

muscle physiology lecture 33

hypertrophic signaling - applications

general health - enhancements of longevity, lifespan, healthspan, improve cardiovascular health. an elevated mTOR activity has a relationship with enlargement of the heart, treating with rapamycin combats this because it may be a tool to improve cardiac function and repair cardiac hypertrophy. AMPK and insulin regulate glucose differently. angiogenesis - early restoration of blood, access to nutrients in diabetic patients. AMPK - terminal healing is compromised but maybe early healing is enhanced. depends on the injury. also, depending on the nature and location of the injury, you might induce apoptosis. APS - eccentric stress sounds the anabolic alarm - eccentric loading. more disruption to sarcolemma where arachidonic acid is located, this leads to more PLA2 activity, followed by more COX activity, followed by a greater buildup of prostaglandins, followed by more MAPK signaling and MAPK cross-talks. interleukin-15 increases myosin accretion in human skeletal myogenic cultures. mTOR may be sensitive to intracellular calcium levels. introducing SAC inhibitors while loading the muscle attenuates PKB activation and eccentric loading seems to elicit a greater effect. exercise induced muscle damage is a major trigger for the increase in MGF levels and eccentric stress seems to be a more potent trigger. eccentric activation of the tissue is associated with a greater reduction in myostatin, a myokine, with autocrine, paracrine, and endocrine functions. (PKB inhibition) speed of contraction matters too as slow eccentric stress may generate more MGF than fast eccentric stress size principle also matters a lot. mechanotransduction belongs to the fibers that were activated. passive fibers do not really participate in the sensation of and response to the application of mechanical loads. workloads that involve a low load and high duration do not activate PKB-mTOR-P70S6K, while workloads that involve a high load and a short duration does.