



2020 NSCA

NATIONAL CONFERENCE

#NSCACon20

CONFLICT OF INTEREST STATEMENT

I have no actual or potential conflict of interest in relation to this presentation.

Owner of The BFR PROS – Unbiased BFR education

ABOUT ME

Professional Credentials

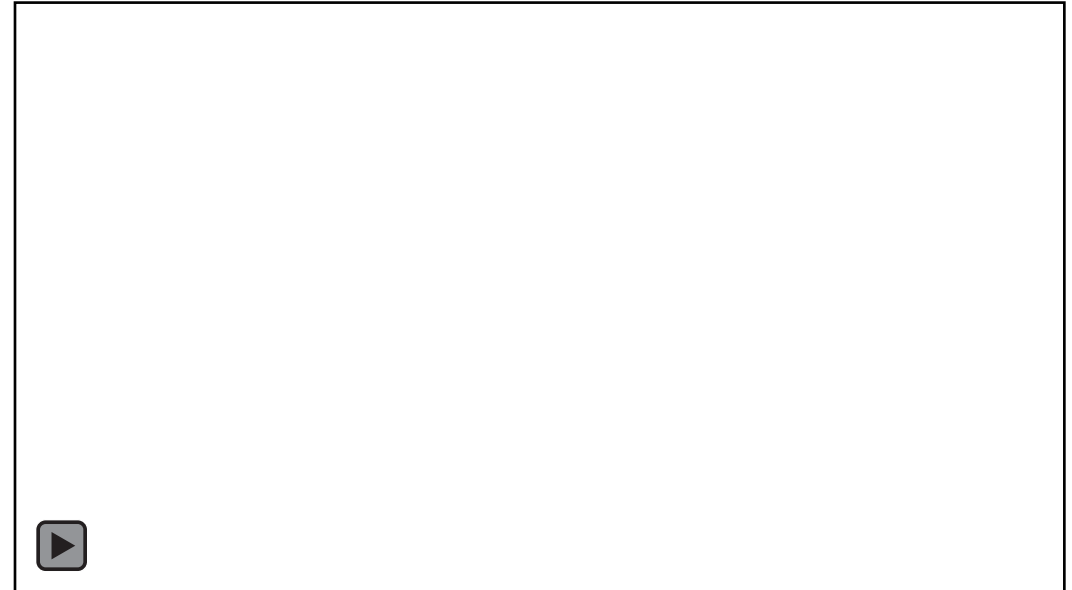
- Doctor of Physical Therapy
- MS Health Promotion Management
- Certified Strength & Conditioning Specialist
- The Human Performance Mechanic
- Co-Founder of The BFR PROS – www.bfrtraining.com
- Published BFR Author
- @thehpm / @thebfrpros on IG

Personal



OVERVIEW

- What is Blood Flow Restriction Training?
- The Fundamentals (Muscle Fiber and Fiber Types & Motor Units)
- LL-BFR > LL?
- Fatigue
 - Central vs. Peripheral
- How BFR Likely Works (As of 2020)?



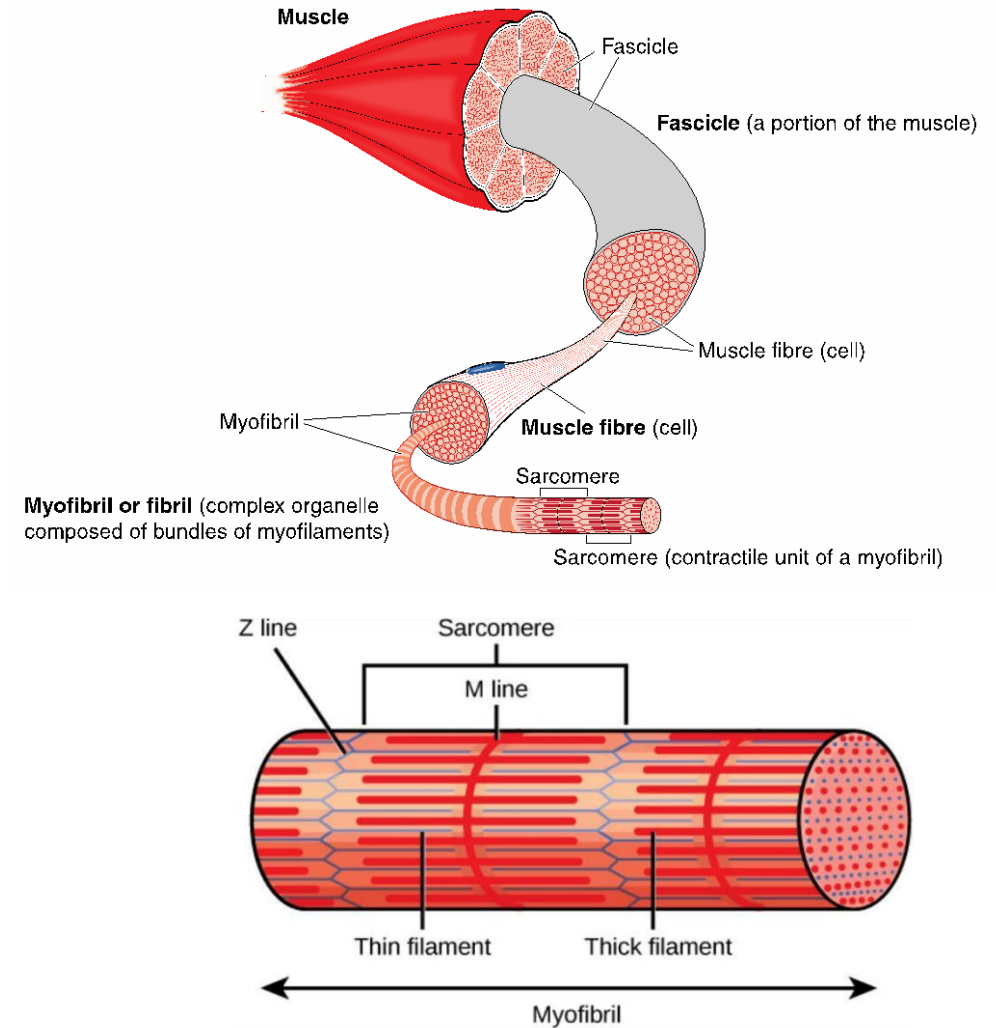
WHAT IS BFR TRAINING?



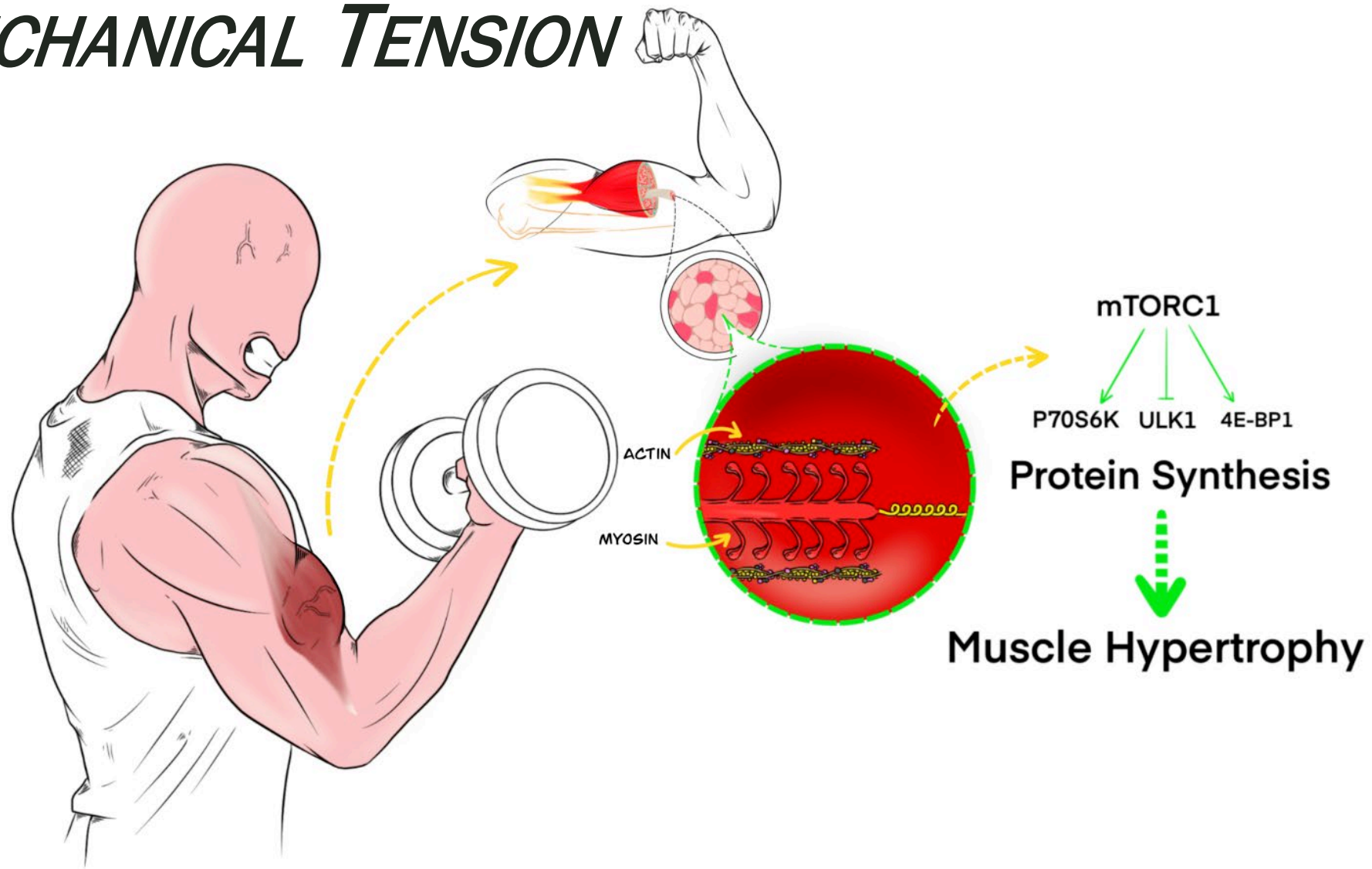
THE FUNDAMENTALS

Muscle Structure

- A muscle fiber is composed of many myofibrils that contain the sarcomeres
- Sarcomeres are the contractile unit of a myofibril
- Actin/Myosin = creation of mechanical tension → force
- Mechanical tension = **growth**



