

Cell Communication

1. You discover a receptor that forms a high affinity interaction with its ligand. In which of the following types of cell communication is the receptor most likely involved?
 - A. Paracrine
 - B. Endocrine
 - C. Neurotransmission
 - D. Direct cell-to-cell contact

2. Which of the following receptors would generate the fastest cellular response?
- A. Steroid receptor
 - B. Receptor tyrosine kinase
 - C. Ligand-gated ion channel
 - D. G-protein coupled receptor

3. In a signaling pathway that utilizes a heterotrimeric GTP-binding protein which of the following components could activate a downstream pathway?

A. G_{α} -GDP

B. $G_{\alpha\beta\gamma}$

C. $G_{\beta\gamma}$

D. G_{β}

4. Which of the following is the best example of negative feedback?

A. Protein kinase A activates phosphodiesterase

B. MAP kinase activates a guanine-nucleotide exchange factor for Ras

C. Protein kinase C activates phospholipase C

D. MAP kinase kinase phosphorylates Map kinase

5. Cholera toxin adds an ADP-ribose to G_{as} which inhibits its GTPase activity. Which of the following is the most likely effect of cholera toxin?
- A. Increased concentration of cAMP
 - B. Decreased concentration of cAMP
 - C. Increased concentration of Ras-GTP
 - D. Decreased concentration of Ras-GTP

6. Asthma is often managed through the administration of corticosteroids which decrease the activity of immune cells. Corticosteroids affect which process in immune cells?
- A. Protein Kinase A activity
 - B. Map Kinase activity
 - C. Gene expression
 - D. Concentration of cytosolic calcium

7. You discover a domain in phospholipase C that resembles a GTPase-activating protein. The most likely effect of this domain is which of the following?
- A. Increase amount of active phospholipase C
 - B. Increase amount of $G_{\beta\gamma}$
 - C. Decrease amount of Ras-GTP
 - D. Decrease amount of G_{α} -GTP

8. A few minutes after stimulating a G-protein coupled receptor with its ligand which would of the following would have the highest concentration?
- A. Receptor-ligand complex
 - B. G_{α} -GTP
 - C. cAMP
 - D. Phosphodiesterase

