Beyond Training

Fixing the Biochemical Roadblocks That Limit Performance & Recovery

Dr. Tara Dunne, BSc, MA, ND

Surpass Human Performance

About The Presenter

Dr. Tara Dunne, BSc, MA, ND

- **Bachelor of Science**
- **Master of Health and Wellness**
- **Doctor of Naturopathic Medicine**
- **Sports Nutrition / IronMan Sports Nutrition**
- **Founder of Surpass Human Performance**

Unique opportunity in practice to combine clinical medicine / primary care with sports biochemistry

SURPASS
HUMAN PERFORMANCE

Training Is Not the Limiting Factor

- Athletes are not under-trained
- Plateaus persist despite intelligent programming

Training vs Adaptation

Training provides the signal

Biochemistry determines the response

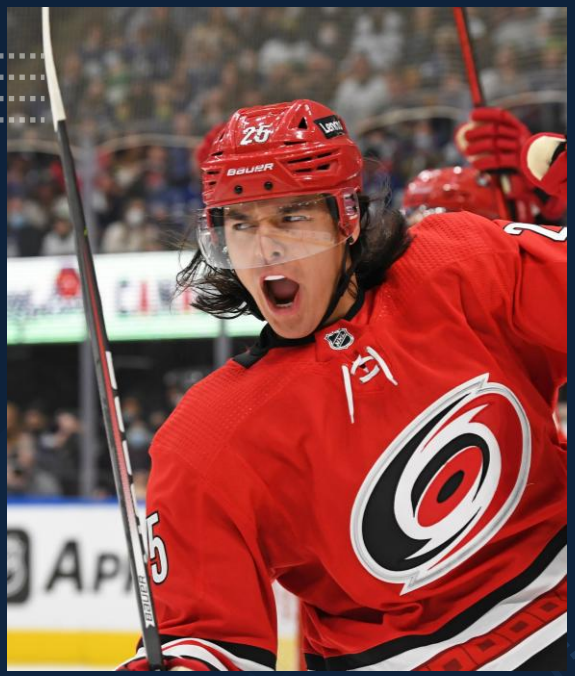
Training usually assumes these systems works:

- Substrate availability (adequate fueling/hydration)
- Oxygen delivery
- Mitochondrial ATP production
- Hormonal signaling

Reality:
Athletes often train
on top of broken systems.

I'll offer general strategies but …Individualized Plans Matter!

- Every Athlete is different.
- Not just in skill levels but in how their biochemistry has adapted and works.



Individualized Plans

- Every Athlete is different.
- Different Sports emphasize different energy production patterns

Individualized Plans

Every Athlete is different.

Genetics also has biochemical implications

Performance Roadblocks

STEP ONE: Finding the weakest link

- Substate / co-factor availability (Fueling and hydration availability)
- Oxygen delivery & utilization
- Mitochondrial efficiency
- Hormonal & inflammatory load

Roadblock #1: Fueling / Hydration Mismatch

- Energy availability regulates adaptation
- Fueling supplies substrates and co-factors for energy production
  - Substrates: CHO, fat, amino acids
  - Co-factors: vitamins and minerals (e.g. B vitamins and iron) antioxidants, amino acids

Roadblock #1: Fueling / Hydration Mismatch

- What does improper fueling (LEA/ RED-S) impact?
  - Sometimes referred to as LEA (low energy availability) or historically RED-S (relative energy deficiency in sport)
  - LEA (especially CHO) → Poor glycogen restoration
    - Glycogen=stored glucose primarily stored in liver and muscle tissue)
    - Body is **not adequately replenishing glycogen** in muscle and liver after exercise
    - Chronic energy debt
  - Reduced power at higher intensities
  - Hormonal suppression (especially thyroid & reproductive hormones…more to come on this)
  - Elevated cortisol → poor sleep & recovery

LEA/RED-S vs Stress vs Post-Viral: How to Differentiate

Feature	RED-S	Stress-dominant	Post-viral
AM and PM cortisol	PM High	PM High-normal	PM Variable
Potassium	Normal or ↑	↑	↑
Neutrophils	↓	↓	↓
Fatigue	Often prominent	Subtle	Variable
Performance	Plateau	Plateau	Drop
Infections	Yes	Yes	Yes

Roadblock #1: Fueling / Hydration Mismatch

- Often seeing improper macros consumed
  - Misinformation: Athletes tend to set a daily macros goal but forget to increase on training days
  - Misinformation: Athletes rely on general sports nutrition guidelines and don't factor in the needs of their specific sport (eg: endurance vs power)
  - Misinformation: Athletes with female physiology keep the same macros all month long instead of using monthly hormone fluctuations to guide their intake
  - Misinformation – Most GI distress in athletes from using a "more is more" approach to nutrition / hydration
  - Misinformation: Athletes tend to set macros goals for the day but don't time the intake of these things appropriately.