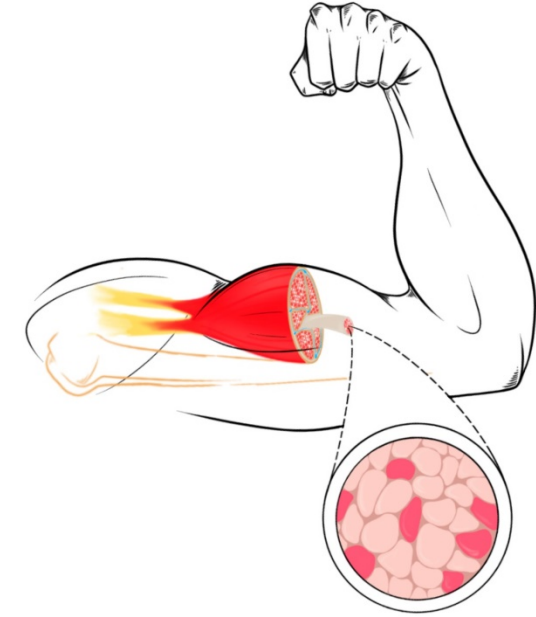
MECHANICAL TENSION



THE FUNDAMENTALS

Muscle Fiber Classification

- Muscle fibers can be broken down into different types based on their physiological characteristics
- Broadly Type I and Type II
- Type II associated with greater absolute growth potential and muscle force generation capabilities
- Type II fibers = primary targets for muscle growth (hypertrophy) and strength

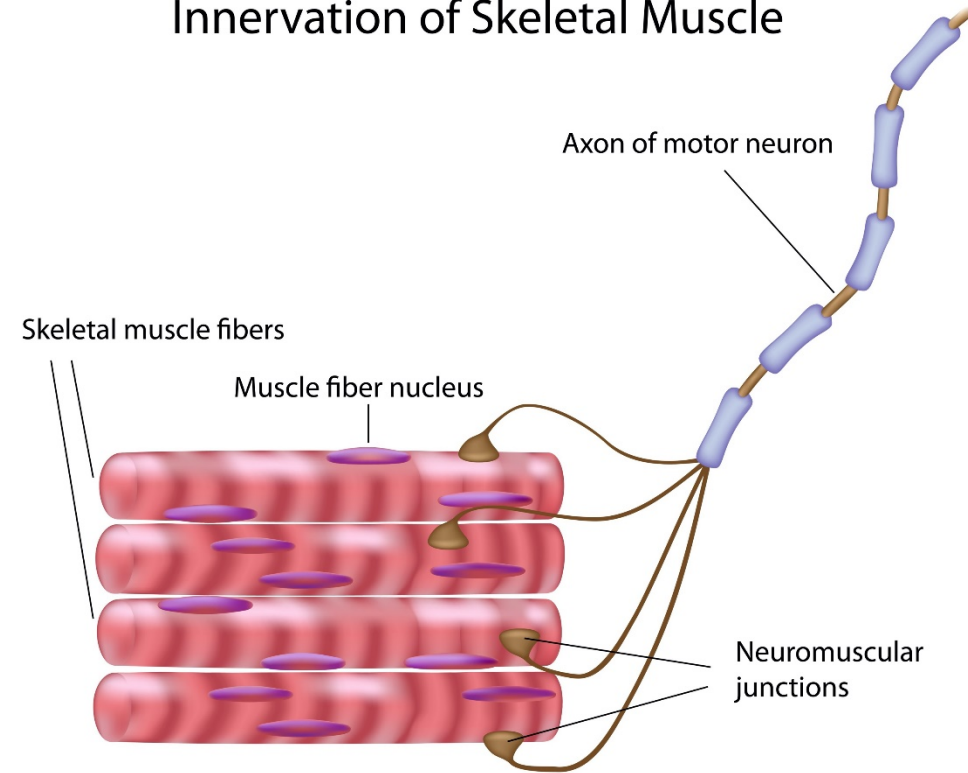


THE FUNDAMENTALS

Motor Unit

- Motor neuron and all its associated muscle fibers it innervates
- Motor units can innervate a few- or hundreds of muscle fibers and is dependent on a number of physiological factors
- Motor units can be classified as low- or high-threshold
- Operate on Henneman's size principle and exhibit an exponential relationship with recruitment

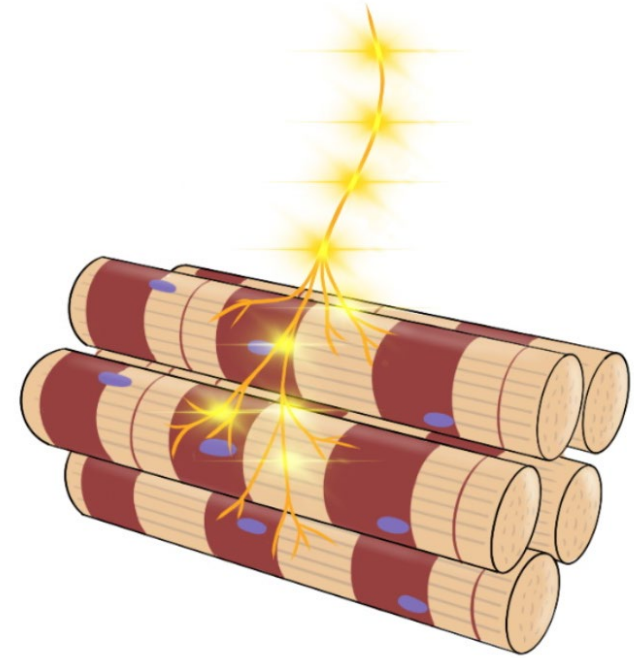
Innervation of Skeletal Muscle



THE FUNDAMENTALS

Low-Threshold Motor Units

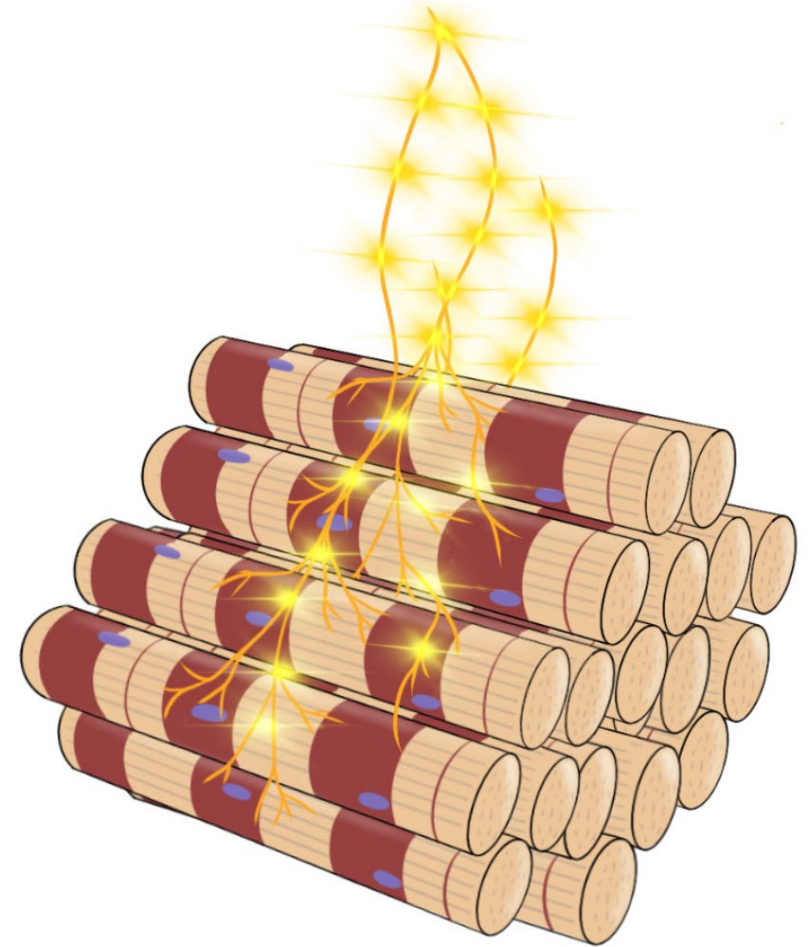
- Low-threshold motor units mainly composed of type I fibers
- Oxidative -> O_2 required
- Recruited for non-fatiguing, low-load tasks
- Minimal hypertrophic/strength potential when recruited due to low total number of fibers associated with activation + they're normally recruited during everyday tasks (low-effort)
 - Still undergo high levels of mechanical tension everyday due to ADLs



