AP Bio FRQ Fridays

2018 #2
Cell Communication, Tonicity, & Immune System

NOTES

FRQ Friday #5

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IL released

pore formed

IL cause immune response

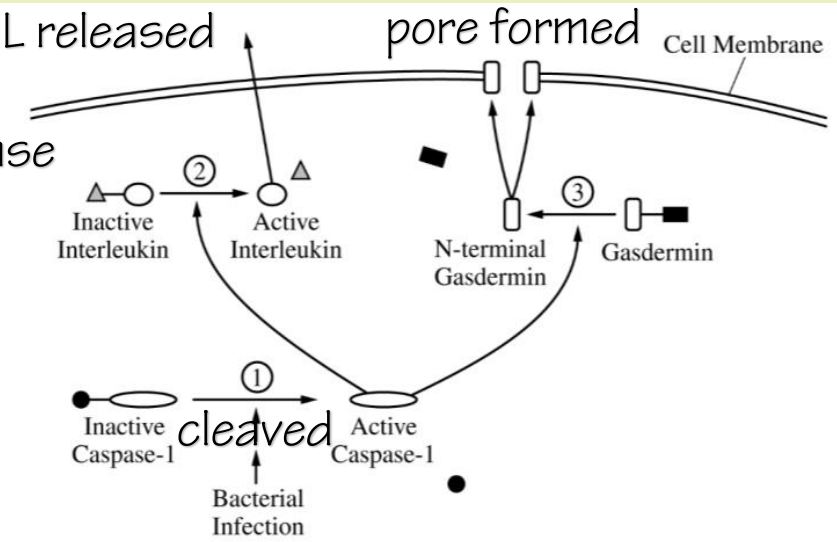

Figure 1. Cellular response to infection by pathogenic bacteria

Some pathogenic bacteria enter cells, replicate, and spread to other cells, causing illness in the host organism. Host cells respond to these infections in a number of ways, one of which involves activating particular enzymatic pathways (Figure 1). Cells normally produce a steady supply of inactive caspase-1 protein. In response to intracellular pathogens, the inactive caspase-1 is cleaved and forms an active caspase-1 (step 1). Active caspase-1 can cleave two other proteins. When caspase-1 cleaves an inactive interleukin (step 2), the active portion of the interleukin is released from the cell. An interleukin is a signaling molecule that can activate the immune response. When caspase-1 cleaves gasdermin (step 3), the N-terminal portions of several gasdermin proteins associate in the cell membrane to form large, nonspecific pores.

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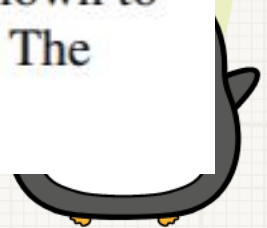
CELL FRACTIONS CONTAINING DIFFERENT CELLULAR PROTEINS

	Aconitase (Krebs cycle protein) Mitochondria	DNA polymerase Nucleus	GAPDH (glycolytic protein) Cytoplasm	Sodium-potassium pump Membrane	NF-κB (Immune response protein)
Whole cell sample	+	+	+	+	+
Fraction 1	+				
Fraction 2		+			+
Fraction 3			+		+
Fraction 4				+	

+ = presence of protein

Researchers created the model in Figure 1 using data from cell fractionation studies. In the experiments, various parts of the cell were separated into fractions by mechanical and chemical methods. Specific proteins known to be located in different parts of the cell were used as markers to determine the location of other proteins. The table below shows the presence of known proteins in specific cellular fractions.



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(a) **Describe** the effect of inhibiting step 3 on the formation of pores AND on the release of interleukin from the cell.