Roadblock #1: Fueling / Hydration Mismatch

- Fuel timing matters
  - Want to have energy available before, during and after training.
    - After training, specifically refueling within 30-60 minutes of completing sport. This nutrition will enhance (or limit!) adaptation
    - Focusing on protein (30-60 grams post depending on male vs female vs age-and-stage physiology)
    - Focusing on carbohydrate if there is heavy cardiovascular elements to the training (30-60 grams post depending on male vs female vs age-and-stage physiology)

Roadblock #1: Fueling / Hydration Mismatch

- **Improper hydration also seen often**
- **Inadequate amounts of sodium during training / sport**
  - Sweat rate calculations
  - Serum investigations
  - Pre and Post exertion urinalysis
  - Typically recommended a MINIMUM of 400 mg sodium per day and then between 400-1200 mg per hour during training, especially for endurance sport, for training longer than 1 hour

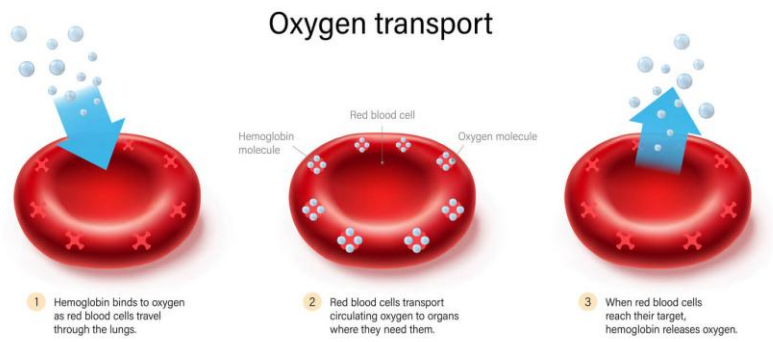
Roadblock #2: Oxygen Utilization / Iron

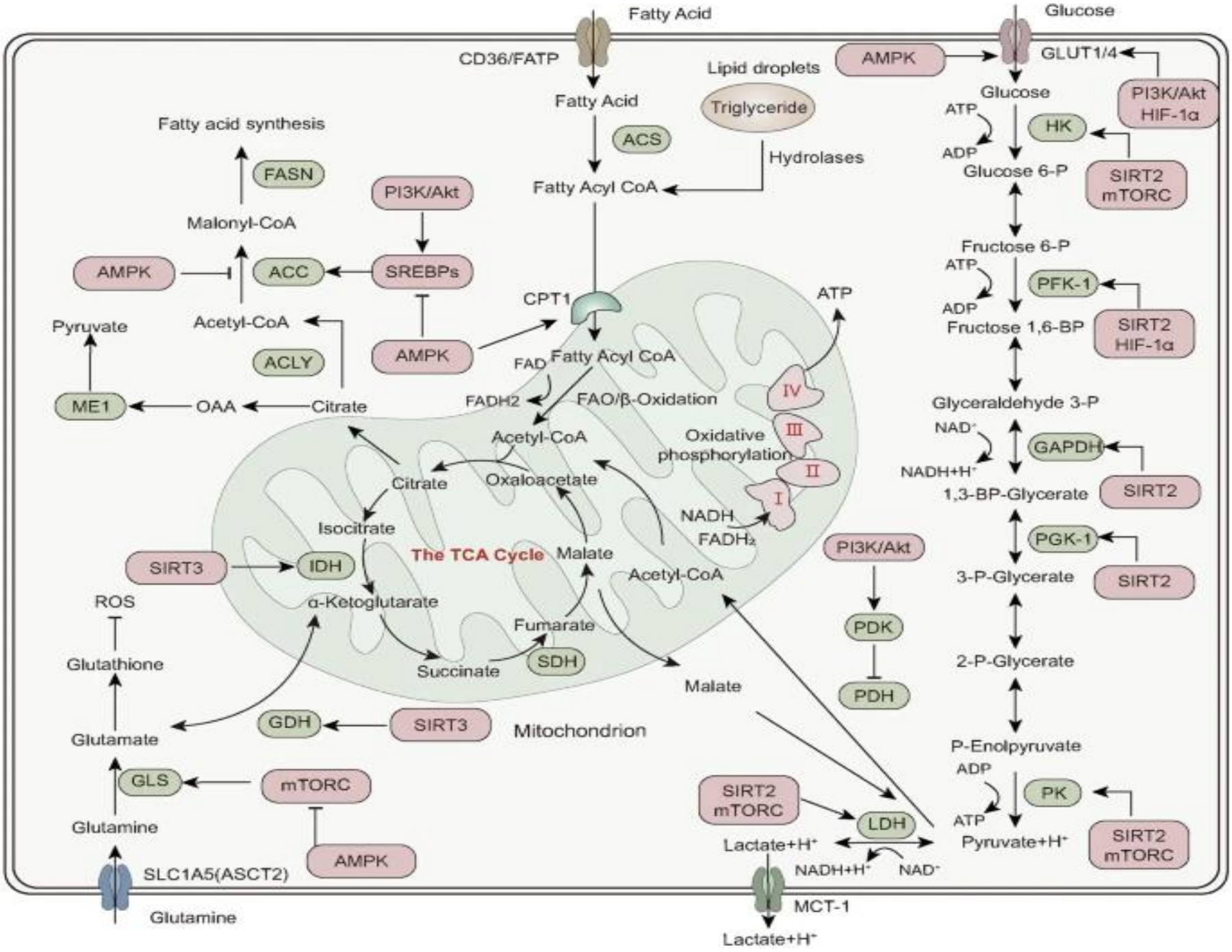
- VO2max ≠ performance
  - Delivery vs cellular use (extraction)
  - Mismatch is common
  - Moxy can help us see delivery vs extraction
    - Moxy is a non-invasively measure muscle oxygen saturation (SmO_2) and total hemoglobin in real-time during exercise