THE FUNDAMENTALS

High-Threshold Motor Units

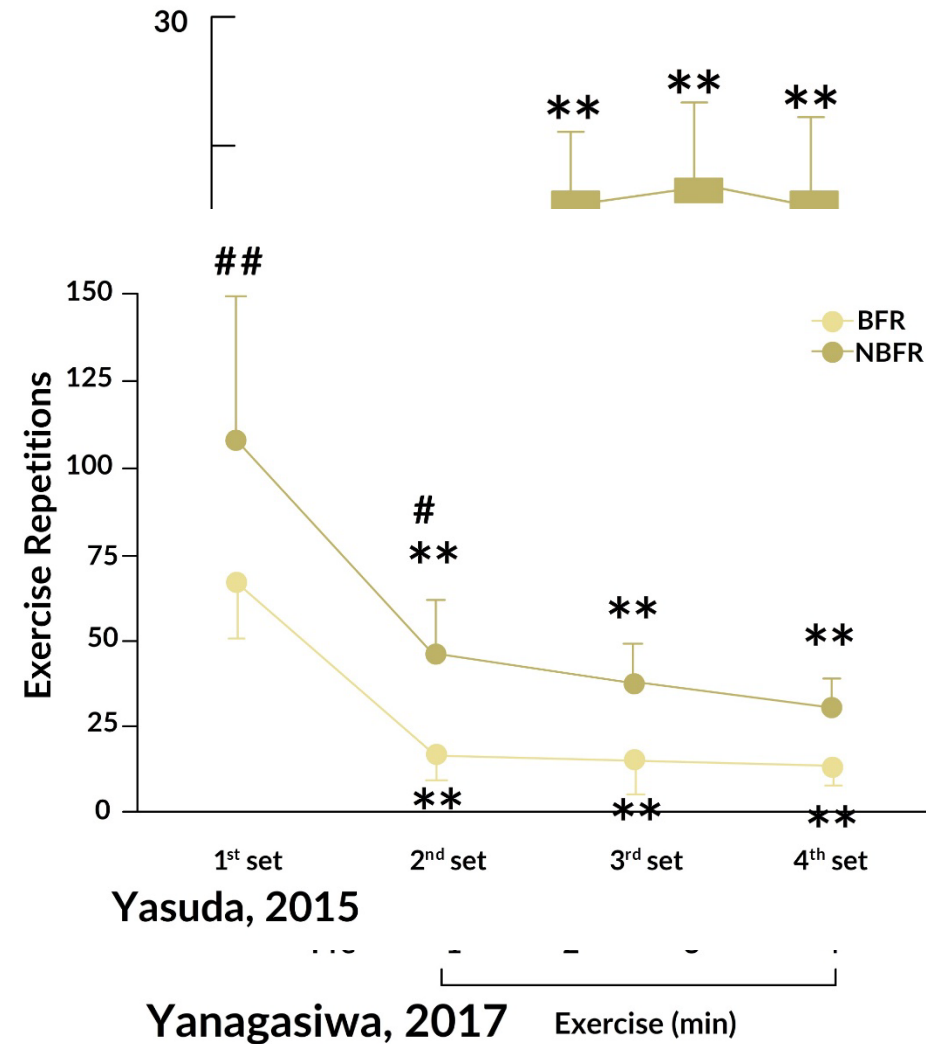
- High-threshold motor units contain significantly more type II fibers (although high amounts of type I fibers still present)
- Exponential recruitment as a result of motor unit properties
- Recruited for high effort and/or fatiguing contractions -> + muscle activation levels
- Anaerobic/glycolytic -> La/H⁺/Pi
- Significant muscle hypertrophy/strength growth potential when recruited consistently x weeks



BFR AND LOW-LOAD RESISTANCE TRAINING

Some Consistent Observations From The Research

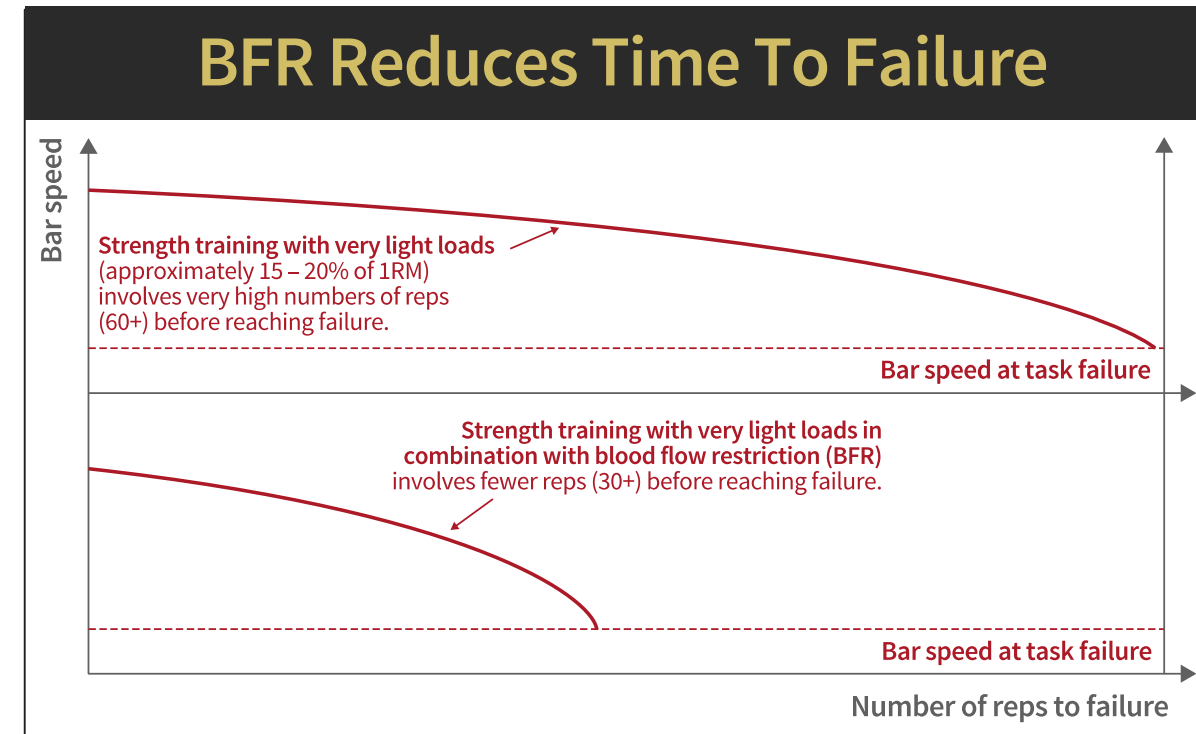
- Higher Perceived Exertion compared to no-BFR LL conditions (Yasuda, 2013)
- Greater muscle deoxygenation over LL exercise (Yanagasiwa, 2017)
- Higher Levels of muscle activation over LL exercise (Yasuda, 2014)
- Greater heart rate & blood pressure response compared to no-BFR conditions (May, 2018)
- Significantly reduced repetitions required to reach volitional failure during exercise (Yasuda, 2015; Buckner, 2019 and MANY more...)



BFR AND LOW-LOAD RESISTANCE TRAINING

...Yet similar muscle hypertrophy when LL exercise is taken to volitional fatigue in a chronic RT protocol (Fahs, 2015; Farup, 2015; Gavanda, 2020)

****All of these factors suggest that BFR augments fatigue at any given repetition compared to LL training leading to similar long-term outcomes on muscle growth...****



Adapted from Chris Beardsley

So if we get similar amounts of hypertrophy long-term between conditions....

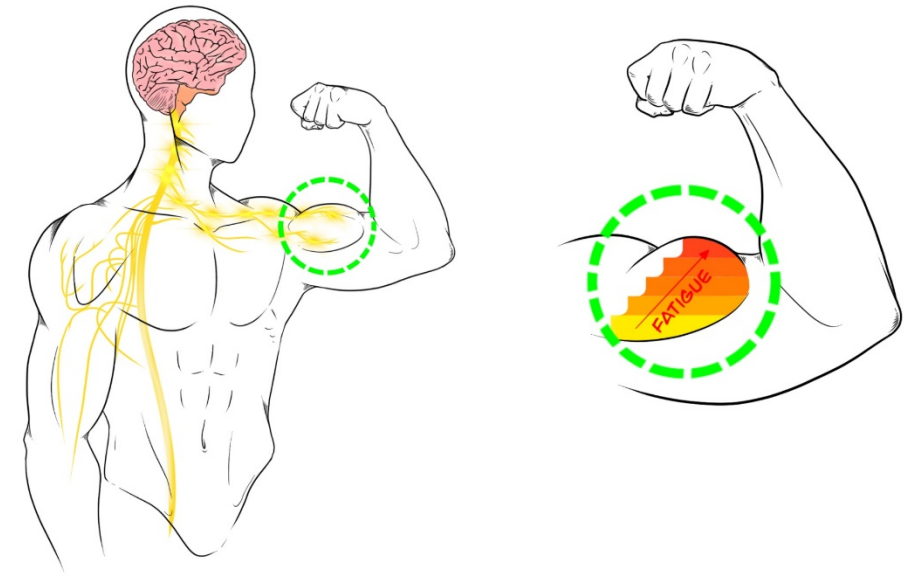
...what's the likelihood that there are completely separate mechanisms driving similar levels of growth?



FATIGUE

Definition

- Temporary and reversible loss in maximal muscle power or force that occurs during exercise and is reversible with rest (Gandevia, 2001; Enoka & Duchateau, 2008)
 - Immediately post-exercise -> Loss of MVC (Peyrard, 2019; Copithorne, 2019a)
 - Sustained for days (Alvarez, 2020)
- Broken down further into Central and Peripheral
 - Central – inhibits muscle activation
 - Peripheral – facilitates further recruitment of non-fatigued muscle fibers -> muscle activation

