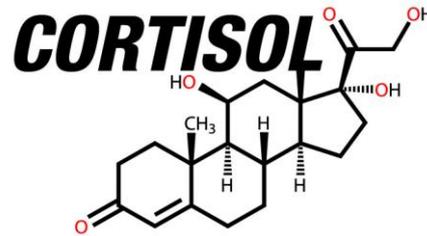


# The Missing Piece: The Need for Recovery

## Understanding Links between Recovery, Stress and Inflammation



Developed by:

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Engage.. Ignite.. Empower..©

## Defining Recovery

Return to normal state of health, mind or body – process that involves rest, refueling (replenish), rehydration, regeneration (repair), re-synthesis, restoration, reduction (inflammation), replacement and ultimately a return to *homeostasis* (discussed next).

- Recovery is least understood piece of exercise-adaptation cycle.

### Types:

- *Immediate recovery* between successive efforts (e.g., between reps within a set).
- *Short-term recovery* between interval sprints or weight training sets (e.g., rest interval between sets/intervals).
- Training recovery between successive workouts or competitions – affected by events outside of exercise session – **MOST important!**

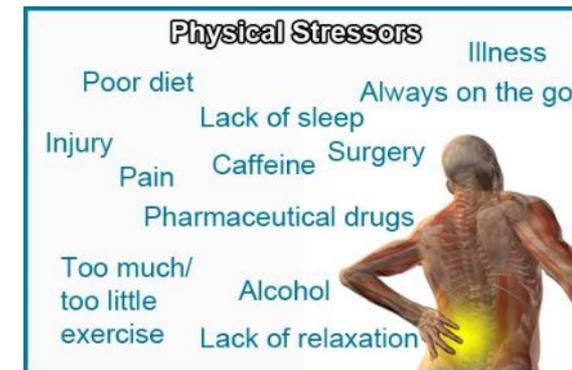


## Homeostasis

**Definition:** Property of a system in which variables are regulated so that internal conditions remain stable and relatively constant (e.g., pH, temperature).

- Any non-specific stimulus that overcomes, or threatens to overcome body's ability to maintain homeostasis = *stressor*.
- Stressors originate from different sources – all manifest physiologically:
  - Environmental (e.g., hot, cold).
  - Chemical (e.g., blood acid-base imbalance, low oxygen supply).
  - Biological (e.g., virus, illness, starvation).
  - Physiological (e.g., pain, vigorous exercise, dehydration).
  - Nutritional (e.g., excessive caloric or carbohydrate restriction, skipped meals).
  - Psychological (e.g., peers, finances).
  - Emotional (e.g., sorrow, fear, anxiety).
  - Social (e.g., life events, personal conflicts).

