

muscle physiology lecture 37

cellular mechanisms of fatigue

if you have low ATP levels, then your ATPases can be affected, although overall dysfunction from depressed ATP stores may not be that severe. however, myosin head needs to be activated by ATP hydrolysis (ATPase) before binding to actin.

the sodium-potassium ATPase activity in the sarcolemma can also be affected because ATP is required to pump sodium and potassium against their gradients. recovery of the cell can be affected. fatigue is a state of performance alteration, which is a matter of regulation, which includes ion behaviors and locations. the resting membrane potential of the cell can also be affected. APB builds up as more ATP is hydrolyzed. calcium kinetics are affected and the sarcoplasmic reticulum can become leaky so their calcium pump function becomes compromised - this might limit the magnitude of the calcium dump on subsequent contractions. some of the ADP-related fatigue may be because of the inhibition of the RyR function in combination with reduced clearance of calcium from the myoplasm.

if ADP is competing with ATP for the binding sites, that will slow down the speed at which calcium could be released. lower ATP:ADP is worse.

at rest, blood has a pH of about 7.4 and muscle is about 7.0, during exercise, blood has a pH of about 7.1 and muscle is about 6.8. the protons detected by nociceptors contributes to that burning sensation and that might contribute to fatigue as well (not as strongly as once was believed)

lactate is good because it does not contribute to acidosis, it actually helps to delay the build up of protons. under regular conditions, calcium kinetics (as calcium leaves sarcoplasmic reticulum, H^+ ions go in to help maintain the charge and when it is time for the muscle to stop contracting, the sarcoplasmic reticulum Ca-ATPases pump the Ca^{2+} back into the reticulum while pumping H^+ ions out) works perfectly fine, however, intense exercise is not a normal condition.