

muscle physiology lecture 30

anabolic cellular signaling

things that turn on mTOR and induce hypertrophy - immune/chemicals, mechanical tension, endocrine system, nutrition. injury and destruction of tissue and how that is regenerated, load profiles, application of loads and what does that do inside of the cell. muscle metabolism in relation to nutrition.

in a phospholipid bilayer, about 10-20% of it in skeletal muscle is arachidonic acid (20 carbon fatty acid polyunsaturated) pretty abundant in skeletal muscle. when you go exercise, you will release a lot of arachidonic acid via phospholipase A₂. arachidonic acid is a substrate for COX so you synthesize a lot of prostaglandins. they signal the MEK-ERK (MAPK) pathway. then, MEK-ERK and mTOR "cross-talk", causing you to grow. sir doctor john robert vane won a nobel prize in physiology or medicine in 1982 for discovering how aspirin suppressed the production of prostaglandins. ibuprofen treatment blunts early transitional signaling responses in human skeletal muscle following resistance exercise. we are still learning about how prostaglandins become an anabolic signaler. MEK - mitogen-activated protein kinase kinase (yes, 2) the phosphorylation cascade relay race from the outside of the cell, to the cell surface, to the inside of the cell. raptor is a hub for cross-talk. in the presence of tissue damage, the body responds with immune and inflammatory cells (neutrophils, macrophages, cytokines) interleukin-15 (cytokine) is released in abundance during tissue damage that is proportional to the amount of damage. appears to promote protein synthesis and inhibit protein degradation. interferon γ - cytokine that interferes with viral replication. endogenous interferon γ is required for efficient skeletal muscle regeneration.

myostatin - myokine (cytokine in muscle) that's mostly autocrine, member of the TGF- β subfamily. inflammatory mediators and pro-inflammatory cytokines physically interact with and inactivates TSC1, which leads to mTORC1 activation.