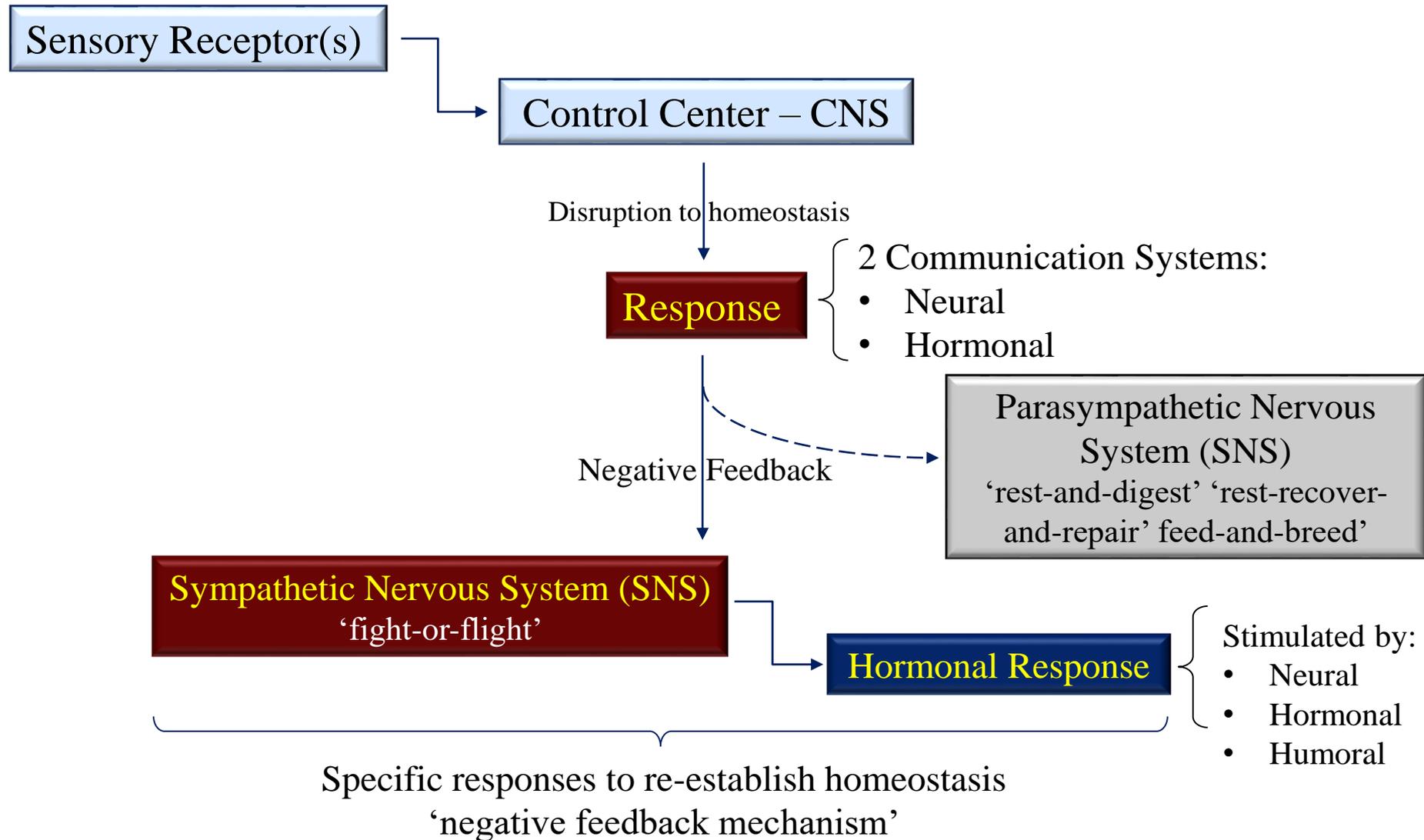
Your body does not know, nor care about the nature of the stressor – they do accumulate...



# Biological Design ....



## Body's Biological Response



## Body's Biological Response

Physiological (Acute, Intense) Stress

→ 'Fight-or-Flight' Response

→ Physiological Work

Allocation of Resources: PNS

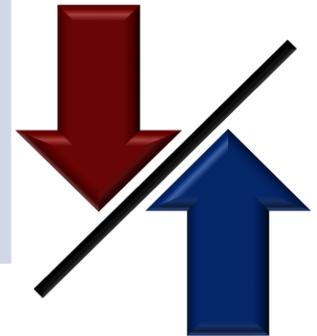


Allocation of Resources: SNS



### Acute Events Inhibited

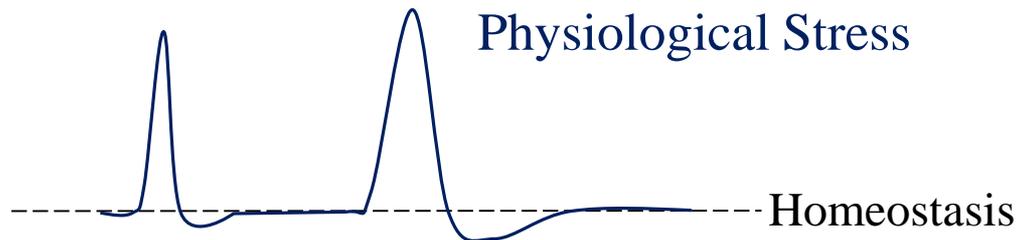
Decreased salivary and digestive enzyme secretion, and digestion.  
Decreased stomach/small intestinal contractility.  
Decreased pain perception (analgesia).  
Decreased growth, repair and maintenance.  
Decreased reproduction capacity.



