

# FRQ Friday – 3/5

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2018 #2

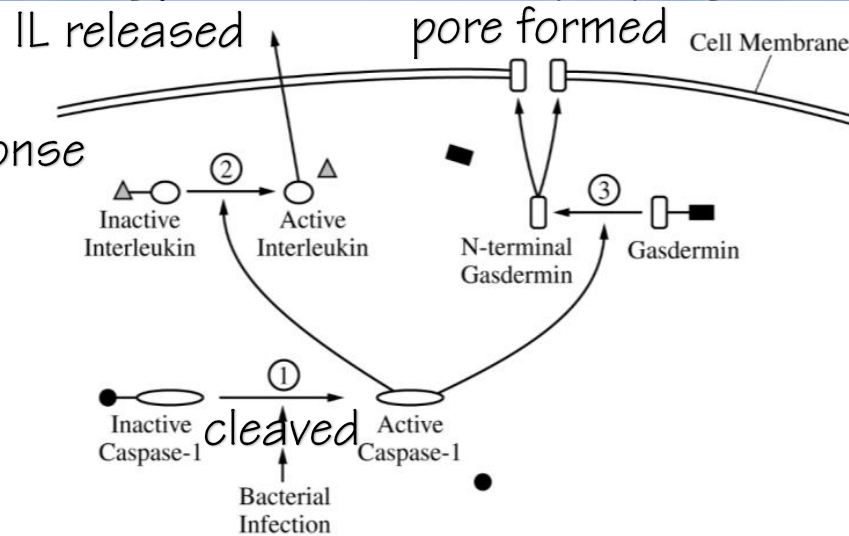
2018 #8



# FRQ 2018 #2

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IL cause  
immune response



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- $\kappa$ B (Immune response protein)
Whole cell sample	+	+	+	+	+
Fraction 1	+				
Fraction 2		+			+
Fraction 3			+		+
Fraction 4				+	

+ = presence of protein

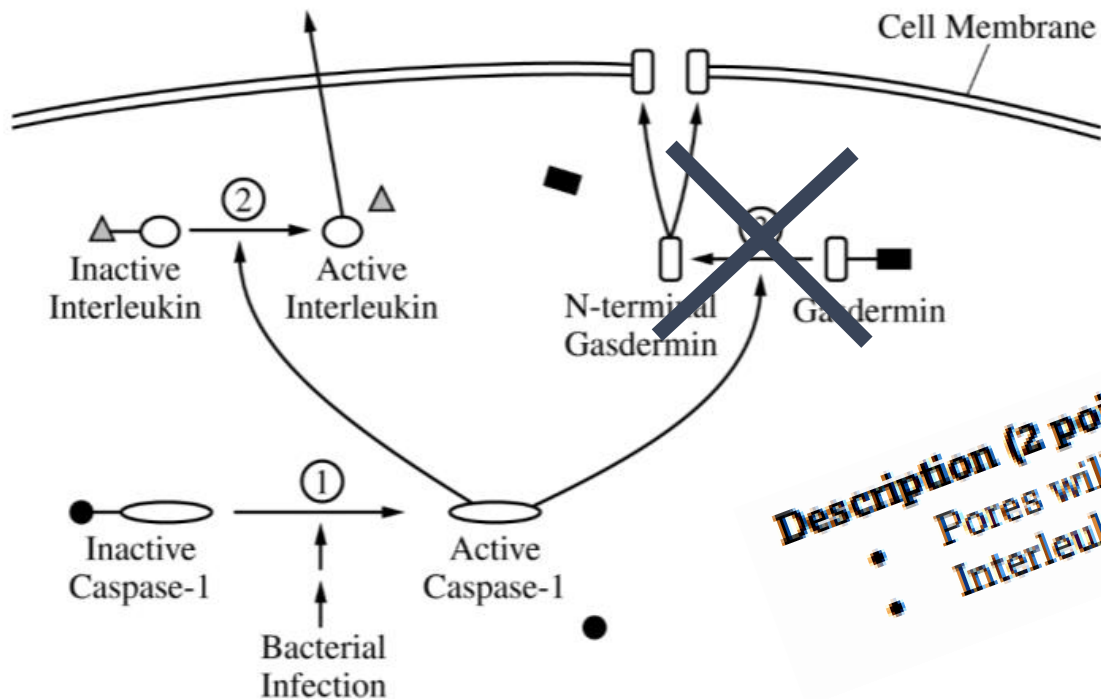
Researchers created the model in Figure 1 using data from cell fractionation studies. In the experiments, various parts of the cell were separated into fractions by mechanical and chemical methods. Specific proteins known to be located in different parts of the cell were used as markers to determine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