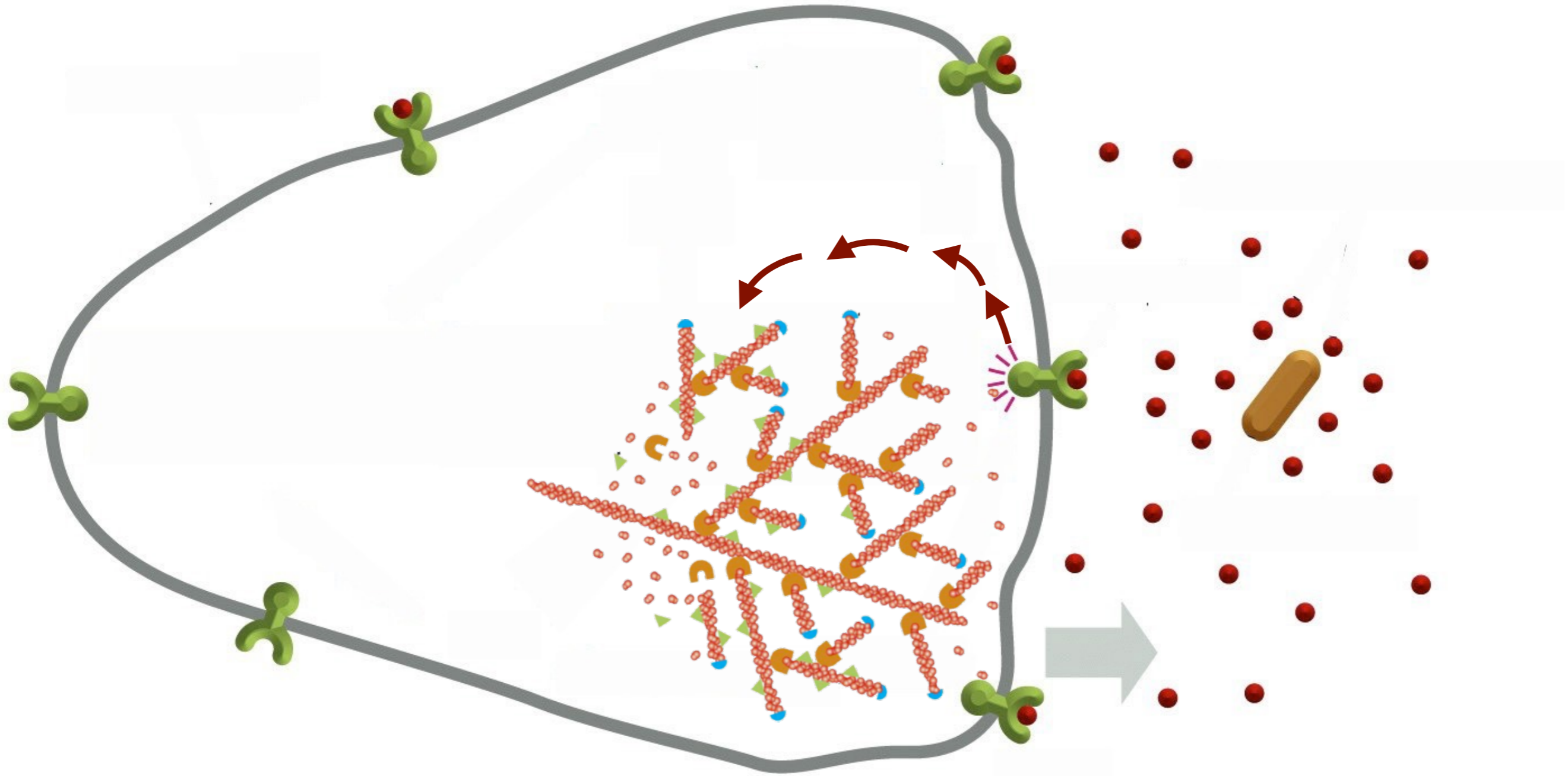
9. You are studying a signaling pathway a G-protein coupled receptor. You calculate the response to the receptor's ligand by measuring the increase in cytosolic calcium. Curiously, you find that the increase in cytosolic calcium is less after each addition of the ligand. The most likely cause is which of the following?
- A. Phosphorylation of the G-protein coupled receptor
 - B. Depletion of calcium stores in the ER
 - C. Inactivation of calcium channels
 - D. Decrease catalytic activity in $G\alpha_s$

Application Questions

Neutrophils chase bacteria through tissues or across a substratum.