Roadblock #2: Oxygen Utilization / Iron

- **Why is iron focus so important?**
  - Iron is usually framed around oxygen transport

    Oxygen Transport = Each hemoglobin molecule contains 4 heme groups
    Each heme group contains one iron atom (Fe^{2+})
    Oxygen binds directly to iron, not to the protein itself
  - BUT it is also central to mitochondrial function:
    Supports electron transport chain enzymes
  - So even “borderline” iron deficiency can impair ATP production before anemia ever appears.
    This is why athletes can feel flat long before hemoglobin drops.




Fatty Acid
CD36/FATP
Fatty Acid
ACS
Fatty Acyl CoA
Lipid droplets
Triglyceride
Hydrolases
Glucose
AMPK
GLUT1/4
PI3K/Akt
HIF-1α
Glucose
ATP
ADP
HK
Glucose 6-P
SIRT2
mTORC
Fructose 6-P
ATP
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HIF-1α
Glyceraldehyde 3-P
NAD+
NADH+H+
GAPDH
SIRT2
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PGK-1
SIRT2
3-P-Glycerate
2-P-Glycerate
P-Enolpyruvate
ADP
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mTORC
Fatty acid synthesis
FASN
Malonyl-CoA
AMPK
ACC
SREBPs
PI3K/Akt
Acetyl-CoA
ACLY
Pyruvate
ME1
OAA
Citrate
CPT1
ATP
FAD
FADH2
Fatty Acyl CoA
FAO/β-Oxidation
Acetyl-CoA
Oxidative phosphorylation
IV
III
II
I
NADH
FADH2
Citrate
Oxaloacetate
Isocitrate
The TCA Cycle
Malate
Acetyl-CoA
SIRT3
IDH
α-Ketoglutarate
Fumarate
Succinate
SDH
ROS
Glutathione
Glutamate
GDH
SIRT3
Mitochondrion
Malate
PI3K/Akt
PDK
PDH
GLS
mTORC
Glutamine
AMPK
SLC1A5(ASCT2)
Glutamine
SIRT2
mTORC
LDH
Lactate+H+
NADH+H+
NAD+
MCT-1
Lactate+H+

Roadblock #2: Oxygen Utilization / Iron

- **Why is iron focus so important?**

- **RBC Turnover increases with training load**
  - **Most of the body's iron is tied up in RBCs**
  - **RBC turnover exceeds replacement**
  - **This changes how iron is used**

- **Iron Beyond Ferritin Objectively**
  - **Look for any iron dysfunction (not just low ferritin)**
  - **Important to look at RBC's, hemoglobin, hematocrit, TIBC and saturation as well as ferritin**

Roadblock #2: Oxygen Utilization / Iron

Iron Deficiency / Functional iron deficiency common in:

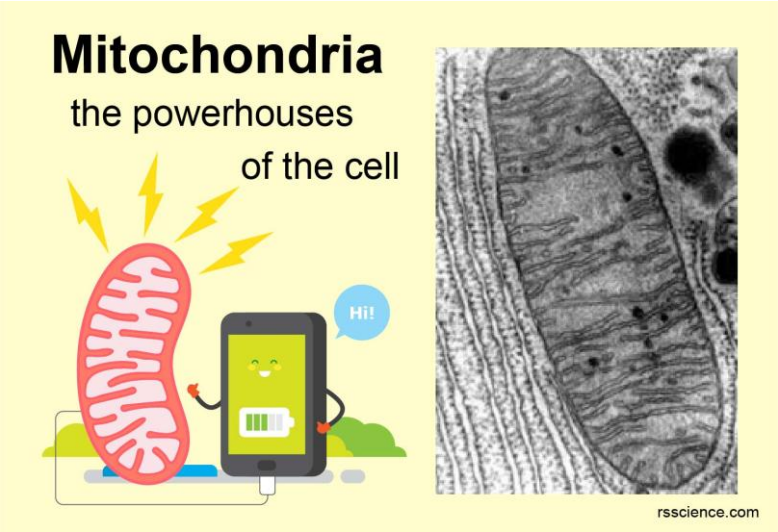
- Endurance athletes
- Female athletes
- High-intensity team sports (eg hockey, short burst maximal effort)
- Frequent Training Sessions elevate hepcidin
  - Hepcidin blocks availability - Hepcidin is a hormone that regulates iron by inhibiting the transport of iron from the diet and its release from storage in the liver and macrophages.
  - When estrogen declines, hepcidin increases. Consideration here is cycle timing with objective testing ang perimenopause / menopause

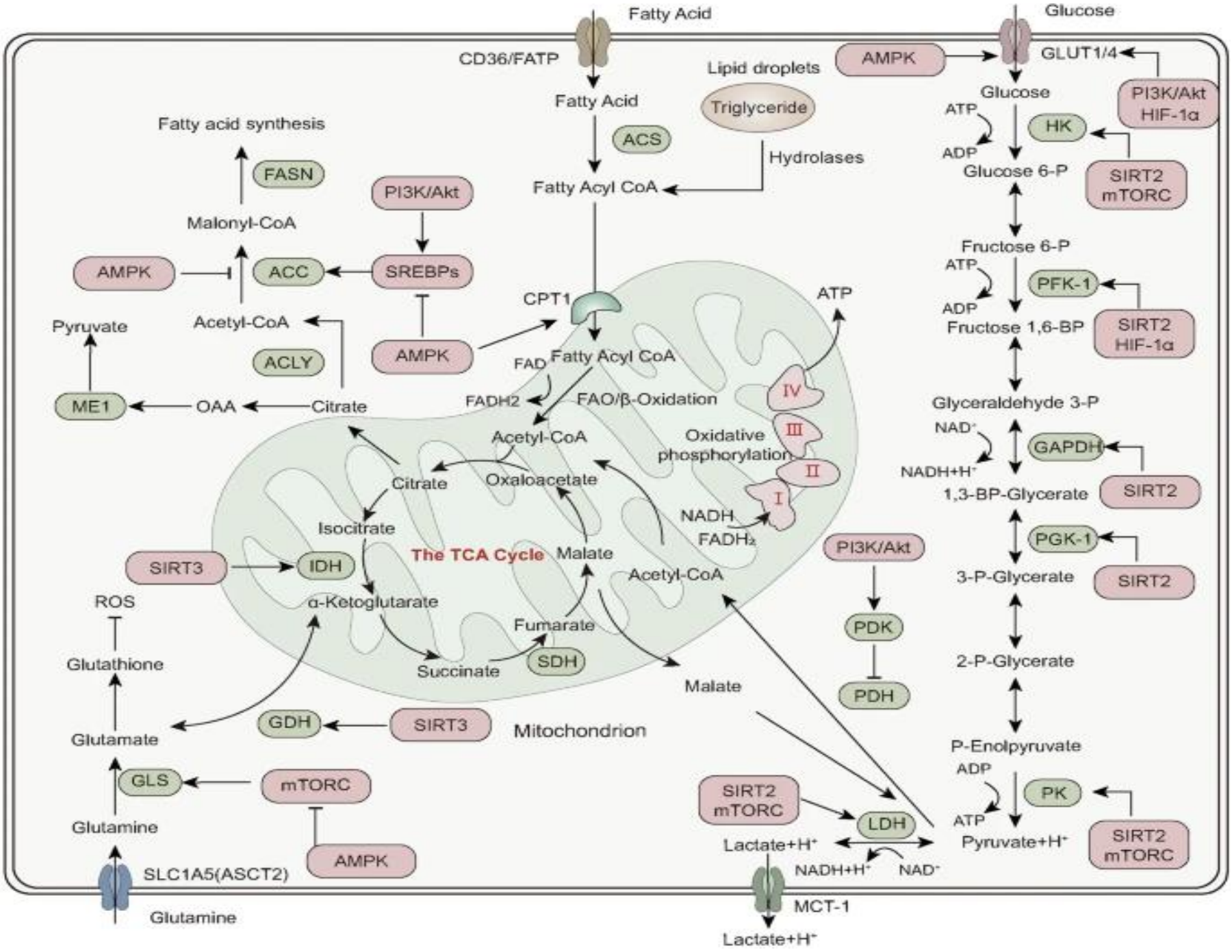
Subjective and objective indicators of iron deficiency/dysfunction:

- Early lactate rise
- “Legs give out before lungs” “Heavy legs”
- High perceived effort at submaximal workloads
- Loss of repeat sprint ability / poor repeatability

Roadblock #3: Mitochondria

- ATP production = Energy
- ATP production issues limit output
- Recovery is energy-dependent
  - Rate / quality of recovery depends on ATP efficient ATP production

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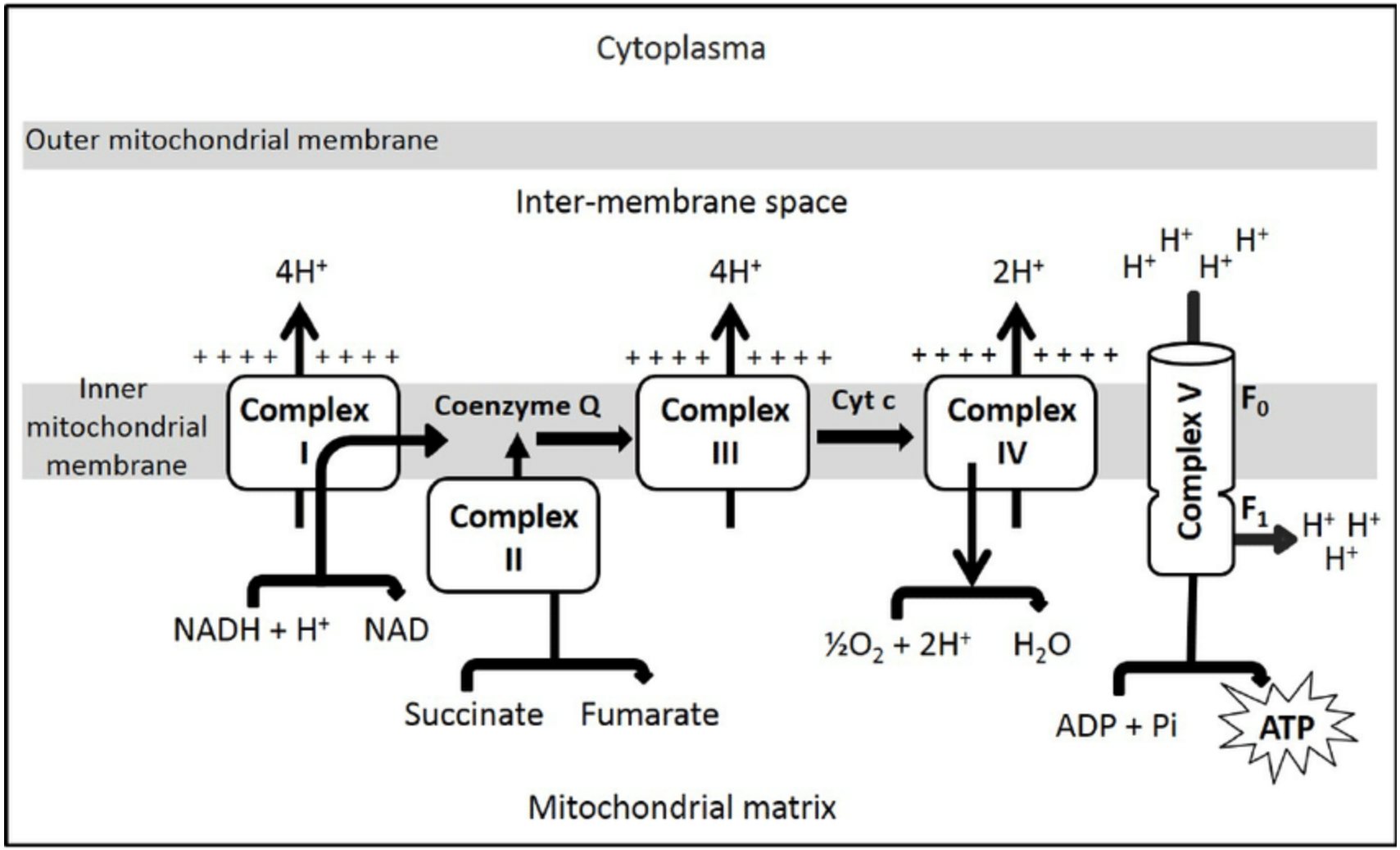
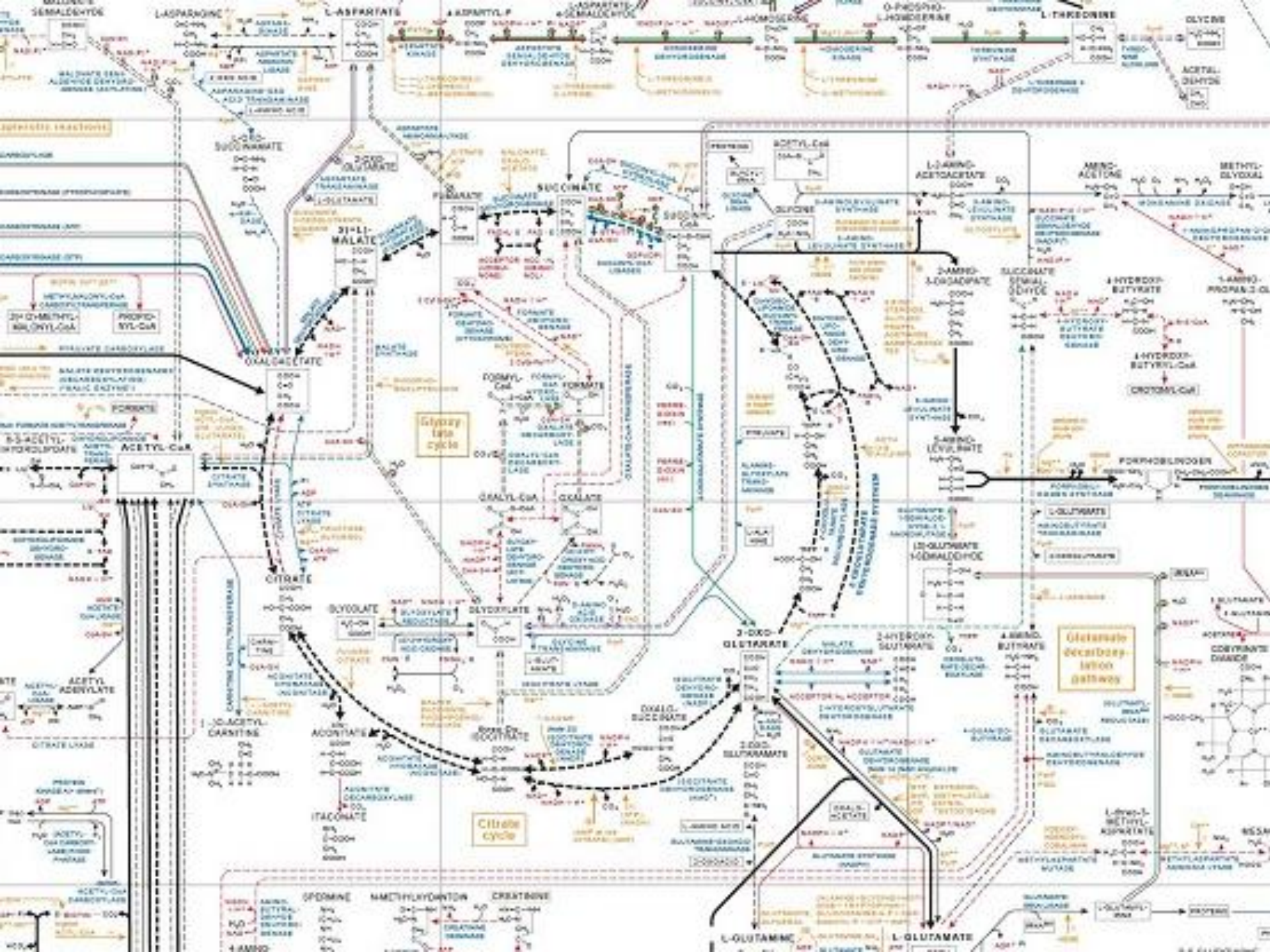
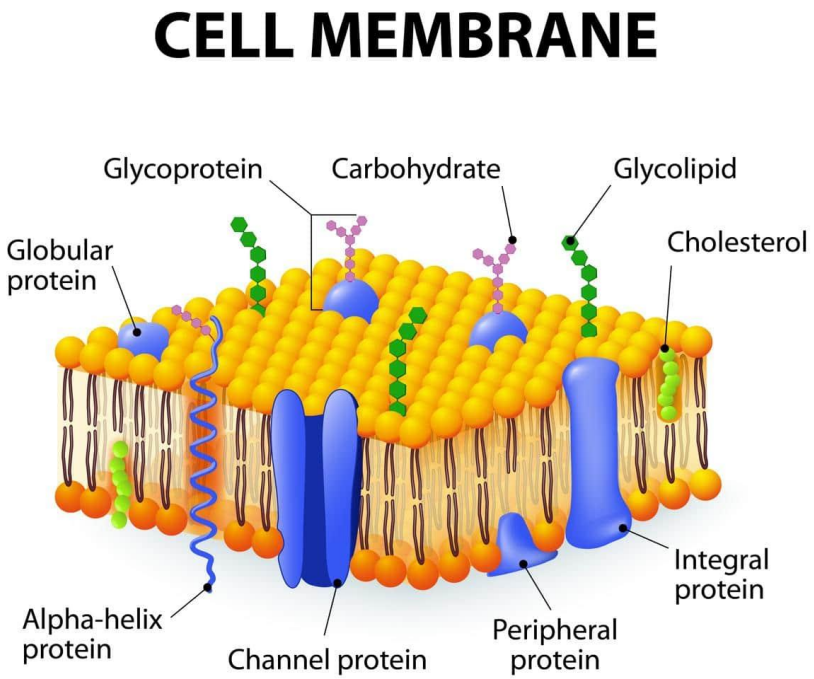