**Description (2 points)**

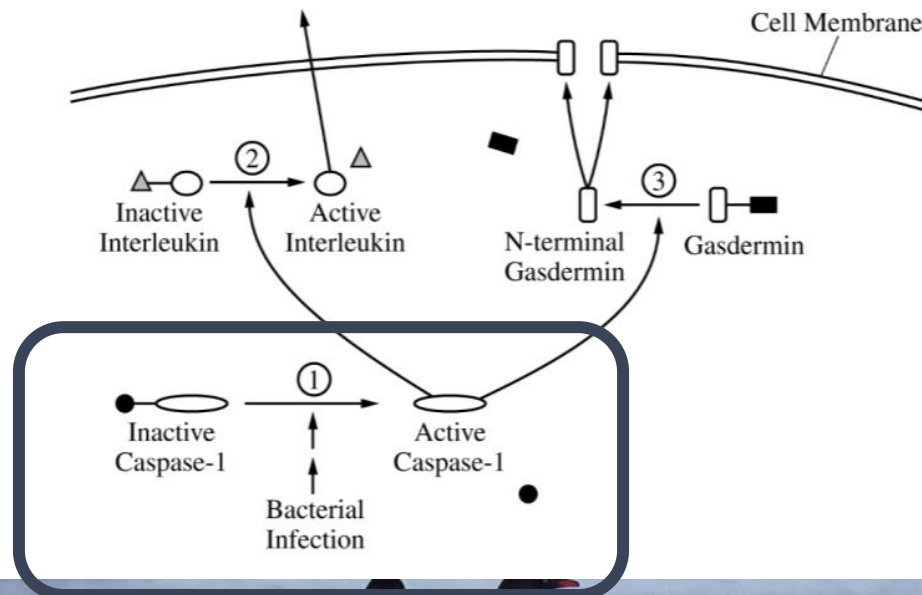
- Pores will not form.
- Interleukin release will not be affected/interleukin release continues.

Figure 1. Cellular response to infection by pathogenic bacteria

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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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(c) A student claims that the NF- $\kappa$ B 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.

CELL FRACTIONS CONTAINING DIFFERENT CELLULAR PROTEINS

	Aconitase (Krebs cycle protein)	DNA polymerase	GAPDH (glycolytic protein)	Sodium- potassium pump	NF- $\kappa$ B (Immune response protein)
Whole cell sample	+	+	+	+	+
Fraction 1	+				
Fraction 2		+			+
Fraction 3			+		+
Fraction 4				+	

+ = presence of protein

### Justification (1 point)

- NF- $\kappa$ B and glycolytic enzymes/GAPDH are found together (in the cytoplasm).

