐ D. the process of RNA synthesis

How does the presence of hydrophobic R groups impact the folding of a protein.  
Make a drawing of a soluble polypeptide with lots of hydrophobic R groups in water.

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Draw

Erase



Reset

A membrane protein often has regions that are in water and others that are within the membrane's interior; make a schematic of where the protein's hydrophobic R groups are likely to be localized in the two regions.

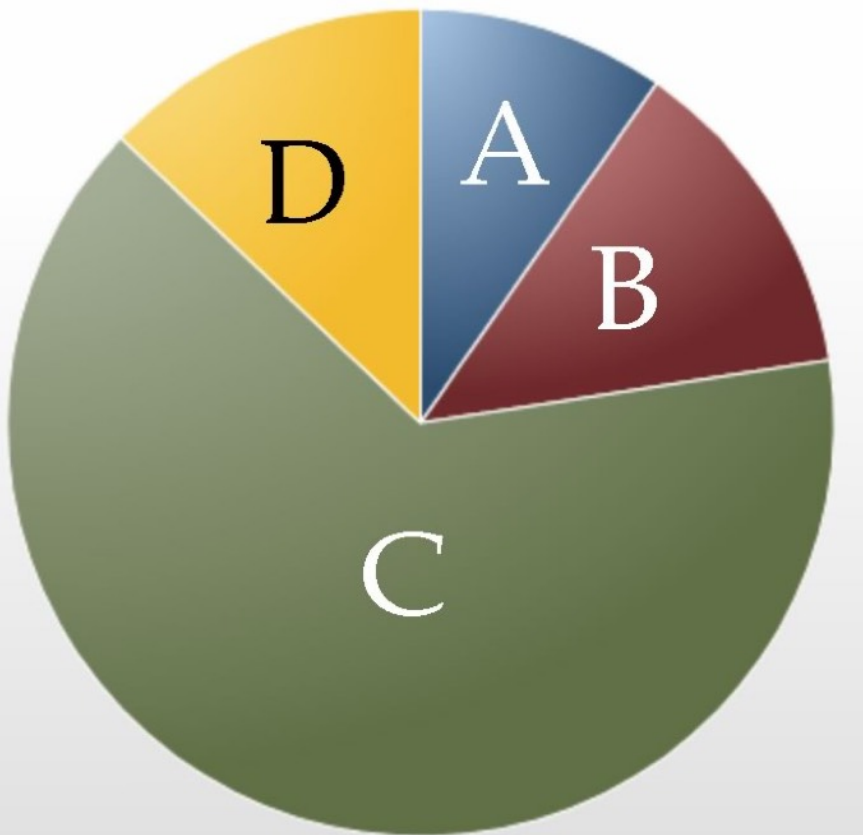
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Draw

Erase

Think about an aqueous (water-soluble) protein.

What type of missense mutation is likely to have a greater effect on its folding and function?



- ☐ change an interior hydrophilic amino acid to hydrophobic amino acid
- ☐ change an interior hydrophobic amino acid to a hydrophilic amino acid
- ☐ change an exterior hydrophilic amino acid to a hydrophobic amino acid
- ☐ impossible to predict
- ☐ I, personally, have no idea

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**An important clue is evolutionary conservation (what does that mean? how can I know whether a particular site is conserved?)**

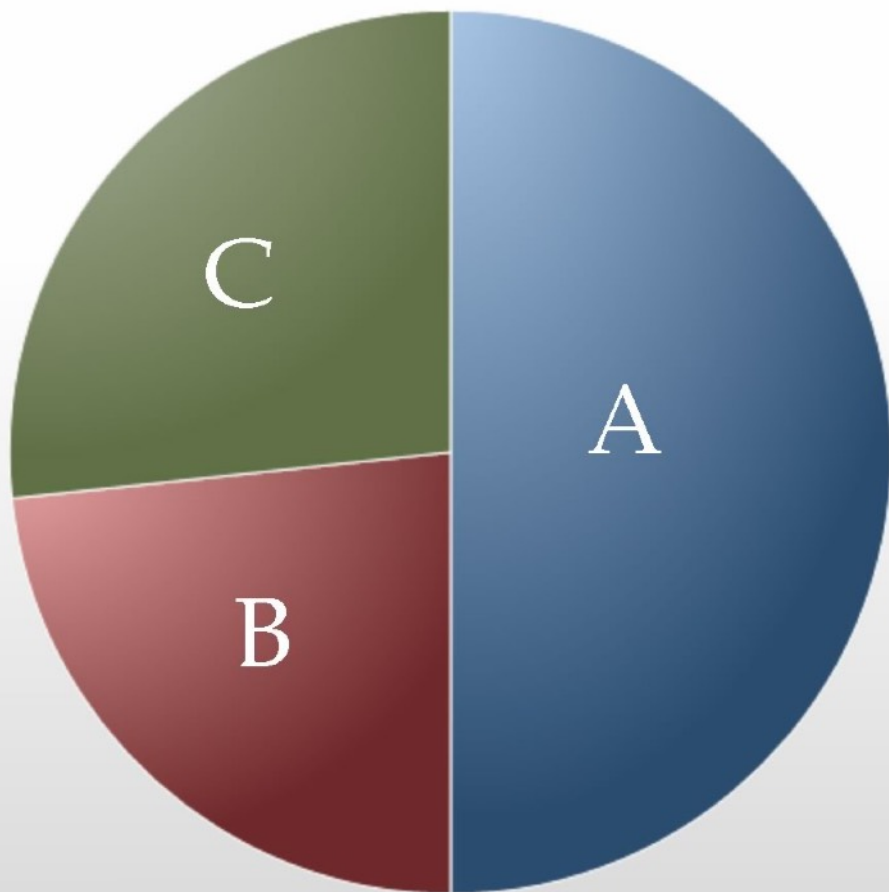


Consider an essential gene; in the absence of a functional gene product, the cell dies.

A mutation occurs that leads to a non-sense codon. Which situation is likely to have the highest probability of leading to the cell's death?

- ☐ the mutation is near the start of the coding sequence
- ☐ the mutation is near the end of the coding sequence
- ☐ the mutation influences the start of transcription
- ☐ impossible to predict

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## Questions to answer:

- **Why so many tRNA genes?** How, in the most basic terms, do different tRNAs differ from one another?
- How might the **concentration of various tRNAs** and the frequency of various codons influence the rate of polypeptide synthesis?
- What is the **minimal number** of different tRNA-amino acid synthetases in a cell?
- Would you expect a ribosome to make **mistakes** in amino acid incorporation or polypeptide termination? How are such mistake similar to and different from mutations?

## Questions to answer:

- How would you explain the **terms “up-stream” and “down-stream”** in terms of gene structure.
- What effects on polypeptide synthesis arise from neglecting **codon bias**?
- Why don't release factors cause the premature termination of translation at non-stop codons?

## Questions to answer:

- What will happen if a ribosome starts translating an mRNA at the "wrong" place?
- When analyzing the effects of a particular non-sense or mis-sense mutation (allele), what factors would you consider first?
- How would you go about reengineering an organism to incorporate non-biological amino acid in its proteins