

2. Many insects rely on pheromones (chemical signals) that are released by the females to find mating partners. Scientists hypothesize that, in a certain type of moth, the behavior of male moths in response to pheromones is regulated by the extracellular signaling molecule 20E.
- A. Many receptors are embedded in the plasma membrane. **Describe** the polarity of the portion of the receptor that is inside the membrane.

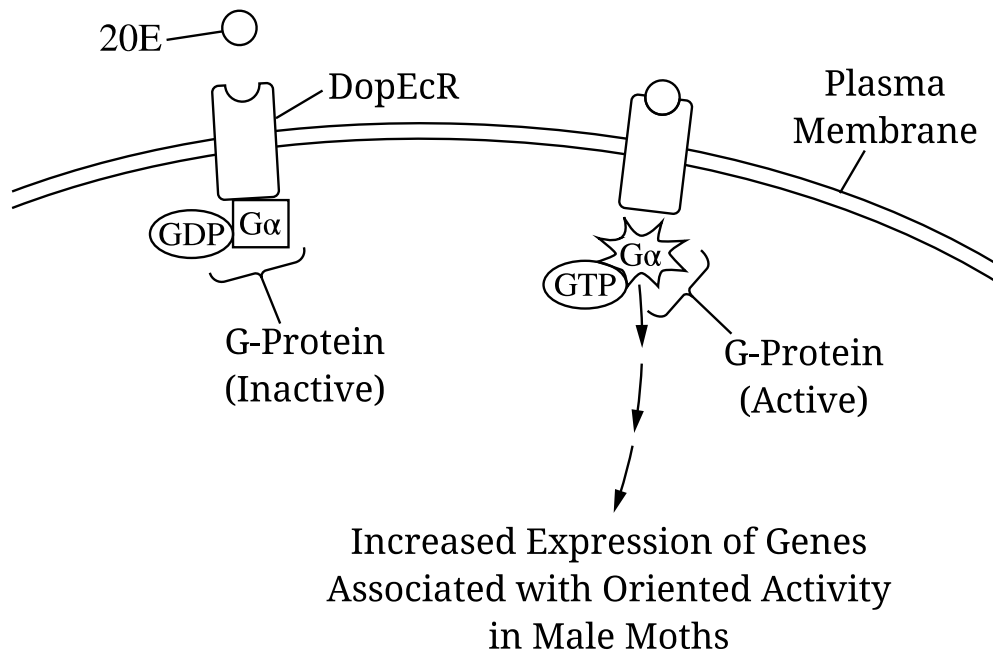
To investigate whether the binding of 20E to its receptor, DopEcR, affects behavior in moths, scientists injected male moths with saline (control solution) or with small interfering RNA molecules (siRNAs) that inhibit the expression of the gene encoding DopEcR. The scientists then exposed the moths to the pheromone and determined the percent of total time observed that the moths engaged in general activity, defined as movement in any direction. The scientists also determined the percent of the general activity time that the moths spent in oriented activity, defined as movement toward an area of high pheromone concentration (Table 1).

Table 1. Average General and Oriented Activity in Male Moths Injected With Saline or siRNA Molecules

Treatment	General Activity (percent of total time observed, average $\pm 2SE_{\bar{x}}$)	Oriented Activity (percent of general activity, average $\pm 2SE_{\bar{x}}$)
Male moths injected with saline (control solution)	95 $\pm$ 5	60 $\pm$ 4
Male moths injected with siRNAs that inhibit expression of the gene encoding DopEcR	90 $\pm$ 8	25 $\pm$ 6

DopEcR is a G protein-coupled receptor. When 20E binds to DopEcR, GTP displaces the GDP bound to the G protein, and a signaling pathway is activated. The scientists hypothesize that this leads to the transcription of genes associated with the oriented activity observed in the male moths (Figure 1).

Figure 1. A simplified model of a signaling pathway activated by the binding of 20E to its receptor, DopEcR



B.

- Using the template in the space provided for your response, **construct** an appropriate type of graph that represents the data in Table 1. Your graph should be appropriately plotted and labeled.
- Based on the data in Table 1, **determine** the type of activity that was affected by inhibiting the expression of the DopEcR receptor.

C.

- Based on Table 1, **identify** the treatment group in which the oriented activity was greater than 50% of the general activity.
- The scientists studied some moths with a mutation in the gene encoding the G protein. The mutation prevents GTP from displacing the GDP bound to the G protein. Based on Figure 1, **predict** the effect of this mutation on the oriented activity in male moths exposed to the pheromone.