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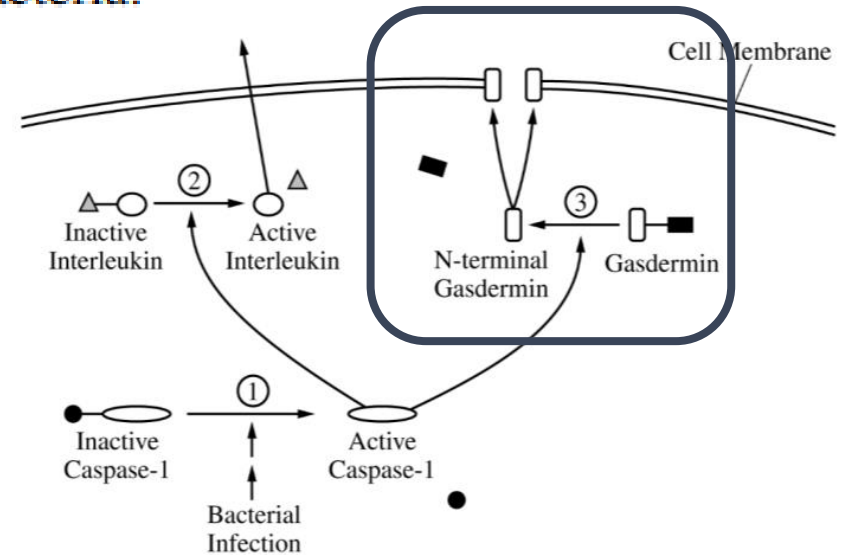
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(c) A student claims that the NF- $\kappa$ B protein is located in the cytoplasm until the protein is needed for transcription. **Justify** the student's claim with evidence. **Identify TWO** fractions where N-terminal gasdermin would be found in cells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

CELL FRACTIONS CONTAINING DIFFERENT CELLULAR PROTEINS

	Aconitase (Krebs cycle protein)	DNA polymerase	GAPDH (glycolytic protein)	Sodium- potassium pump	NF- $\kappa$ B (Immune response protein)
Whole cell sample	+	+	+	+	+
Fraction 1	+				
Fraction 2		+			+
Fraction 3			+		+
Fraction 4				+	

+ = presence of protein



## Identification (2 points)

- Fraction 3
- Fraction 4

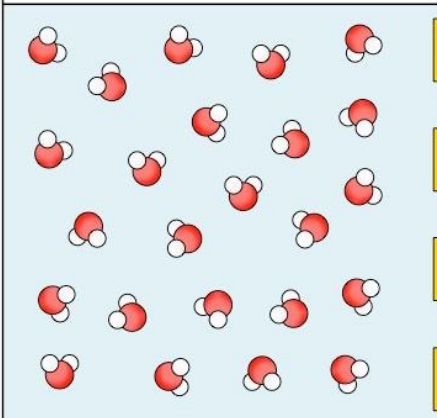
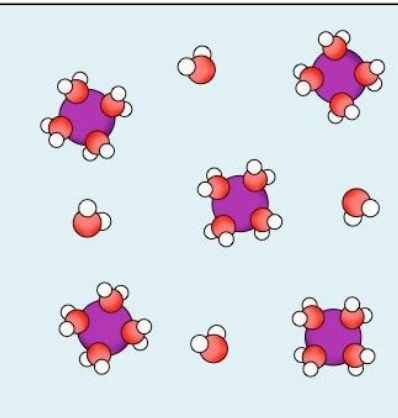


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(d) Describe the most likely effect of gasdermin pore formation on water balance in the cell in a hypotonic environment.

HYPOTonic

Low solute concentration	High solute concentration
number of water molecules = 24 number of solute molecules = 0	number of water molecules = 24 number of solute molecules = 5
	
number of <b>free</b> water molecules = 24	number of <b>free</b> water molecules = 4

