Motor Proteins and Striated Muscle Practice Problems:

1. Consider a vertebrate skeletal muscle cell with thick filaments that are about 2⌠m long and thin filaments that are about 1.75⌠m long.
a) What is the length of the A band and the I band in a muscle with a sarcomere length of 4⌠m? (1 mark)
b) What is the length of the H-zone of each A band at a sarcomere length of 4⌠m? At a sarcomere length of 3.5⌠m? (2 marks)
c) What is the length of overlap between the thin and thick filaments on each side of the H-zone at a sarcomere length of 4 ⌠m? (1 mark)
d) As the sarcomere contracts, decreasing in length, what happens to the length of the **I** band? Explain. (2 marks)

2. When biochemists isolate pure preparations of actin or myosin from a skeletal muscle, they first grind up the muscle in a homogenizer (a piece of equipment similar to a blender) to break up the fibrous connective tissue and cell membranes. They then place the resulting homogenate of ground up muscle into an ATP solution containing high levels of the compounds EGTA and EDTA, which act as Ca^{2+} chelators, effectively removing Ca^{2+} from solution. Only then will they attempt to purify either actin or myosin. Explain why ATP must be present and Ca^{2+} must be absent in the isolation solution in order to isolate actin separate from myosin. (4 marks)

3. You are studying myosin V, a class of myosin which can be used in cells to transport vesicles by "walking" along actin filaments, towards the plus ends of these filaments. You have identified a mutation in the gene for myosin V that affects its ATP binding site. This mutant myosin V (M) can still perform the entire cross-bridge cycle, but it takes much longer to hydrolyze ATP compared to a wild type myosin V (W).
a) Would the duration of the cross-bridge cycle be longer in M or W? Explain. (1 mark)
b) Would the duty cycle (the proportion of the cross-bridge cycle in which myosin is bound to actin) be longer in M or W? Explain. (2 marks)
c) How would this difference in duty cycle affect the ability of M to transport a vesicle along an actin filament? Explain. (2 marks)

4. A human genetic disease is caused by defects in the voltage-gated Na^+ channel that is expressed in skeletal muscle. These defective channels sometimes fail to inactivate after they are opened by a depolarizing graded potential.
a) How would this defect affect the changes in membrane potential observed in skeletal muscle during an action potential?
b) Why would this defect cause paralysis? Explain.
c) In some patients, the paralysis is most often observed following a period of prolonged exercise. In these patients, the channel fails to inactive only when extracellular K^+ levels are higher than normal (the channel inactivates properly when extracellular K^+ is normal). How might exercise result in high concentrations of extracellular K^+?