Expression of the gene encoding DopEcR is low in the male moths during their first few days as adults, when they are sexually immature. Gene expression rapidly increases as the moths reach sexual maturity. The scientists claim that this increase in gene expression increases the likelihood of males finding females with whom to mate.

D.

- Use evidence from the information provided to **support** the scientists' claim.
- Based on Figure 1, **explain** how an inhibitor of the DopEcR pathway might serve as an effective chemical to protect crops from moth damage.

1. Polycystic kidney disease (PKD) is an inherited disease that causes water loss from the body and affects cell division in the kidneys. Because water movement across cell membranes is related to ion movement, scientists investigated the role of the Na^+/K^+ ATPase (also known as the sodium/potassium pump) in this disease. Ouabain, a steroid hormone, binds to the Na^+/K^+ ATPase in plasma membranes. Individuals with PKD have a genetic mutation that results in an increased binding of ouabain to the Na^+/K^+ ATPase. The scientists treated normal human kidney (NHK) cells and PKD cells with increasing concentrations of ouabain and measured the number of cells (Figure 1) and the activity of the Na^+/K^+ ATPase (Figure 2) after a period of time. The scientists hypothesized that a signal transduction pathway that includes the protein kinases MEK and ERK (Figure 3) may play a role in PKD symptoms.

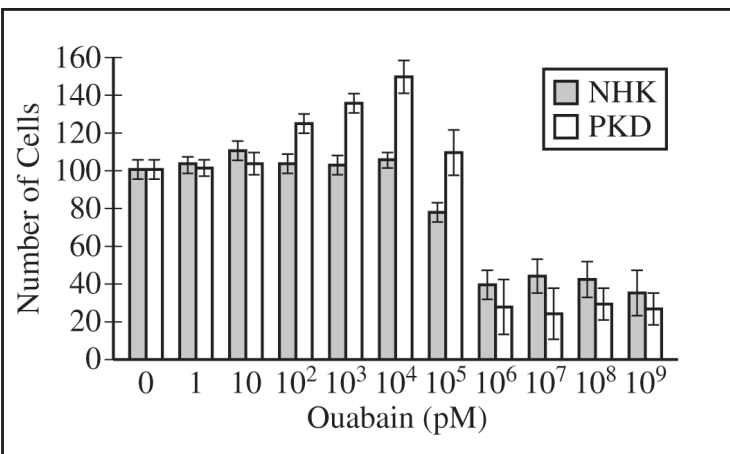


Figure 1. Cell number compared with the number of cells at 0 pM ouabain. Normal human kidney (NHK) cells and polycystic kidney disease (PKD) cells were treated with increasing concentrations of ouabain. Error bars represent $\pm 2SE_{\bar{x}}$.

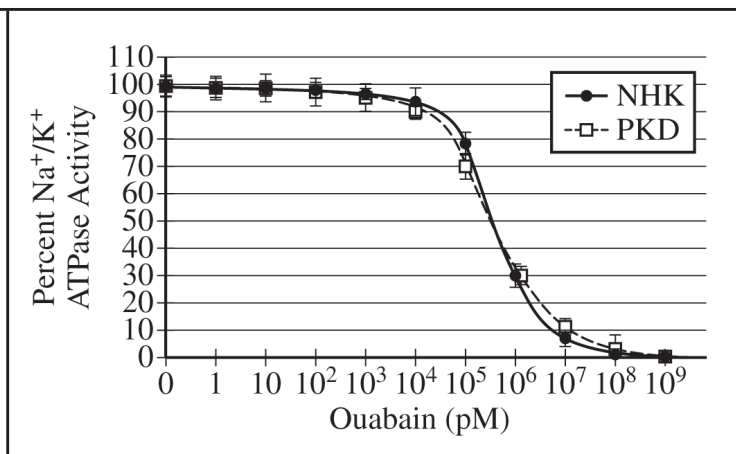


Figure 2. Percent Na^+/K^+ ATPase activity of NHK and PKD cells treated with increasing concentrations of ouabain. Error bars represent $\pm 2SE_{\bar{x}}$.

