

mTOR Part 6 of 7

mTOR is turned on by:

- >Need
- >Nutrition
- >Assumption of nutrition

AMPK (Gray character) turns off atrophy by:

- Turns off mTOR and turns on protein degradation!
- >FOXO>Lysosomal system/Ubiquitin Proteasome System>Protein Degradation
- >UIK1>Lysosomal system>Protein Degradation
- Turn this off:
- >TSC1/2>Rheb>mTOR>P70S6K>rpS6>Translation
- >Raptor>p7-S6k/4E-BP1

AMPK balances Renin-Angio-Aldosterone: increases blood pressure and blood volume (fluid retention)

AMPK and insulin both regulate glucose intake (but differently)

- >ATP goes through hydrolysis, binds to gamma unit on AMPK (stops glycogen synthase), which activates GLUT4/LKB1
- >Unlike insulin, AMPK needs exercise

Eccentric Loading

- >More disruption to sarcolemma (Arachidonic acid), increased PLA2, COX activity, meaning more prostaglandins and MAPK signaling
- >Increased PKB activation
- >Triggering ILGF (Mechano-growth factor)
- >Reduces myostatin (inhibits PKB)
- >Increased Integrin signaling
- Slow eccentric stress may generate more MGF than fast eccentric stress
- Size principle (high load)
- although: low load, blood flow restriction, GH and MAPK

Aerobic + Anaerobic

- >Aerobic:
 - ATP> ATPase>ADP>Adenylate Kinase> AMP (binds to gamma on AMPK and LKB1 binds to alpha unit)> AMPK (inhibits raptor) (activates TSC2)> FOXO/UIK1>Lysosomal system> Protein degradation
 - Promotes mitochondria biogenesis (not mTOR)
- >Anaerobic:
 - mTOR and p70s6k activation... Hypertrophy (inhibits TSC1/2)
- >Concurrent training:
 - Power (RFD) is generated from strength training only

Specificity of Adaptation

- >Applying uniaxial loads activates mTOR signaling different than multi axial loads
- >Alfredson protocol (requires mechanical loading)

Nutrition:

- >Carbs and AMPK
 - Keto; AMPK inhibits mTOR
 - Glycogen loading inhibits AMPK

>Protein

-Leucine, lysine and arginine (90% activation mTOR)