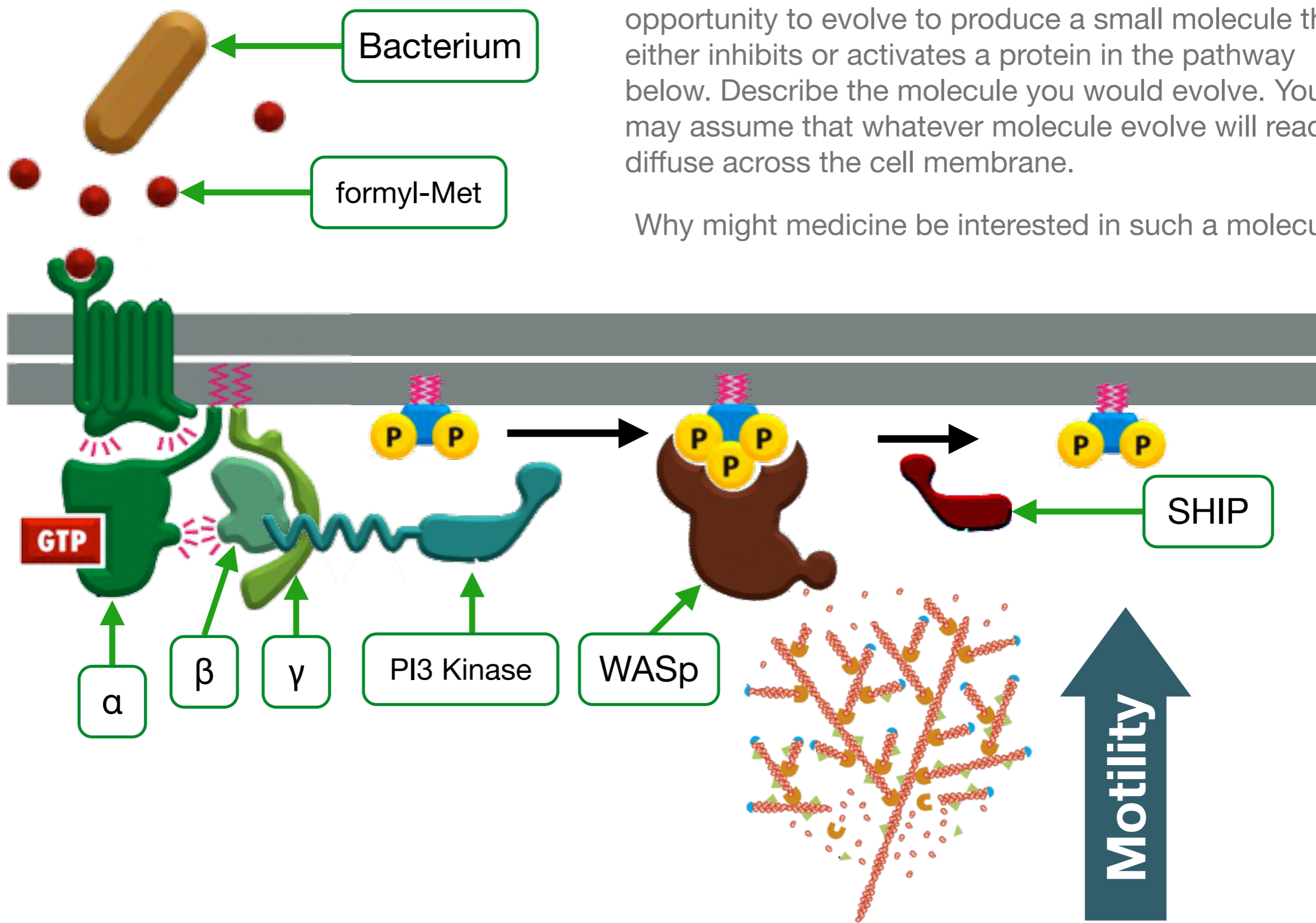


Bacterial molecules activate signaling pathway in neutrophils to initiate actin polymerization.



1. Study the diagram of the signaling pathway that allows a neutrophil to track a bacterium. You are a bacterium trying to escape from a neutrophil. You have the opportunity to evolve to produce a small molecule that either inhibits or activates a protein in the pathway below. Describe the molecule you would evolve. You may assume that whatever molecule evolve will readily diffuse across the cell membrane.

Why might medicine be interested in such a molecule?



2. A patient presents with shortness of breath, fatigue and swelling in their legs. The patient's systolic blood pressure is less than 90 mm HG and tests indicate acute heart failure with reduced cardiac output. You recall hearing that epinephrine increases the contractility of cardiomyocytes, and you reason that increasing contractility of cardiomyocytes will increase cardiac output. Epinephrine binds the beta-adrenergic receptor, which is a G-protein coupled receptor, on cardiomyocytes. Diagram a pathway that connects the beta-adrenergic receptor with an increase in cardiomyocyte contractility.

Reviewing the patient's history, you discover the patient has been taking a beta-blocker to control an arrhythmia. The beta-blocker binds the beta-adrenergic receptor and prevents its activation by epinephrine. Is there an alternative method to activate the pathway and increase contractility in cardiomyocytes?