### Acute Events Activated

Increased cardiopulmonary responses.  
Increased vasodilation.  
Increased mobilization of fuels.  
Increased blood clotting ability.  
Increased large intestinal contractility.  
Increased bladder contractility.  
Increased immune function.  
Increased sweat rates.

Physiological Stress



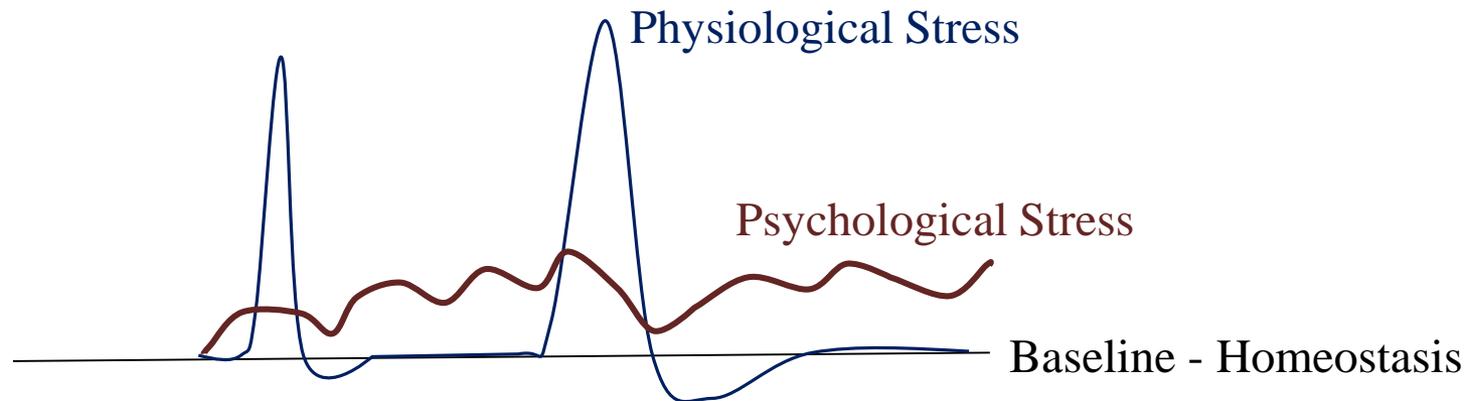
Let's examine more closely ...