Cytoplasma
Outer mitochondrial membrane
Inter-membrane space
4H+
4H+
2H+
H+ H+ H+
H+ H+
Inner mitochondrial membrane
Complex I
Coenzyme Q
Complex III
Cyt c
Complex IV
Complex V
F0
F1
Complex II
H+ H+
H+
NADH + H+
NAD
½O2 + 2H+
H2O
Succinate
Fumarate
ADP + Pi
ATP
Mitochondrial matrix


L-ASPARAGINE
L-ASPARTATE
L-ASPARTATE SEMIALDEHYDE
L-HOMOSERINE
L-THREONINE
GLYCINE
ACETALDEHYDE
SUCCINATE
FUMARATE
L-MALATE
OXALOACETATE
ACETYL-CoA
CITRATE
ISOCITRATE
2-OXOGLUTARATE
SUCCINYL-CoA
GLYOXYLATE
GLYCOLATE
OXALYL-CoA
OXALATE
FORMATE
FORMYL-CoA
ACONITATE
ITACONATE
L-GLUTAMATE
L-GLUTAMINE
L-GLUTAMATE 1-SEMIALDEHYDE
2-AMINO-3-OXOADIPATE
L-2-AMINO-ACETOACETATE
AMINO-ACETONE
METHYLGLYOXAL
4-HYDROXY-BUTYRATE
4-HYDROXY-BUTYRYL-CoA
CROTONYL-CoA
5-AMINO-LEVULINATE
FORMIMINOGLYCINE
SUCCINATE SEMIALDEHYDE
2-OXO-GLUTARAMATE
2-HYDROXY-GLUTARATE
4-AMINO-BUTYRATE
L-GLUTAMATE 1-SEMIALDEHYDE
L-threo-3-METHYL-ASPARTATE
SPERMINE
METHYLHYDANTOIN
CREATININE
Glyoxylate cycle
Citrate cycle
Glutamate decarboxylation pathway

Roadblock #3: Mitochondria

Common issues with the mitochondria

- Cell membrane permeability issues
- Impaired lactate oxidation
- Infection (yeast/fungal, bacterial, viral)



Roadblock #3: Mitochondria

1. Cell membrane permeability issues

- Reactive oxygen species (ROS) attack membrane lipids and proteins.
  - ROS are normal byproducts of cell metabolism, especially from **mitochondria** during ATP production.
  - Common examples include: Superoxide (O_2^-), Hydrogen peroxide (H_2O_2), Hydroxyl radicals (•OH)

