

## muscle physiology lecture 28

### anabolic cellular signaling

muscle metabolism - muscles constantly changing in size, grow and shrink to meet demands of the environment. based on protein turnover. - the balance of synthesis and degradation. hypertrophy - synthesis outpaces degradation. not just actin and myosin - structural stuff that accounts for hypertrophy.

the reason why we adapt to exercise because it threatens us, so the body reevaluates itself and rebuilds its structures to become stronger. high stresses will induce adaptation. hypertrophy is an adaptation that reinforces skeletal muscle - fitness, self-preservation, and survival.

Compatibility with your environment. how does resistance training cause hypertrophy? - mTOR (mostly complex 1) reception, transduction, response.

insulin's activation of PDE - insulin binds to its receptor, PI3K gets activated, PKB gets activated, PKB phosphorylates PDE. PDE converts cAMP to AMP, so PKA does not get activated, so HSL and perilipin do not get phosphorylated, and lipolysis does not happen. Akt/PKB is super busy.

major domains - proliferation & growth go together (get enough territory before dividing it) the loss of PKB signaling contributes to diabetes while the gain of function of PKB drives cancer.

why are signaling cascades important? - we need information to be relayed from the outside environment to the inside of our cells.

the finish line of a signaling cascade is typically in the nucleus,

regulating genetic expression. transcription - in nucleus, cell machinery

copies the gene sequence into messenger RNA (mRNA) translation -

ribosome reads the mRNA sequence and translates it into the

amino acid sequence of the protein. "stop" codons (UAA, UAG, UGA)

tell the ribosome when the protein is completed.