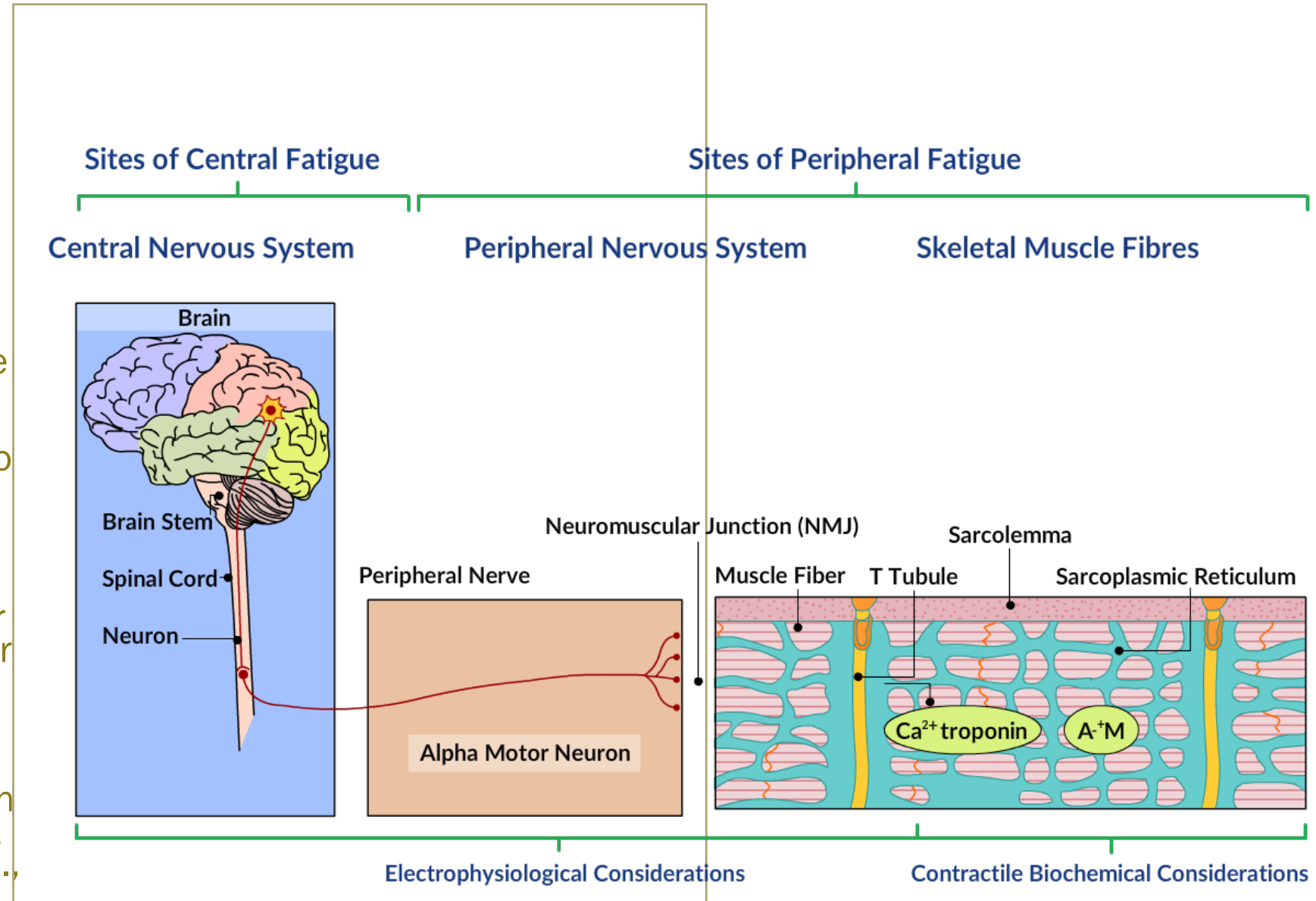


 **Pheasyque**

FATIGUE

Central

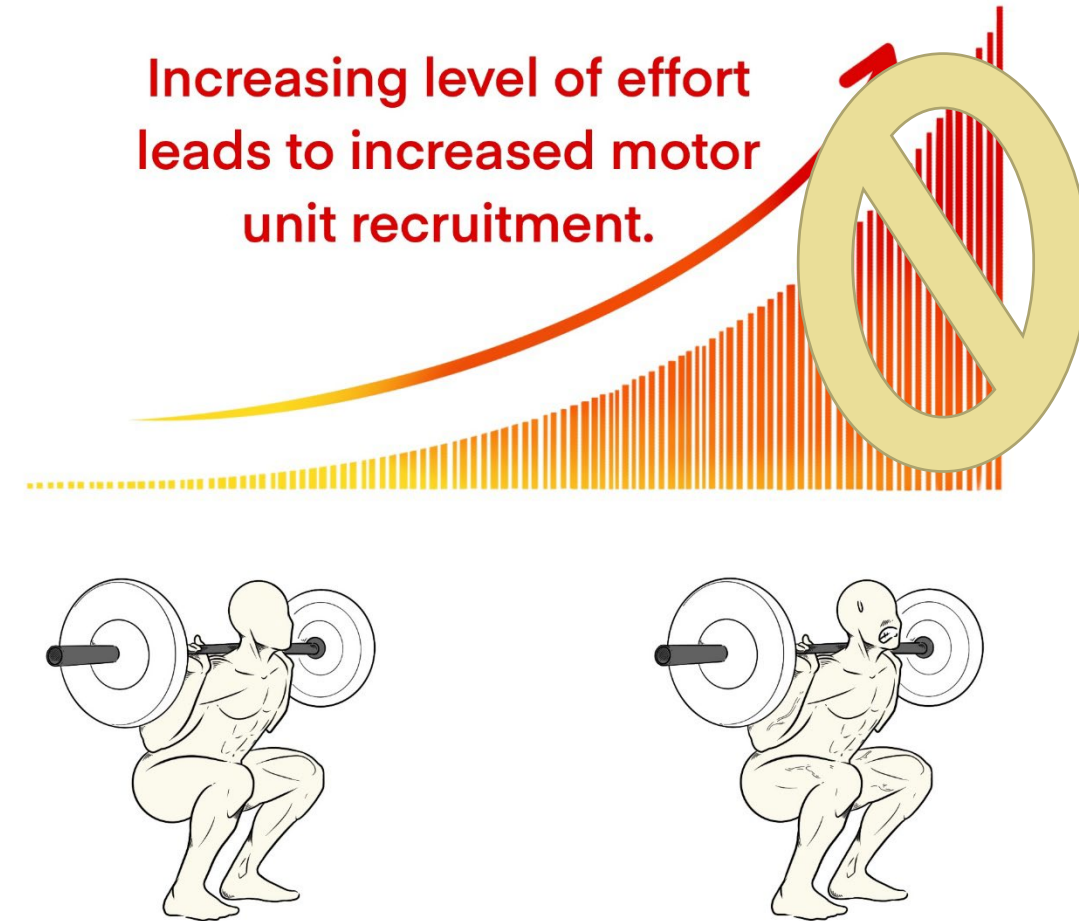
- Physiological processes proximal to the NMJ of the alpha motor neuron that ultimately result in a reduction of motor drive to the motor nerve (Amann, 2015)
 - AKA inhibitory!
- Occurs in the brain (motor cortex), spinal cord and/or the group III/IV afferents
- Mostly dissipates post-exercise although prolonged CNS fatigue can occur for days (Thomas et al., 2018; Fernandes et al., 2019)



FATIGUE

Central Fatigue Mechanism?

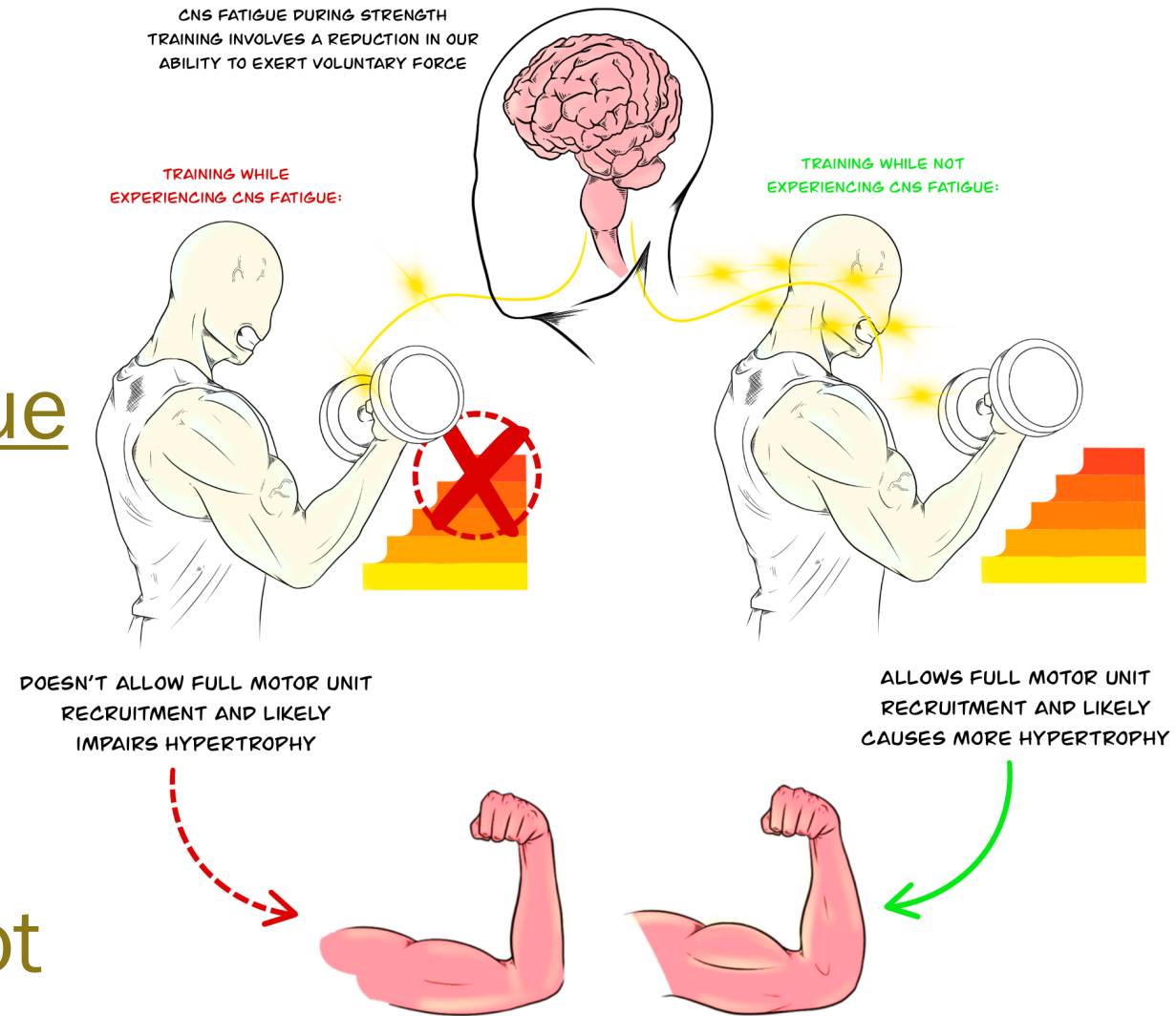
- Duration dependent? (Smith et al., 2007; Taylor & Gandevia, 2008; Thomas et al. 2015)
 - Neuroinflammatory Model of Fatigue (Vargas, 2014; MacDonald et al, 2003)
- Amount of neural drive dependent on effort and reductions in effort reduce motor drive (and outcomes) (Hollander, 2017; Jiang, 2017)