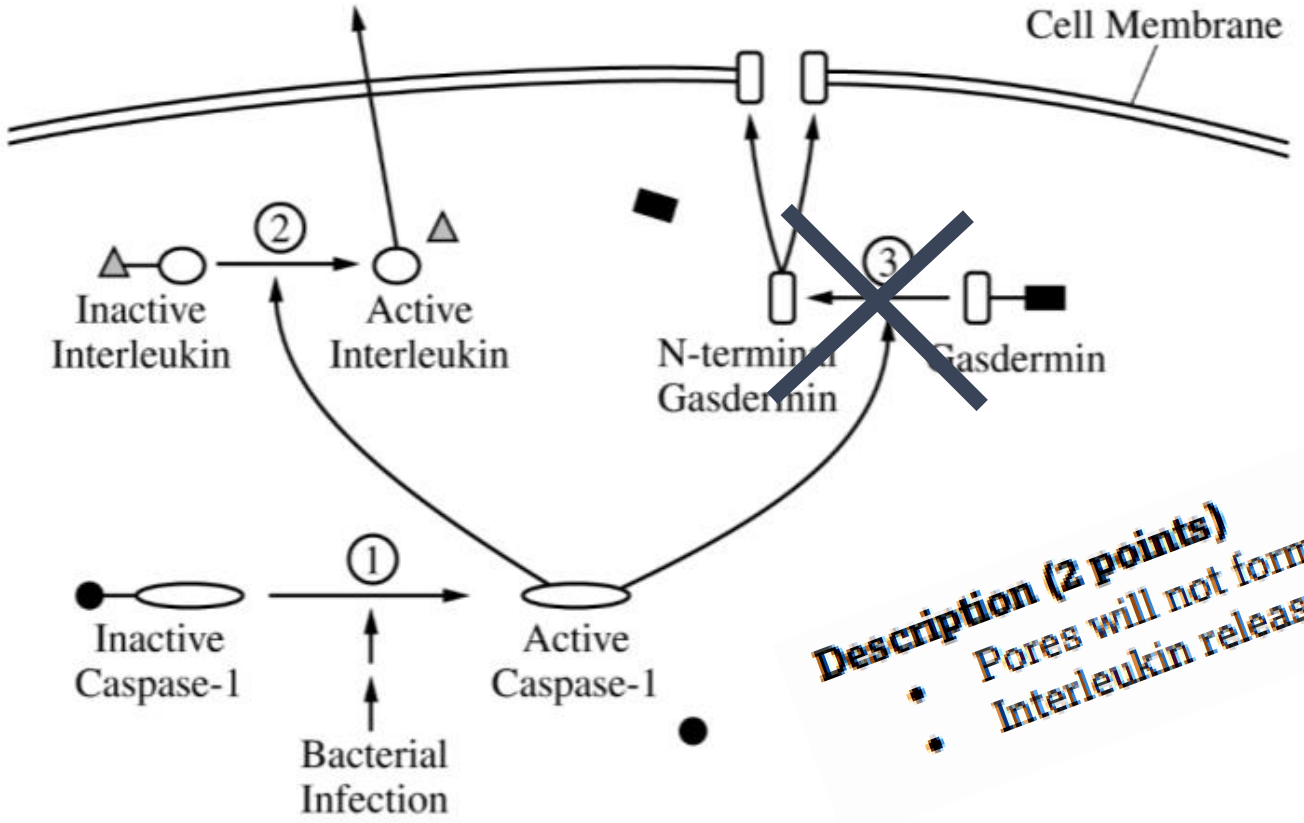

Figure 1. Cellular response to infection by pathogenic bacteria

Description (2 points)

- Pores will not form.
- Interleukin release will not be affected/interleukin release continues.

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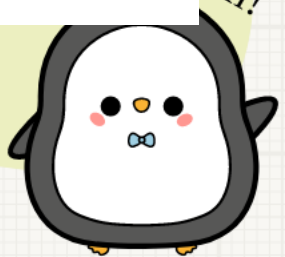
2.a. Inhibiting step 3, the cleavage of caspase activated gasdermin by caspase-1, will prevent the association of gasdermin proteins and the subsequent formation of pores in the cell membrane. This will not affect the release of interleukin from the cell because interleukin does not require the pores to leave the cell.



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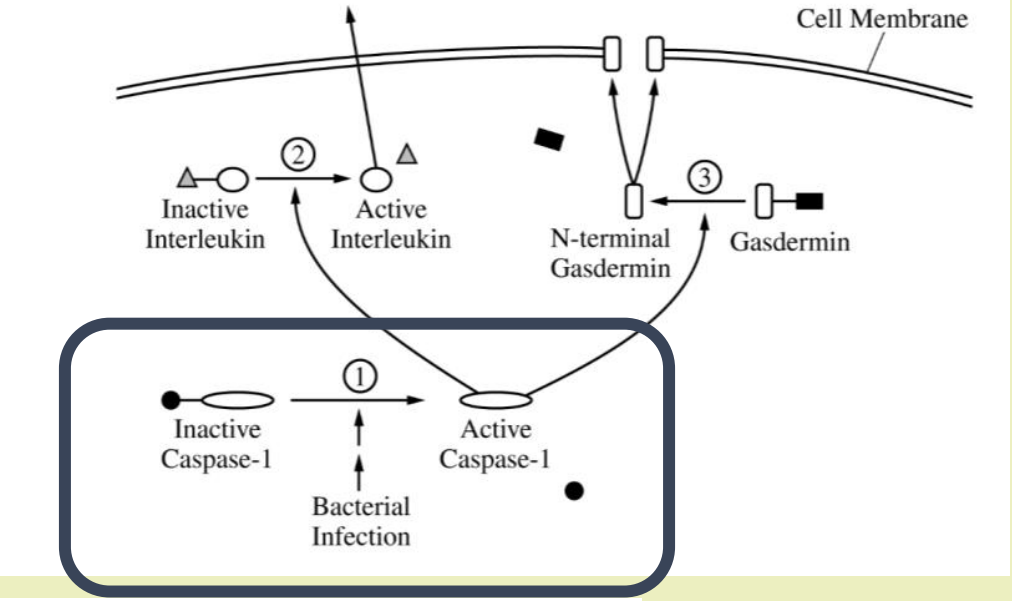
(b) **Make a claim** about how cleaving inactive caspase-1 results in activation of caspase-1. A student claims that preinfection production of inactive precursors shortens the response time of a cell to a bacterial infection. **Provide ONE reason** to support the student's claim.