# Consequences ....



## How We Live Today

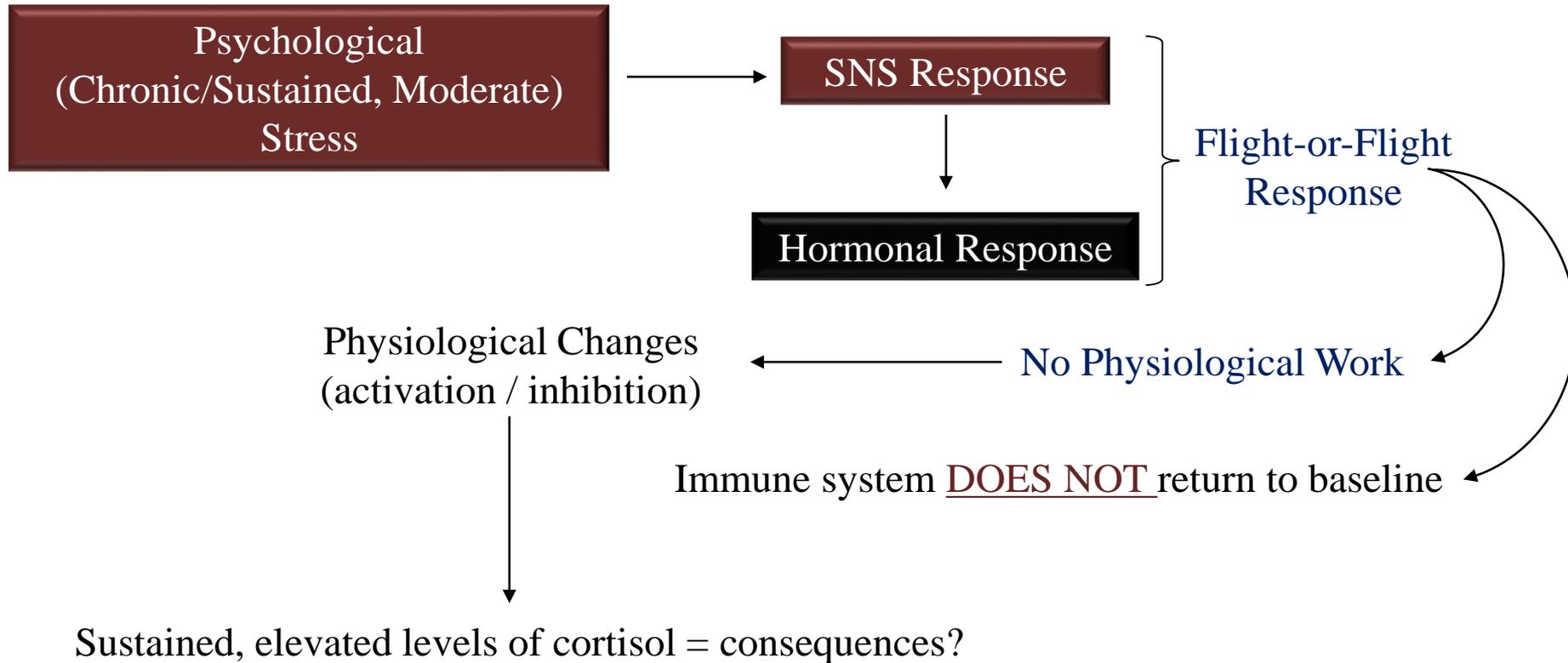


**Remember..... Your body does not know, nor care about the nature of the stressor – they do accumulate...**

What is the difference here – stress or duration of stress?

- The human body is designed to tolerate stress in dosages – adapt or perish, but time is needed to restore homeostasis.
- Kelly McGonigal: TED Talk  
[http://www.ted.com/talks/kelly\\_mcgonigal\\_how\\_to\\_make\\_stress\\_your\\_friend?utm\\_campaign=ios-share&utm\\_medium=social&source=email&utm\\_source=email](http://www.ted.com/talks/kelly_mcgonigal_how_to_make_stress_your_friend?utm_campaign=ios-share&utm_medium=social&source=email&utm_source=email)

# Consequences ....



Stress = elevated cortisol (sustained) = loss of inflammatory control (immune cells desensitized to cortisol's regulatory effect) – sustains inflammation = prolonged damage and disease progression.

# Consequences ....

Recall these changes:

What controls this?



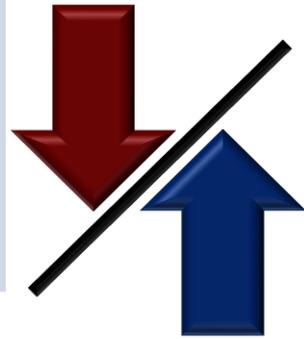
Neuroendocrine systems.

SNS (short-term)

Hormonal (long-term)

## Acute Events Inhibited

Decreased salivary and digestive enzyme secretion, and digestion.  
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## Increased Levels

- Epinephrine / norepinephrine
- **Glucocorticoids** (e.g., **cortisol**, aldosterone).
- Glucagon – breaks down glycogen in liver.
- Prolactin – help decrease other hormones.
- Anti-Diuretic Hormone (ADH)

## Decreased Levels

- Human growth hormone (HGH).
- Insulin.
- Testosterone.
- Estrogen and progesterone.

# Consequences ....



Effects of elevated cortisol ... What does this mean to you?

Target / Hormone	Physiological Role	Effect of Sustained Cortisol
Human Growth Hormone (HGH)	Promote tissue growth and repair – promote fat utilization	Stimulates somatostatin in hypothalamus that inhibits HGH release from the pituitary gland. May also impact HGH need for fetal development
Estrogen	Reproductive function; fat distribution in hips/thighs; fat utilization	Inhibits estrogen production in ovaries, fat cells and adrenal gland; decreased fertilization; decreased libido, increased abdominal fat
Testosterone	Muscle synthesis; may block abdominal fat deposition due to cortisol	Inhibits testosterone production in testes; decreased muscle synthesis; erectile dysfunction; increased abdominal fat
Thyroid stimulating hormone (TSH)	Manufactures thyroxin and triiodothyroxine to regulate metabolism	Inhibits TSH release – can suppress metabolism up to 20% = 250-300 kcal per day or 25-30 lbs. per year
Leptin	Helps regulate appetite and caloric intake	Increased resistance to leptin
Ghrelin	Increases hunger; slows metabolism – prevent starvation and malnutrition	Increased sensitivity within ghrelin receptors