FATIGUE

End-Result of Central Fatigue

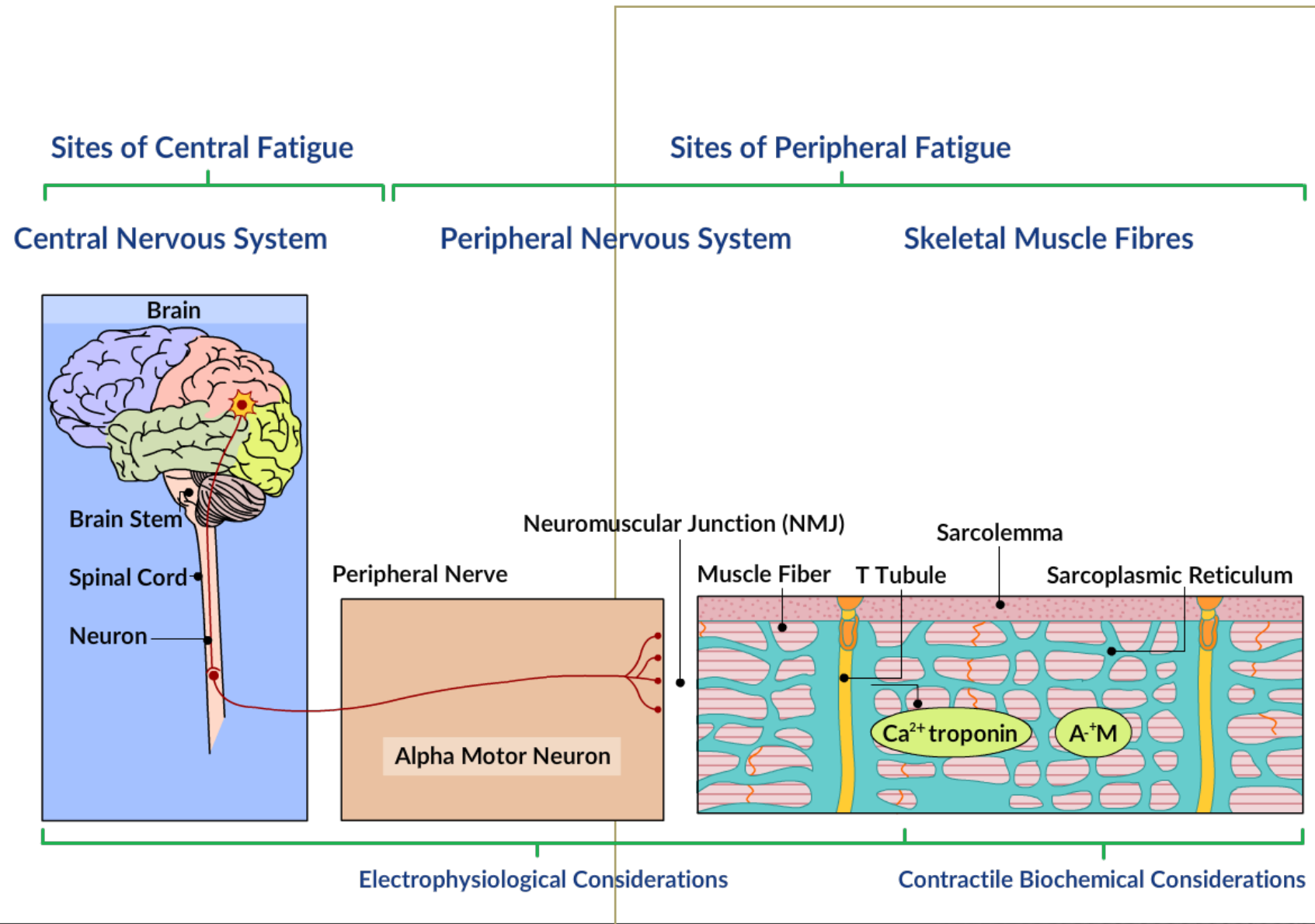
- Leads to reductions in overall motor unit recruitment (type II fibers) despite maximal intent
 - In a practical sense, “not recovered enough”



FATIGUE

Peripheral

- Physiological processes distal to the NMJ of the alpha motor neuron that ultimately result in a reduction of muscle fiber force and contraction velocity (Amann, 2015)
- Interferes with excitation-contraction coupling process and/or crossbridge cycle (Fitts, 2008; Allen & Trajanovska, 2012)
- Mostly dissipates post-exercise except for muscle damage (unaccustomed exercise) (Alvarez, 2020)

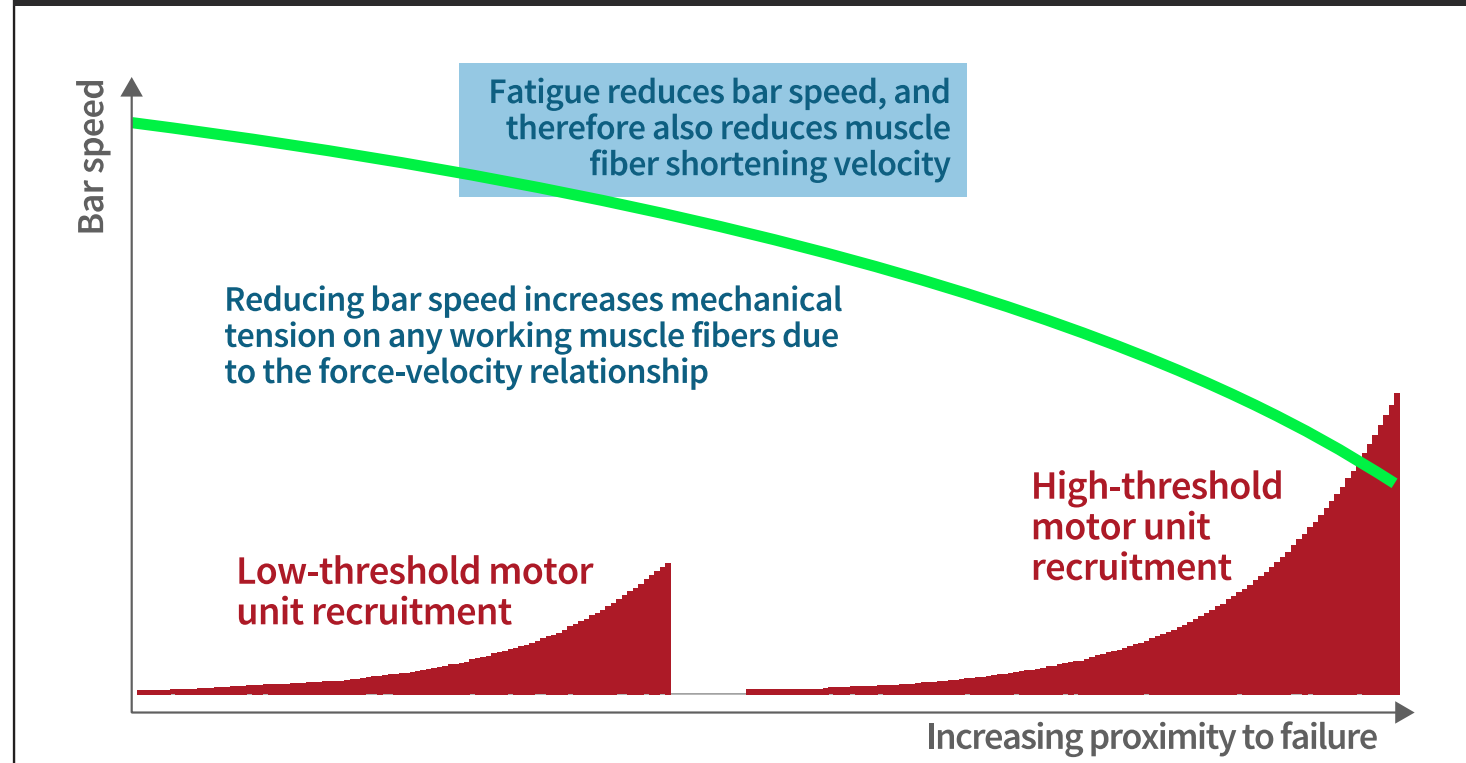


FATIGUE

Bar Speed and MU Recruitment

- Effort = critical
- Muscle fiber contraction velocity slows with peripheral fatigue (Sanchez-Medina, 2011)
- Increased neural drive needed to maintain force production
- High threshold MU recruitment results
- Hypertrophic stimulus occurs