HYPERTonic

net movement of water molecules

**Description (1 point)**

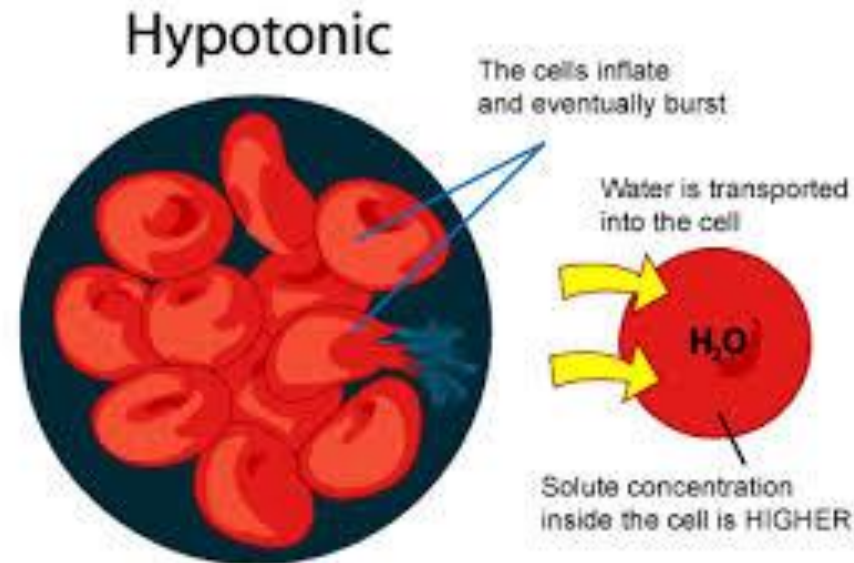
- Water enters the cell.



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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).

# FRQ 2018 #8

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Acetylcholine receptor (AChR) proteins are found at the synapse between neurons and skeletal muscle cells. Acetylcholine released from neurons binds to a specific site on the receptor proteins, which causes an ion channel in the receptors to open and allow sodium ions ( $\text{Na}^+$ ) to enter muscle cells. The resulting depolarization of muscle cells initiates muscle contractions. Another molecule, nicotine, can also bind to certain types of AChR proteins and activate the receptors.

A researcher is investigating two different types of AChR proteins: type 1 and type 2. To determine which stimuli activate the receptors, the researcher exposes muscle cells expressing the different types of receptor proteins to stimuli and observes the results indicated in Table 1.

TABLE 1. RESPONSE OF AChR PROTEINS TO DIFFERENT STIMULI

AChR Protein Type	Acetylcholine	Nicotine
Type 1	+	+
Type 2	+	-

+ indicates activation

- indicates no activation

TABLE 1. RESPONSE OF AChR PROTEINS TO DIFFERENT STIMULI

AChR Protein Type	Acetylcholine	Nicotine
Type 1	+	+
Type 2	+	-

+ indicates activation  
- indicates no activation

(a) Describe the difference in the structure AND function between AChR type 1 and AChR type 2.

Structure (1 point maximum)	Function (1 point maximum)
Binding sites differ in shape/ specificity/number	<ul style="list-style-type: none"> <li>Differential binding of molecules to type 1 and type 2 receptors</li> <li>Activated by one (ACh) molecule or both (ACh and nicotine) molecules</li> <li>No difference in response (both open channels OR both result in depolarization OR both cause muscle contraction)</li> </ul>
Differential binding of molecules to type 1 and type 2 receptors	<ul style="list-style-type: none"> <li>Activated by one (ACh) or both (ACh and nicotine) molecules</li> <li>No difference in response (both open channels OR both result in depolarization OR both cause muscle contraction)</li> </ul>
Receptors activated by one (ACh) or both (ACh and nicotine) molecules	<ul style="list-style-type: none"> <li>No difference in response (both open channels OR both result in depolarization OR both cause muscle contraction)</li> </ul>

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(b) Acetylcholinesterase is an enzyme that breaks down acetylcholine in the synapse. Describe the effect of inhibiting acetylcholinesterase on the muscle cells with AChR type 2.

TABLE 1. RESPONSE OF AChR PROTEINS TO DIFFERENT STIMULI

AChR Protein Type	Acetylcholine	Nicotine
Type 1	+	+
Type 2	+	-

+ indicates activation  
- indicates no activation

## Description (1 point)

- Continued activation
- Repeated opening of sodium channels OR repeated depolarization OR muscle spasms

Next FRQ Friday (3/12)

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2019 #4

2013 #8