# Consequences ....



## Effects of elevated cortisol ... What does this mean to you?

Target / Hormone	Physiological Role	Effect of Sustained Cortisol
Neuropeptide Y	Neurotransmitter released by hypothalamus – stimulates appetite; inhibited by leptin	Activated by cortisol = greater appetite
Cholecystokinin (CCK)	Slows GI motility (movement) – enhance digestion/absorption; suppresses appetite ~ 20 minutes after eating	Can inhibit CCK effect on suppressing appetite
Aging – chromosomes	Telomeres (chromosomal tails) are constantly repaired by telomerase (enzyme) to help keep cells young and healthy	Reduced telomerase activity = telomere shortening and accelerated cellular aging
Immune system	Repair, recovery, resistance to harmful compounds	Compromised immune function – increased risk for injury and illness
Brain – Hippocampus	Learning – consolidation of information from short-term to long-term memory	Becomes damaged and smaller – reduces long-term memory
Brain – neurons	Brain-derived Neurotropic Factor (BDNF) promotes new and healthy neurons; efficient synapses	Decreases BDNF; reduced neurogenesis (new neurons); shortens dendrites = less efficient synapses in brain

## Immune Function as an Example

**Immune System:** Coordinates body's response to injury, infection or some disturbances to homeostasis.

### Immune Response

#### Innate System

- 1<sup>st</sup> line of defense v. tissue damage, infection or altered homeostasis.
- Fast-acting (minutes to hours).

#### Adaptive System

- Offers only short-term protection – relies upon immunological memory (i.e., having responded to same insults/injuries in past).
- Slow-acting (days to respond).

Let's examine more closely ...



## Immune Function as an Example

**Innate System:** Comprised of immune cells (e.g., monocytes, macrophages) and dendritic cells – in circulation seeking out pathogens, etc.. (they identify and signal discoveries, and initiate inflammatory process to contain/promote healing/recovery.

**Acute phase  
response**



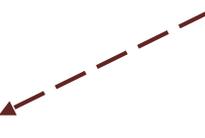
Activates expression of pro-inflammatory immune response genes (e.g. Interleuken-1 [IL-1], Necrosis-factor- $\alpha$  [TNF- $\alpha$ ]) – cytokines (pro-inflammatory or anti-inflammatory messengers)

Promotes T-cell production.



IL-1, IL-6, TNF- $\alpha$  coordinate cell-to-cell communication, control/modify/stimulate neural-endocrine responses.

Promotes vascular permeability (= swelling, heat, redness, pain) – helps reduce bleeding.



Promotes cellular adhesion (= immune cells leaving blood and migrating into tissue).



# Consequences ....



## Immune Function as an Example

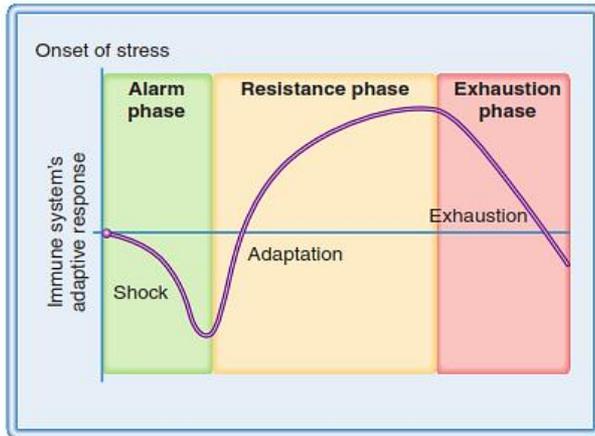


**Innate System:** Inflammation occurs:

- **Systemically** (endocrine) – at systemic levels, IL-6 produces C-reactive protein = elevated core temperature, HR, breath rate, fever, social withdrawal – all accelerate healing and limit infection spread.
- Cytokines (IL-1 [mostly], IL-6 and TNF- $\alpha$ ) also involved in **Hypothalamus-Pituitary-Adrenal (HPA)** axis activation – stimulate glucocorticoids (**cortisol**) = negative feedback on immune cells to suppress further synthesis/release of more cytokines.
  - Protects host from detrimental consequences of overactive immune response (**cortisol = immunosuppressant**).