Bar Speed, Failure & Motor Unit Recruitment

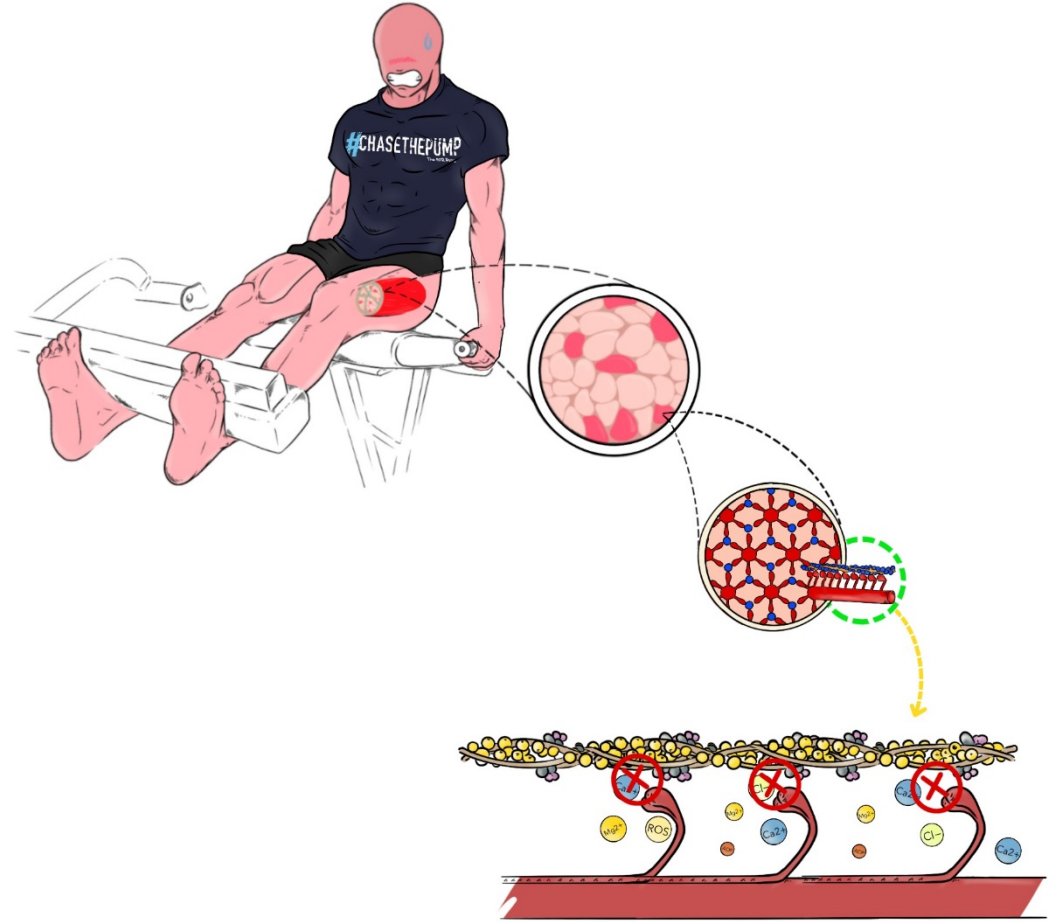


Adapted from Chris Beardsley

FATIGUE

Peripheral Fatigue Mechanisms

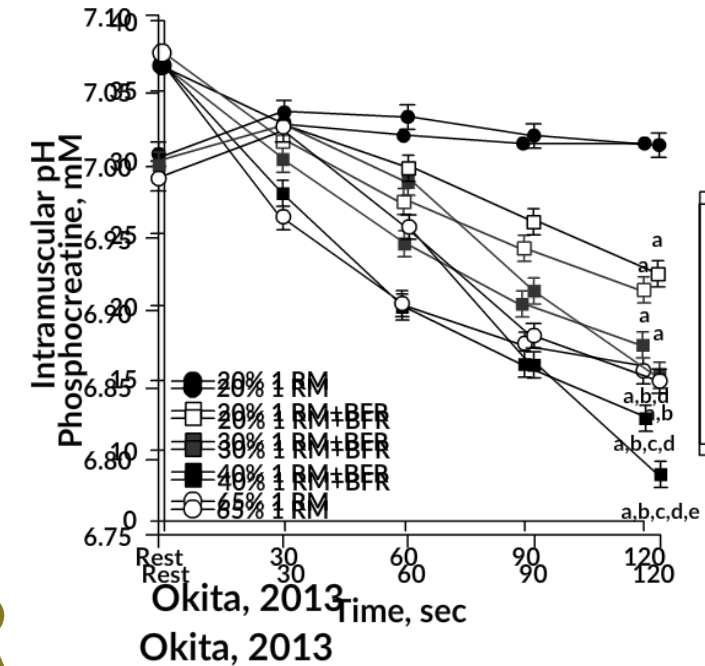
- Multiple processes can influence peripheral fatigue accumulation including (for our purposes) metabolic stress
- Pi^* , H^+ , Ca^{2+} , Lactate (Place, 2010)



FATIGUE

Peripheral Fatigue Mechanisms

- $\text{PCr} \rightarrow \text{Pi} + \text{Cr} + \text{ATP}$; +La and -pH
- Metabolic Stress > with higher loads (65%+ 1RM) OR adding BFR with lower loads (20-40% 1RM) (Okita, 2013)

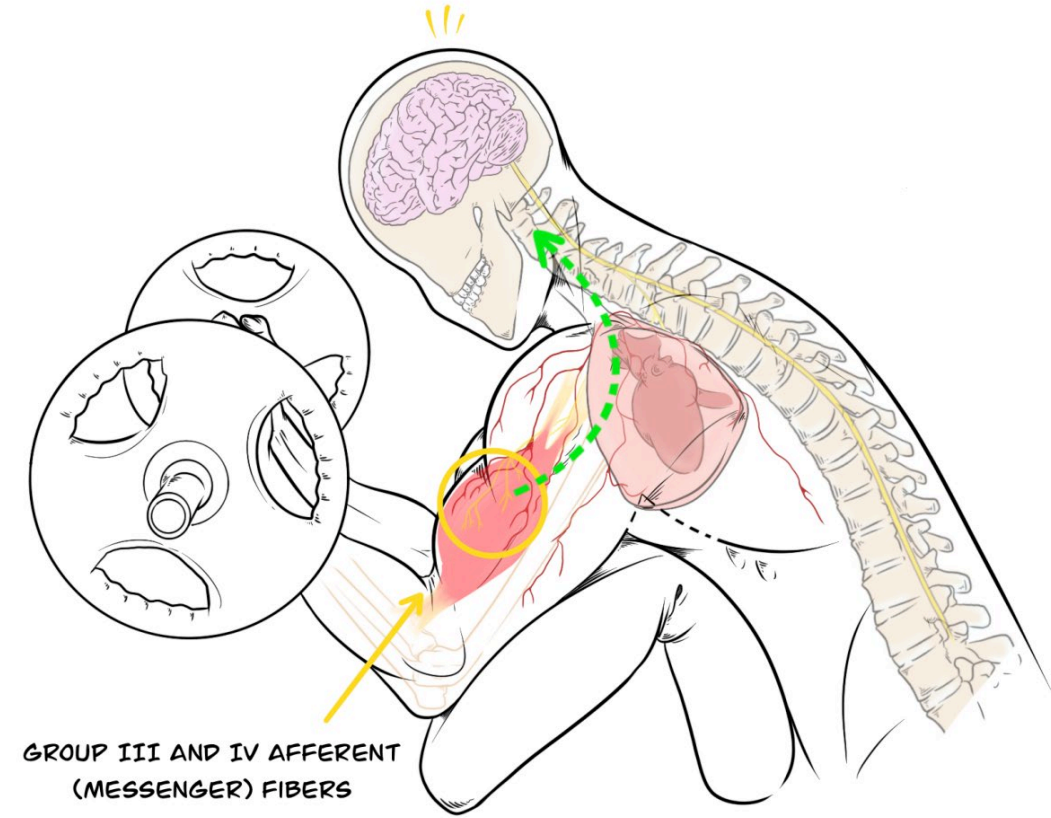


2×30
plantarflexion
repetitions @
various loads @
130% SBP BFR

FATIGUE

Group III-IV Afferents

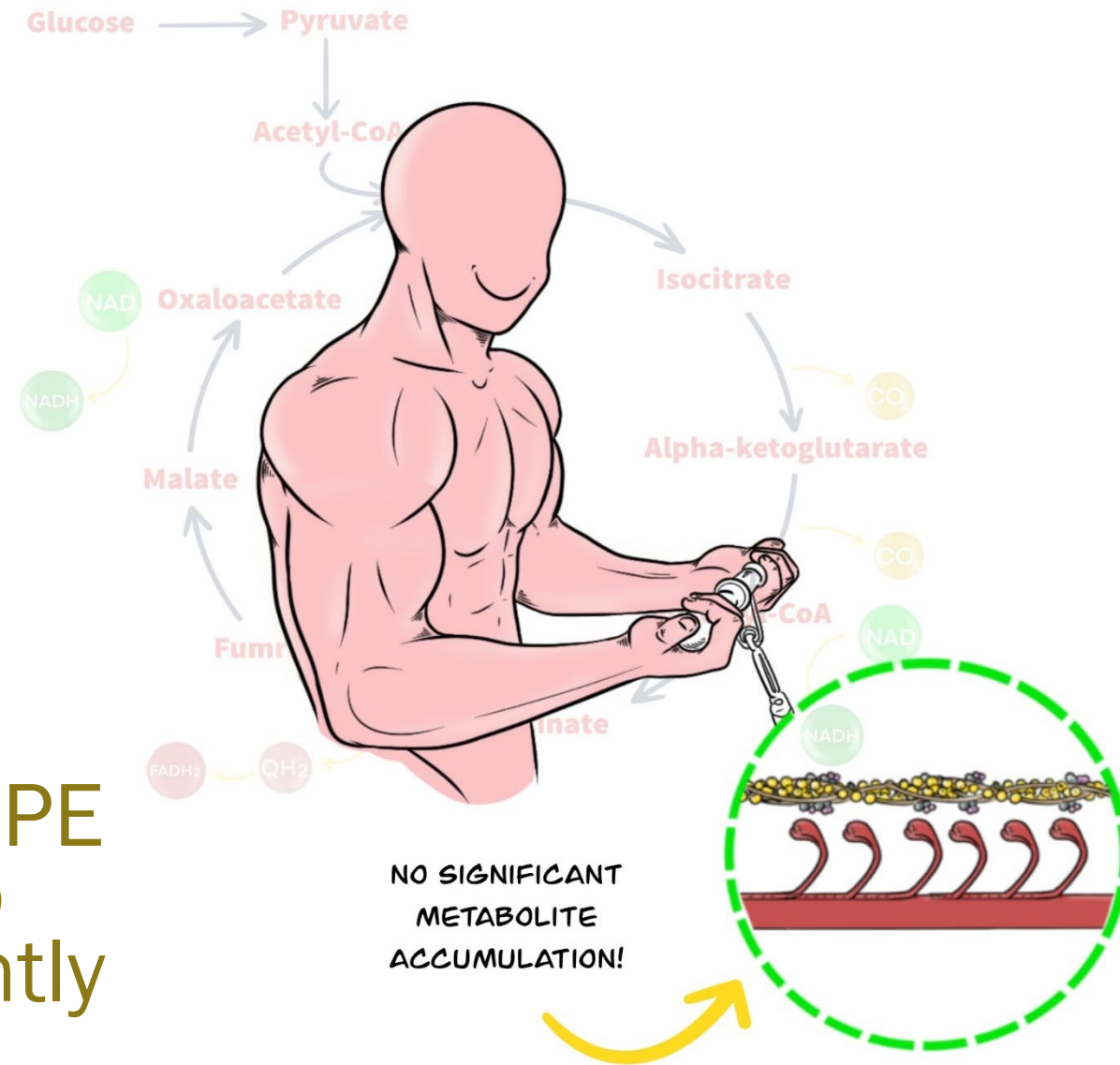
- Plays a role in managing both peripheral and central fatigue
- Signals increase in BP/HR/V in response to O₂ demands (Amann, 2015)
- Role theorized to support exercise performance but it comes at a cost (Pollak, 2014)
 - + Discomfort, Pain, Exertion