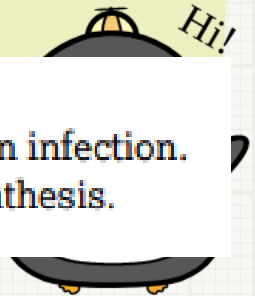


Claim (1 point)

- Removes inhibitor/repressor/inhibitory domain of protein
- Changes the shape/protein structure

Reasoning (1 point)

- Cleaving a precursor/protein/molecule is faster than making one upon infection.
- Cells do not have to wait for transcription and translation/protein synthesis.



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- Cells do not have to wait for transcription and translation/protein synthesis.

b. Cleaving inactive caspase-1 may alter the interaction of R-groups within the protein, resulting in a shape change in the cleaved molecule's tertiary structure that exposes an active site. If the cell did not constantly produce inactive caspase-1, the protein would have to be transcribed and translated before performing its function, a process which requires more enzymes and more time than a simple cleavage of a polypeptide.

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(c) A student claims that the NF-κB protein is located in the cytoplasm until the protein is needed for transcription. **Justify** the student's claim with evidence. **Identify TWO** fractions where N-terminal gasdermin would be found in cells infected with pathogenic bacteria.

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Fraction 3			+		+
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Justification (1 point)

- NF-*k*B and glycolytic enzymes/GAPDH are found together (in the cytoplasm).

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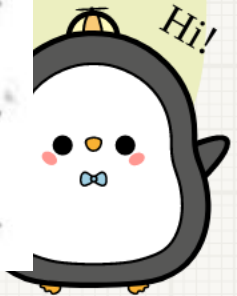
Justification (1 point)

- NF-κB and glycolytic enzymes/GAPDH are found together (in the cytoplasm).

~~and fraction 3, the cytosol~~

f: The student's claim is correct because fraction 2, the nucleus, tested positive for NF-κB protein. Fraction 2 is the nucleus because it contains DNA polymerase, an enzyme found only in the nucleus where DNA replication occurs and NF-κB would be needed to regulate transcription. Fraction 3 is the ~~cytosol~~ cytoplasm because this fragment tested positive for glycolytic proteins which aid in glycolysis, a process performed in the cytoplasm. N terminal gondesmin would be found in fraction 3 and fraction 4



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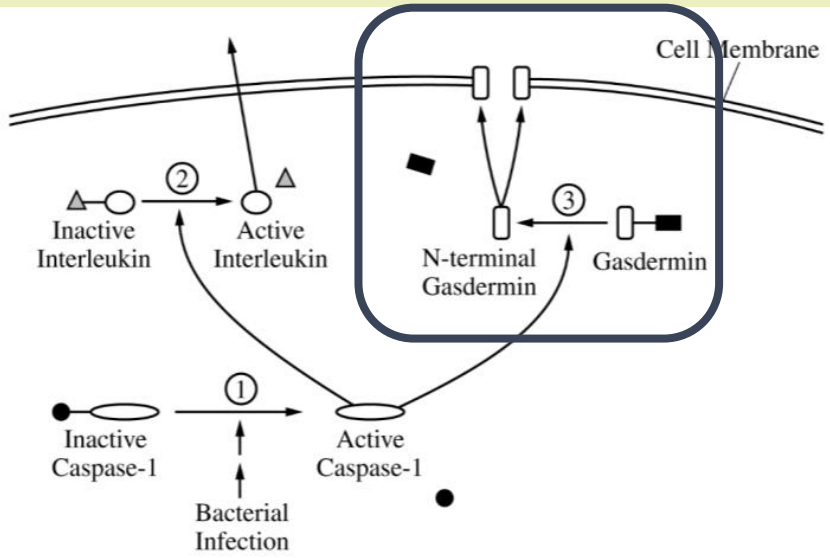

Identification (2 points)

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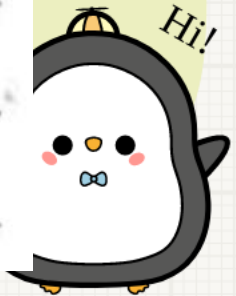