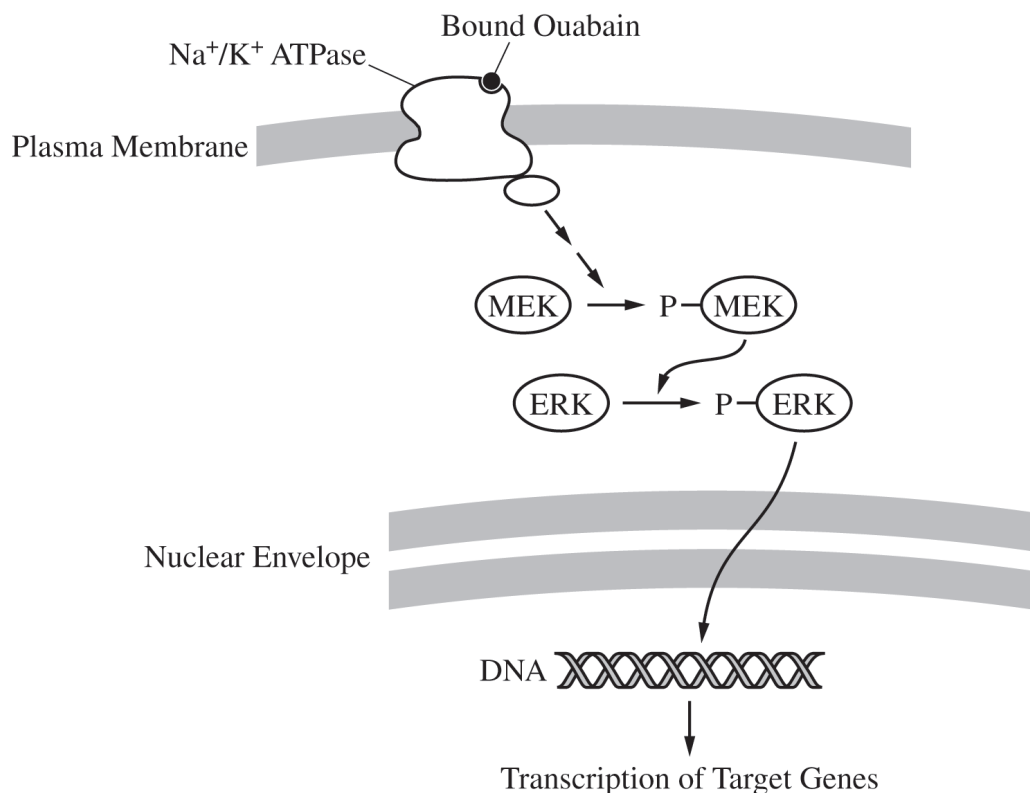


Figure 3. Signal transduction pathway hypothesized to play a role in the increased number of PKD cells

- (a) **Describe** the characteristics of the plasma membrane that prevent simple diffusion of Na⁺ and K⁺ across the membrane. **Explain** why ATP is required for the activity of the Na⁺ / K⁺ATPase.
- (b) **Identify** a dependent variable in the experiment represented in Figure 1. **Justify** the use of normal human kidney (NHK) cells as a control in the experiments. **Justify** the use of a range of ouabain concentrations in the experiment represented in Figure 1.
- (c) Based on the data shown in Figure 2, **describe** the relationship between the concentration of ouabain and the Na⁺ / K⁺ ATPase activity both in normal human kidney (NHK) cells AND in PKD cells. The scientists determined that Na⁺ / K⁺ATPase activity in PKD cells treated with 1 pM ouabain is 150 units of ATP hydrolyzed/sec. **Calculate** the expected Na⁺ / K⁺ATPase activity (units/sec) in PKD cells treated with 10⁶ pM ouabain.
- (d) In a third experiment, the scientists added an inhibitor of phosphorylated MEK (pMEK) to the PKD cells exposed to 10⁴ pM ouabain. Based on Figure 3, **predict** the change in the relative ratio of ERK to pERK in ouabain-treated PKD cells with the inhibitor compared with ouabain-treated PKD cells without the inhibitor. Provide reasoning to **justify** your prediction. Using the data in Figure 1 AND the signal transduction pathway represented in Figure 3, **explain** how the concentration of cyclin proteins may increase in PKD cells treated with 10⁴ pM ouabain.

Write your responses to this question only on the designated pages in the separate Free Response booklet.

8. 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 (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

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

STOP

END OF EXAM

8. Estrogens are small hydrophobic lipid hormones that promote cell division and the development of reproductive structures in mammals. Estrogens passively diffuse across the plasma membrane and bind to their receptor proteins in the cytoplasm of target cells.
- (a) **Describe** ONE characteristic of the plasma membrane that allows estrogens to passively cross the membrane.
- (b) In a laboratory experiment, a researcher generates antibodies that bind to purified estrogen receptors extracted from cells. The researcher uses the antibodies in an attempt to treat estrogen-dependent cancers but finds that the treatment is ineffective. **Explain** the ineffectiveness of the antibodies for treating estrogen-dependent cancers.

STOP

END OF EXAM