Where Strength Fails

Use an investigation to trace muscle weakness to a molecular step.

HS-AP4-3 "Conduct an investigation to analyze the molecular processes involved in sliding filament models to explain and identify changes in disease-related illnesses."

NAME _____ DATE _____

1 READ AND MARK

Read it twice

YOUR JOB Underline each step from nerve signal to muscle shortening. Circle the investigation results, then number the result that helps locate the weak link.

Testing a Weak Link

1. When you lift a backpack, millions of tiny protein interactions make the motion possible. A nerve signal causes a motor neuron to release acetylcholine at the neuromuscular junction. This chemical binds receptors on a muscle fiber and starts an electrical signal along the fiber. The signal triggers calcium release inside the fiber. Calcium exposes binding sites on actin, so myosin heads can attach. Using ATP, myosin heads pull actin, detach, and reset for another pull. This sliding shortens the fiber's working units, called sarcomeres. If a signal fails at any step, the fiber may produce less force.

2. To locate a weak link, a research team studied isolated nerve and muscle preparations under the same conditions. They measured muscle shortening after stimulating each nerve ten times. In untreated preparations, shortening stayed near 10 units from the first signal to the tenth. Next, the team added a chemical that blocks acetylcholine receptors without acting directly on actin or myosin. In those preparations, shortening fell from about 9 units on the first signal to 3 units on the tenth. A nerve signal still arrived, but the muscle fibers did not receive a strong enough signal to contract fully.

3. The team then stimulated the blocked muscle fibers directly with electrical pulses, bypassing the nerve and its junction. On repeated pulses, shortening stayed near 9 units. Direct stimulation still triggered electrical signals in the fibers, calcium release, and actin-myosin sliding. This comparison points to a problem before those internal steps, at the receptors, rather than a broken sliding mechanism. Myasthenia gravis is an illness in which antibodies reduce the number of working acetylcholine receptors. The blocker models one consequence of this illness, but the experiment alone cannot show that antibodies caused weakness in any patient.

2 ANSWER WITH EVIDENCE

Use the passage

1. Why did repeated nerve stimulation produce less shortening in the receptor-blocked preparations? Trace the effect from the receptors to actin and myosin.

2. Compare the untreated and receptor-blocked preparations after ten nerve signals. What does this comparison show about the blocker's effect?

3. What did direct stimulation show about the sliding mechanism in the blocked muscle fibers?

4. How does the blocker model myasthenia gravis, and what cause of weakness can this experiment not establish?

3 YOUR TURN

Your own words

A second preparation has working acetylcholine receptors but cannot release calcium inside its muscle fibers. In four sentences, predict its response to repeated nerve and direct stimulation, explain the actin-myosin result, and name one measurement that would distinguish this defect from receptor blockade.

Where Strength Fails

PART 1 · READ AND MARK

Underline each step from nerve signal to muscle shortening. Circle the investigation results, then number the result that helps locate the weak link. Accept any marking that follows this job and points at the right text.

PART 2 · ANSWERS

#	QUESTION	ANSWER
1	Why did repeated nerve stimulation produce less shortening in the receptor-blocked preparations? Trace the effect from the receptors to actin and myosin.	**Blocked receptors made muscle electrical signals less likely. That reduced calcium release, so fewer actin binding sites were exposed for myosin to pull.**
2	Compare the untreated and receptor-blocked preparations after ten nerve signals. What does this comparison show about the blocker's effect?	**Untreated shortening stayed near 10 units, while blocked shortening fell to 3 units. The blocker reduced shortening under repeated nerve stimulation.**
3	What did direct stimulation show about the sliding mechanism in the blocked muscle fibers?	**The blocked fibers still shortened near 9 units when stimulated directly, showing that their internal calcium response and actin-myosin sliding could still work.**
4	How does the blocker model myasthenia gravis, and what cause of weakness can this experiment not establish?	**Both reduce the effect of working acetylcholine receptors. The blocker experiment cannot establish that antibodies caused weakness in a patient.**

PART 3 · YOUR TURN (SAMPLE ANSWER)

Repeated nerve stimulation would produce little shortening even though the receptors respond. Direct stimulation would also produce little shortening because it bypasses the receptors but cannot restore calcium release. Without enough calcium, binding sites on actin stay covered, so myosin cannot pull actin even if ATP is available. Measuring calcium inside the fibers after direct stimulation would distinguish this defect from receptor blockade, which would still allow calcium release after direct stimulation.

Accept predictions of weak shortening with both kinds of stimulation if the explanation correctly links low calcium to reduced actin-myosin interaction. A suitable distinguishing measurement tests an internal muscle response after direct stimulation.