FATIGUE

Normal LL Exercise (Non-Failure)

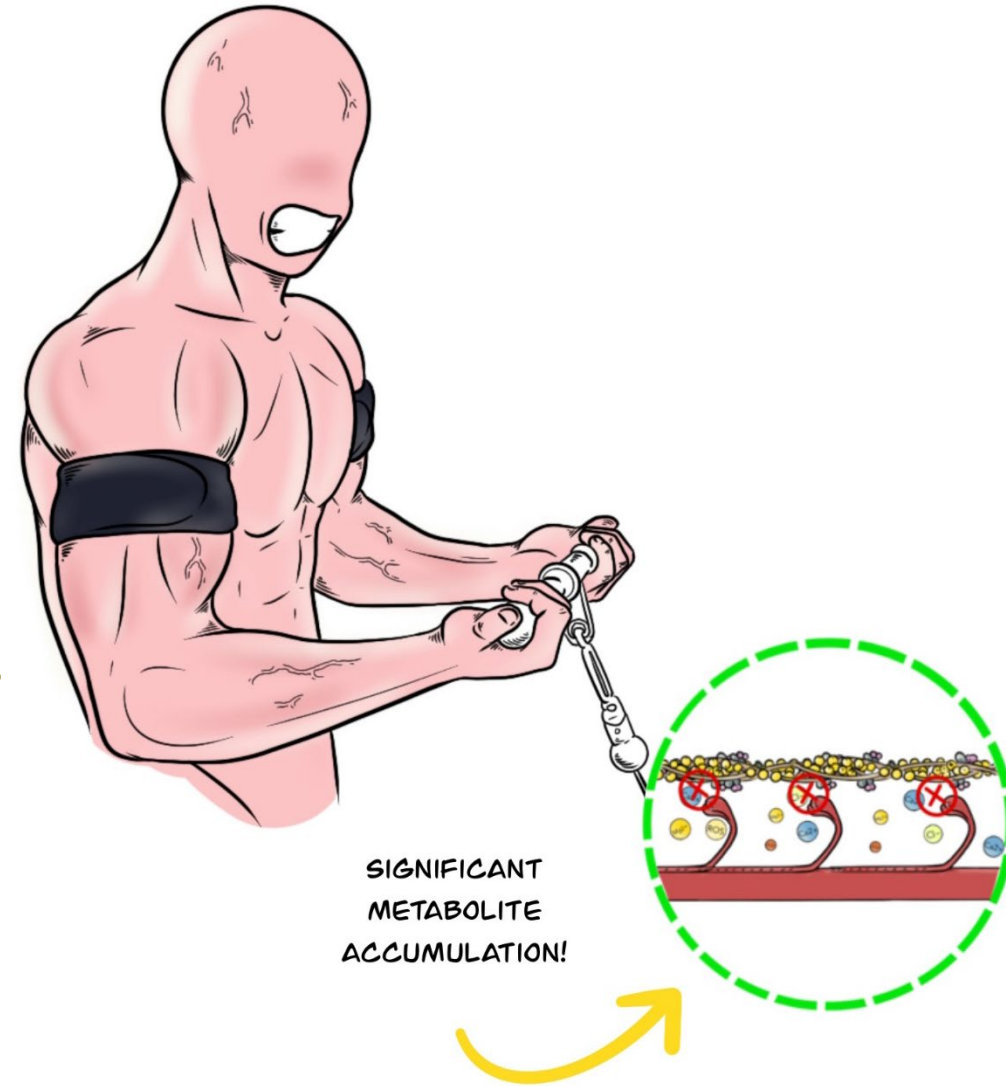
- O_2 levels are managed, afferent activity is attenuated
- Energy is resynthesized, RPE minimal, muscle is able to buffer metabolites efficiently and they clear via venous system during rest



FATIGUE

BFR Exercise

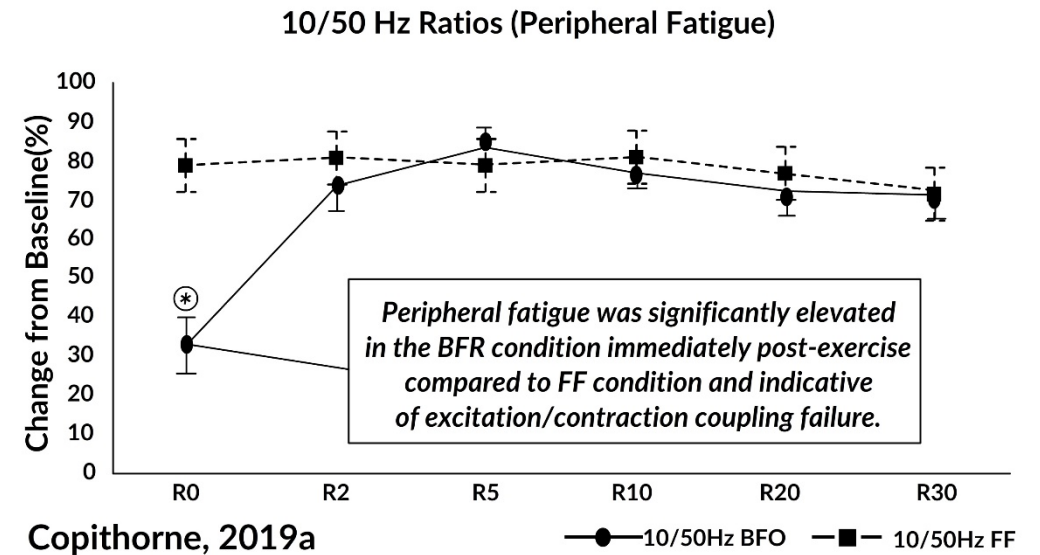
- O_2 levels are reduced (somewhat) (Willis, 2018)
- Energy imbalance occurs
 - Greater reliance on PCr and anaerobic glycolysis (Broxterman, 2017)
- Greater production of metabolites, stimulation of the afferents, perceptual demands (Yasuda, 2010)
- Unable to clear metabolites; exert effects on excitation/contraction and/or cross-bridge cycling at an accelerated rate (Peyrard, 2019; Copithorne, 2019a/b)



FATIGUE

Copithorne (2019)
(2019a)
(2019b) DB10 =

- Peripheral fatigue
- Central Fatigue similar at
- Voluntary contraction recovered BFR at failure
- quicker in BFR
- Recovered quickly to FF
- Voluntary contraction?
- EC failure

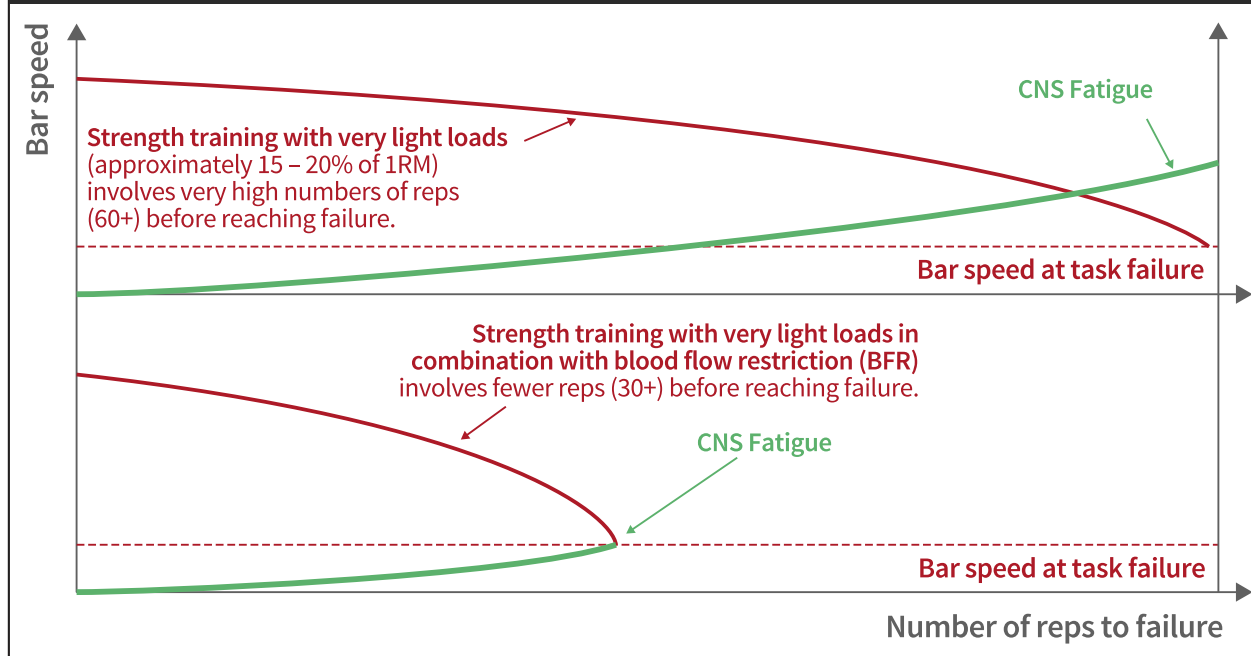


PERIPHERAL FATIGUE ACCELERATION

Potential Mechanisms

- Greater excitation/contraction disruption (Peyrard, 2019; Copithorne, 2019a/b)
- Similar/Slightly less CNS fatigue at failure compared to LL (Copithorne, 2019a; Peyrard, 2019)?
 - Load dependent?

