My artwork and essay relate to several course objectives to varying levels of depth. “Describe how a muscle contraction in induced,” and “Describe the 4 basic functions and 4 characteristics of muscle tissue.” Although I will not necessarily go into the stimulation of a muscle contraction, my entire piece is hinged on the idea that the induction of contraction is possible and has properly occurred, allowing the process I describe. More specifically, my work describes the functional unit itself along with its characteristics, rather than a muscle’s basic function, which is assumed here to be contraction. To fully appreciate the artwork and essay, I think both these objectives need to be taken into account.

The purpose of my piece is to represent similarities between myofilament interaction and canoe rower action, but also how certain elements of rowing can affect vessel motion as mutations affect proper myofilament interaction. This piece portrays two canoes or vessels filled with competition rowers. Each canoe acts as a myosin filament with every oar (including the paddle) representing myosin heads. The interaction of paddle and water parallel actin and myosin and their ability to contract. The winning canoe has several elements that enhance the rower’s abilities, allowing it to pull into the lead, placing it above the other. These elements represent beneficial or deleterious mutations, enhancing production of one, or decreasing ability of the other.

Although this is focused on a microscopic portion of the muscular system, it is crucial for proper function and contractile properties of the sarcomere. Because of this, proper function is critical to maintaining our everyday activities. Alterations in the structure of the myofilament components have been seen to modify them, either enhancing or diminishing their function.

There are four main muscular proteins that make up the sarcomere: actin, myosin, troponin, and tropomyosin. Actin is the protein that makes up most of the thin myofilaments. Tropomyosin is a regulatory protein covering binding sites preventing actin-myosin bonding. Troponin is a regulatory protein working with tropomyosin that binds to actin as well as calcium. Myosin is the protein making up the thick cylindrical myofilament including the many heads that extend out from the strand. Thin filaments are the strands of actin combined with the troponin-tropomyosin complex.

Each myosin head can only pull a very small distance before it reaches its maximum limit and must be re-cocked so it can continue pulling. This process is only permissible by utilizing ATP as an energy source. Contraction is composed of an extensive cyclic process of a “pull-detach-recock” mechanism. This is known as the cross-bridge cycle. This is the key to my art for this project. The action of rower’s paddles are comparable to the motion performed by myosin heads. The paddles are pulled through the water, lifted above the surface, repositioned for the next pull, and immersed once again. Myosin heads have an analogous movement to the paddles as they perform the power stroke, detach, re-cock, and are ready to work once again. ATP supplies the energy needed for each cycle, and the action of the myosin heads. (Betts et al. 2017.) So, what is needed for the duration and intensity of the power stroke to be increased, therefore making the entire muscle more efficient?

There are several factors that may influence the duration of the interaction, but they are not as well studied. Certain residues located within the central tropomyosin, G126 and D137, destabilize the coil-like structure due to their non-canonical properties. However, their mutant canonical counterparts, (G126R & D137L,) when present, were found to increase tropomyosin stability as well as velocity of myofilaments throughout the interaction (Shchepkin et al. 2017). However, these stabilizing mutations effect on actin-myosin force production is unknown. An additional question is if the mutations modified only single actin-myosin interaction or the cooperation of the entire mechanism.

The actin-myosin complex exhibits a longer interaction duration while under an increased stress load with this more stabilized form of tropomyosin, (mutation of both residues.) This correlates with an elevated contraction force. Not only this, but the more tropomyosin that is stabilized, or the more G126R and D137L that is present, the less myosin heads are needed to propel an actin filament, essentially making the muscle more efficient. Comparably, fewer stronger rowers with proper paddles can propel the same canoe faster than many weaker rowers with unsatisfactory paddles. It appears to be the size of the cooperative unit that is affected by these beneficial mutations as opposed to single actin-myosin interactions.

However, some destabilization produces flexibility which allows proper interaction between the thick and thin filaments. This is a major aspect of the cooperative activation of the thin filament by myosin. Similarly, it is important to have a light, flexible canoe that cuts through the water rather than a heavy one pushing through, being inefficient with its rowers’ power. Certain flexibility is provided by the highly conserved Asp-137 found to introduce negative charges on tropomyosin chains, (Sumida et. al, 2008.) So, although highly stable tropomyosin appears efficient, flexible properties are needed for successful contraction to occur.

These mutations appear rather beneficial in this case, as they seem to only enhance muscular function. The longer duration of the interaction logically correlates with a stronger contraction; the more force you can generate per single movement likely leads to a more efficient mechanism. This is analogous to my power-rowing sketch; the greater the stroke of a paddle, the more force is generated in a set amount of time.

This raises the question whether known tropomyosin mutations can cause elongated or reduced interaction duration. The following mutations are all located on the TPM1 gene which is involved in cardiomyopathies, so these mutations are an interest in research. A certain D175N mutation reduced the duration, while an E180G mutation increased it. E40K and E54K, both dilated mutations, result in significantly shorter durations. All these durations are relative to wildtype unmutated tropomyosin, and the duration of actin-myosin interaction that occurs there, (Kopylova et al. 2019.)

It appears that longer interactions generally result in an elevated rate of myofilament movement. This parallels to my power rower artwork since a longer stroke result in the boats moving faster and farther. Therefore, the rowers take as big a “bite” as possible with their paddles as they enter the water, resulting in a larger powerstroke, propelling the boat forward with an elevated force and rate.

In my artwork there are two canoes; the winning one has long oars and uniform paddles shaped to efficiently scoop water quickly, allowing a fast mode of travel. The canoe is slender, cutting through the water with an appropriate amount of flexibility. This portrays the more efficient paddling of the rowers, extended duration of paddles in the water, and the lightweight properties of their craft. The second canoe has smaller oars, and paddles of odd-shape, representing the more inefficient way of propelling the canoe forward. Their vessel also is more “block-like” with a flat nose, being much harder to propel through the water.

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