## Han's Seyle's General Adaptation Syndrome



### 1<sup>st</sup> Phase: Shock or Alarm:

- Initial period of fatigue, weakness and soreness (few days to week).

### 2<sup>nd</sup> Phase: Adaptation or Resistance:

- Continued stress = adaptation (or perish) – changes:
  - Weeks 1-3: Neurological adaptations.
  - Weeks 2-4: Passive tissue strengthening (e.g., tendons).
  - Weeks 3-5: Muscle adaptations begin (2-3 months in older adults).

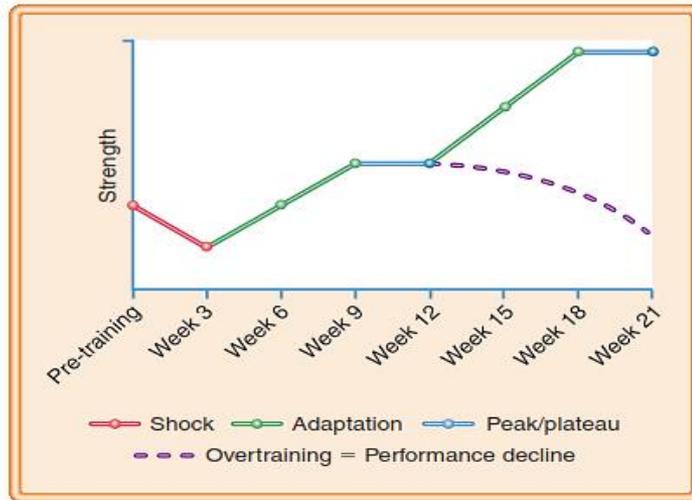
### 3<sup>rd</sup> Phase: Exhaustion:

- Depletion of resources – unable to restore normal function.
- Compromises immune function; increases injury/illness potential.

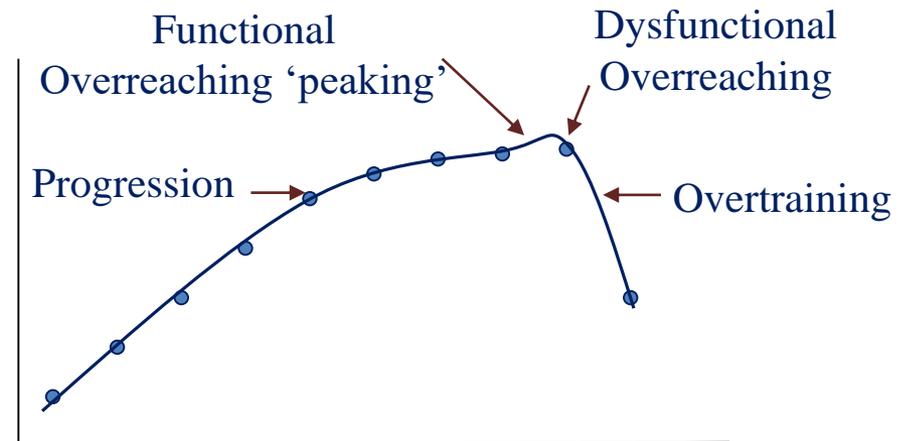
# Consequences ....



## Muscle Response to Training



- Chronic stress – adaptive responses that can be both healthy (continued training) and unhealthy (life stress).