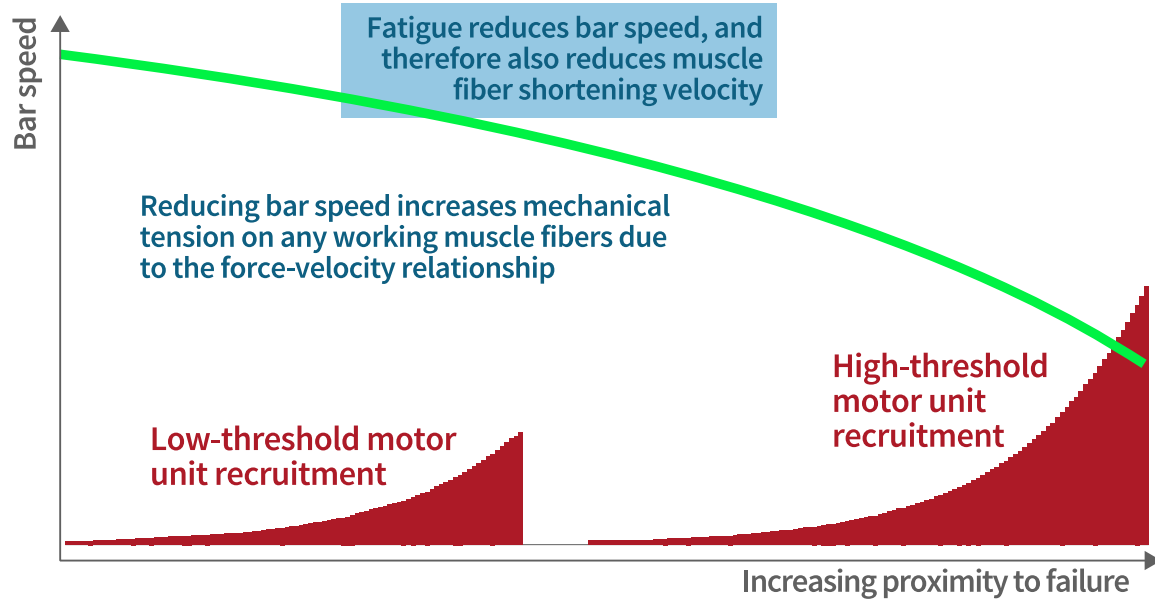
BFR Reduces Time To Failure



Adapted from Chris Beardsley

TAKING IT HOME!

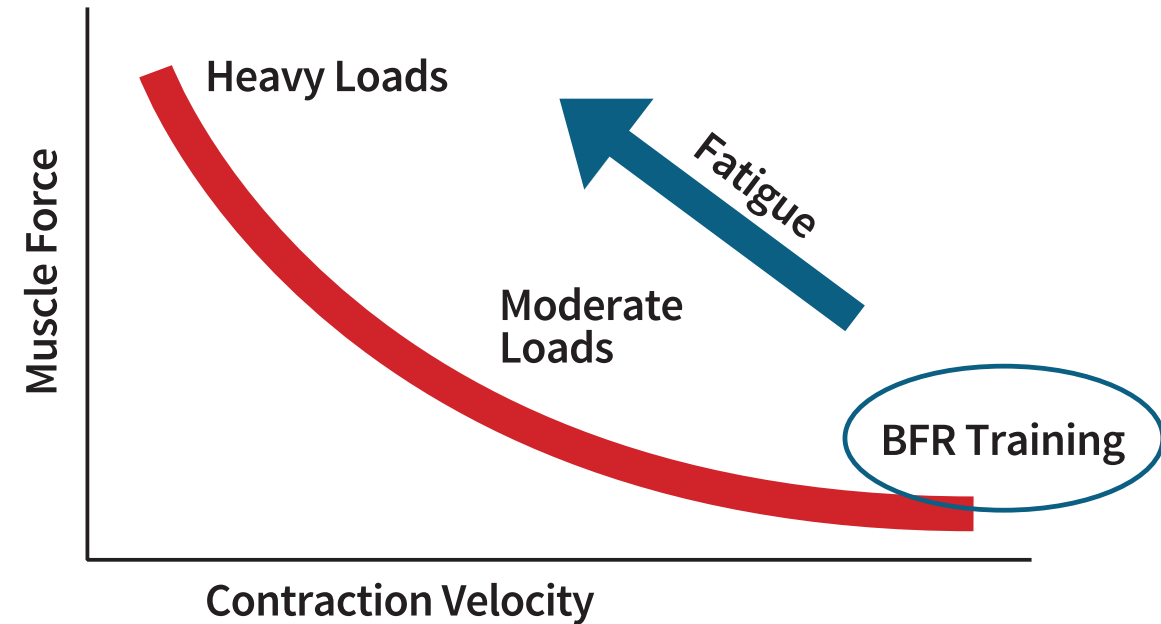
Bar Speed, Failure & Motor Unit Recruitment



Adapted from Chris Beardsley

- Max effort

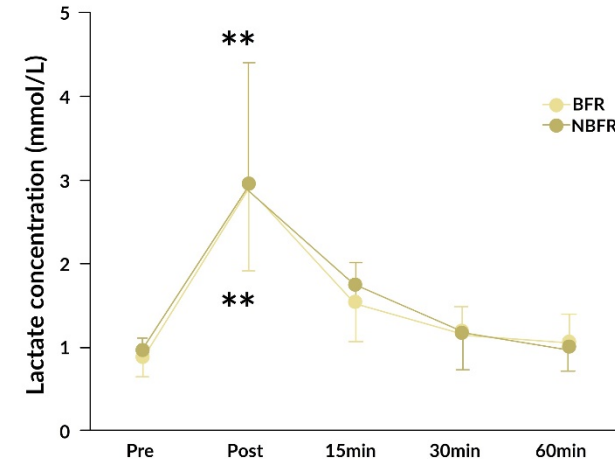
Force-Velocity Curve



CONCLUSIONS

BFR Accelerates Fatigue

- Greater metabolic stress leads to greater peripheral fatigue at a given repetition
- Requires additional exertion (effort) to maintain force production
- Augments high-threshold MU recruitment faster than LL albeit similar levels of overall metabolic stress at task failure (Tanimoto, 2004; Yasuda, 2015)
- Hypertrophic stimulus
- Maybe this all is just nerd-talk and we just need to lift to exertion REGARDLESS of load!



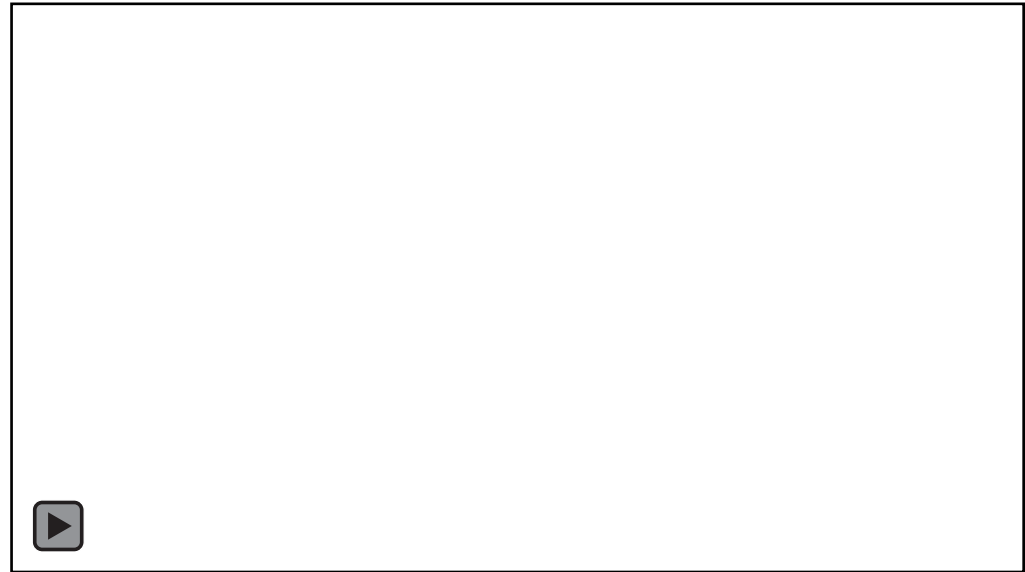
4 sets to failure
of Biceps Curls @
20% 1RM @ 160
mm Hg Applied
BFR Pressure

Yasuda, 2015

THANK YOU!

Special Thanks To:

- Chris Beardsley (@Chrisbeardsley) for his mentorship on helping me dive into fatigue mechanisms with BFR
- Eugen Loki (@Pheasyque) for the amazing illustrations
- Meena Rajgarhia for creating the charts from the research
- You all for allowing me to have this amazing platform to discuss something that is near and dear to my heart!



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