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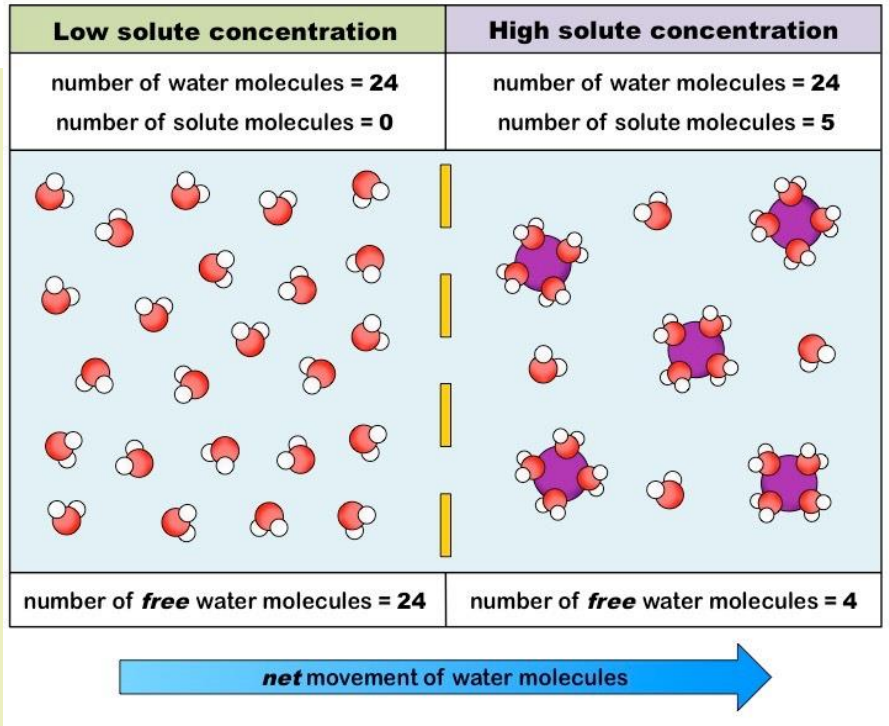
(d) **Describe** the most likely effect of gasdermin pore formation on water balance in the cell in a hypotonic environment.

HYPOtonic



HYPERtonic

Description (1 point)

- Water enters the cell.

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(d) **Describe** the most likely effect of gasdermin pore formation on water balance in the cell in a hypotonic environment.

Description (1 point)

- Water enters the cell.

d. In a hypotonic environment, water will move into the cell via osmosis through the pores because by definition, a hypotonic environment would have a higher water potential than the cell

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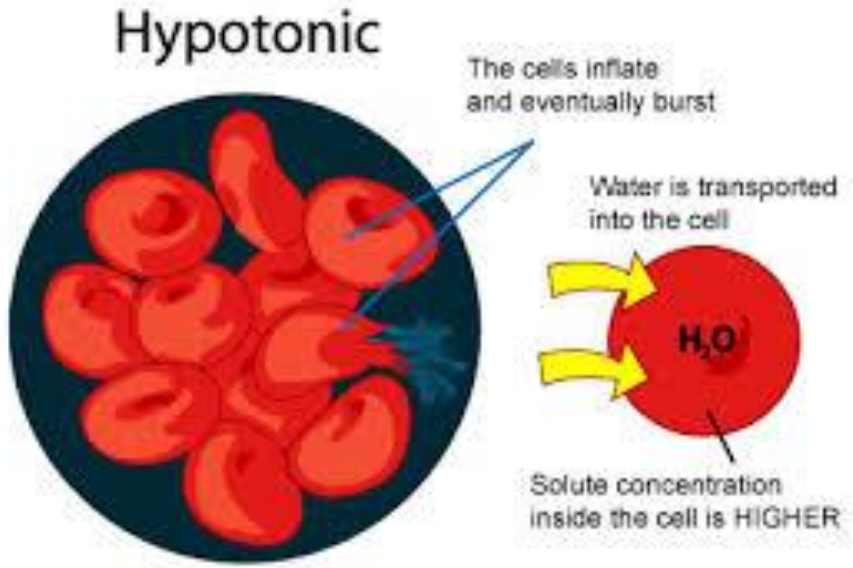
(e) **Explain** how gasdermin pore formation AND interleukin release contribute to an organism's defense against a bacterial pathogen.



Explanation (2 points)

- Cell lysis destroys infected cells OR cell lysis prevents bacteria from replicating.
- Interleukin signaling will stimulate immune cells/components of the immune system (to destroy the infected cells or bacteria).

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e. Gasdermin pore formation causes the flow of water into the cell, which may cause the infected cell to burst. This would prevent the spread of the infection. because phagocytes could the digest the components of the cell along with the pathogens. The release of interleukins activates the adaptive immune response by stimulating B and T lymphocytes to divide and produce antibodies or kill infected cells, respectively.