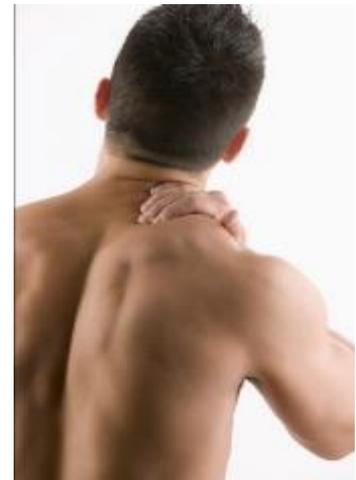
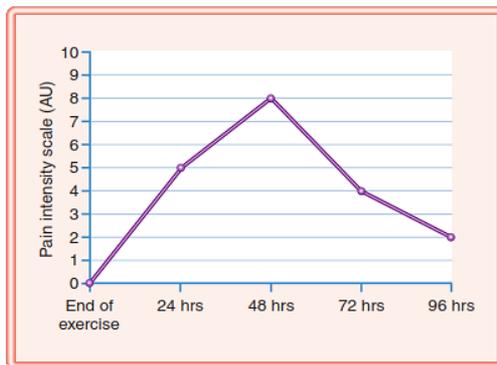
**Training Goal:** Peak performance at appropriate interval to optimize performance by pushing limits (short-term) of load/volume/rate = **Overreaching**.

- Excessive loads, volumes or rates may retard recovery (due solely to exercise program or aggregated effects of other stressors) – leads to ‘dysfunctional overreaching’ and/or ultimately ‘overtraining.’

## Delayed-Onset of Muscle Soreness (DOMS)

Onset occurs between 12-72 hours post-exercise – believed causes:

- Mechanical stresses placed upon muscle and tissue (i.e. eccentric action) = micro-tears within muscle fibers (myofibrils) – causes disarrangement of sarcomeres.
- Triggers immune response – releases histamines and prostaglandins.
- Increases localized edema (accumulation of fluid) = inflammation inside muscle compartment.
  - Both stimulate nociceptor (pain) sensations.



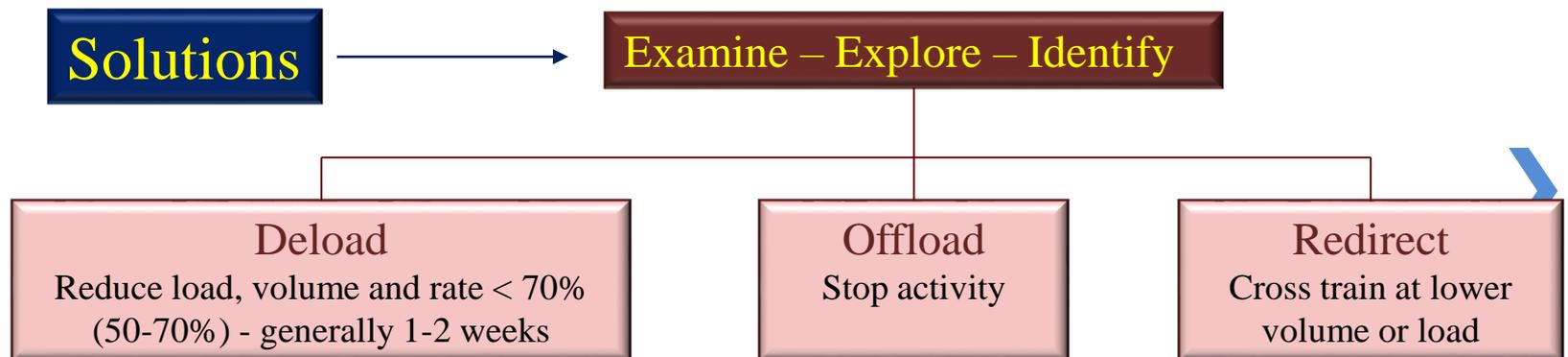
*Reference:* Cheung, K., Hume, P., & Maxwell, L. (2003). Delayed onset muscle soreness: Treatment strategies and performance factors. *Sports Medicine*, 33(2): 145 – 164.

# Monitoring Recovery ...



Overtraining Syndrome – be aware and modify programming as necessary.

- Generally attributed to inadequate recovery – also excessive training or burnout.
  - Symptoms include:
    - Decreased performance over 1-2 week period.
    - Increased resting heart rate and/or blood pressure.
    - Decreased body weight
    - Reduced appetite or loss of appetite; nausea
    - Disturbed sleep patterns and inability to attain restful sleep
    - Muscle soreness and general irritability.
    - Lack of motivation / adherence.



# Thank You..!!

For Your Commitment to Excellence

Questions .. ??



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