- ROS Productions exceeding antioxidant defenses → Oxidative Stress

- This happens with **intense training / overtraining,** infection, autoimmune diseases (cancer), autoimmune disease treatment (chemotherapy), poor antioxidant status (COq10, vitamin E, glutathione, selenium, etc), allergy, **alcohol, smoke (fires)**

Mechanism

ROS → **lipid peroxidation-**→Membrane becomes **rigid or unstable** → Transporters and receptors malfunction

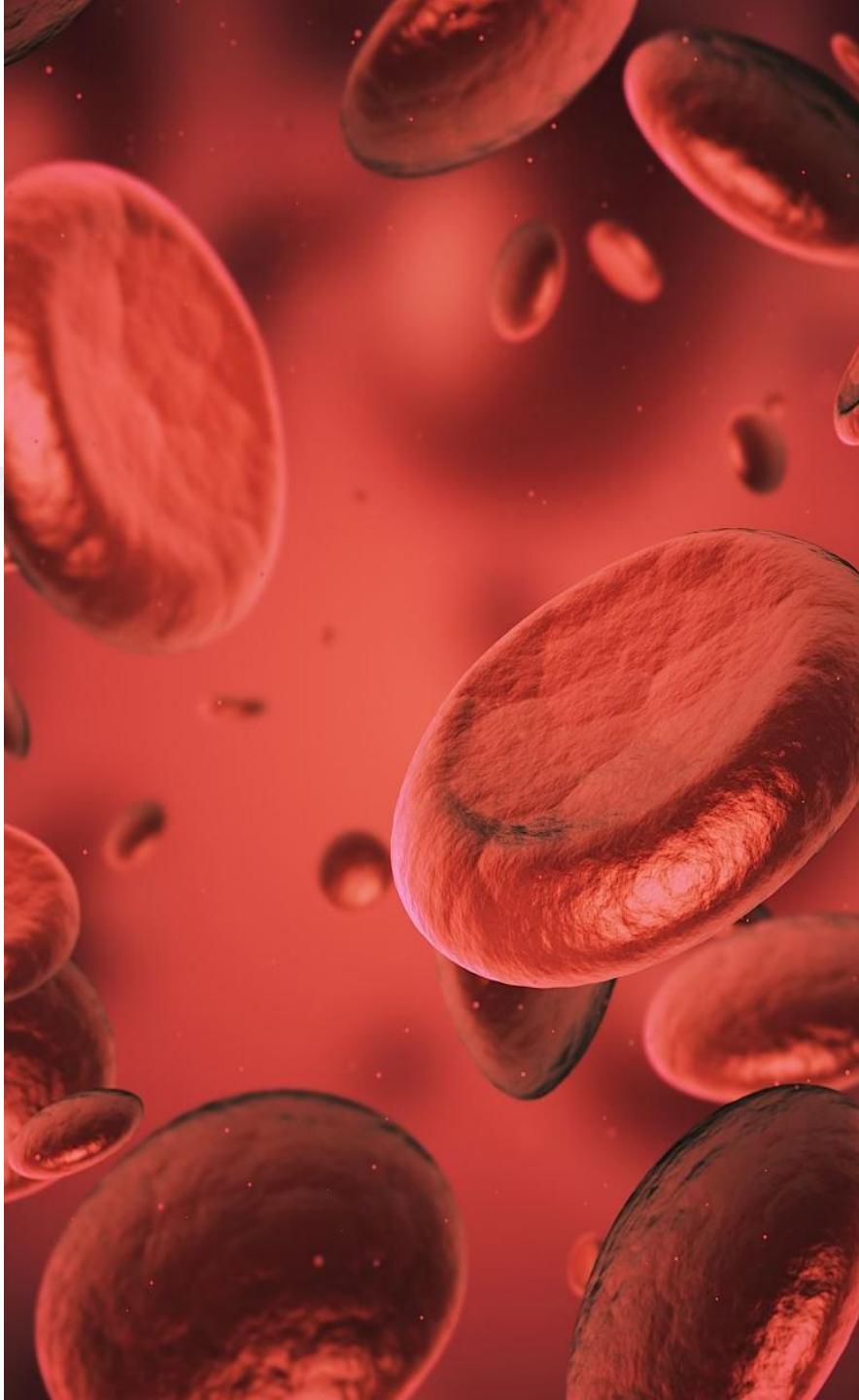
Altered ion gradients (Na^+, K^+, Ca^{2+})

Roadblock #3: Mitochondria

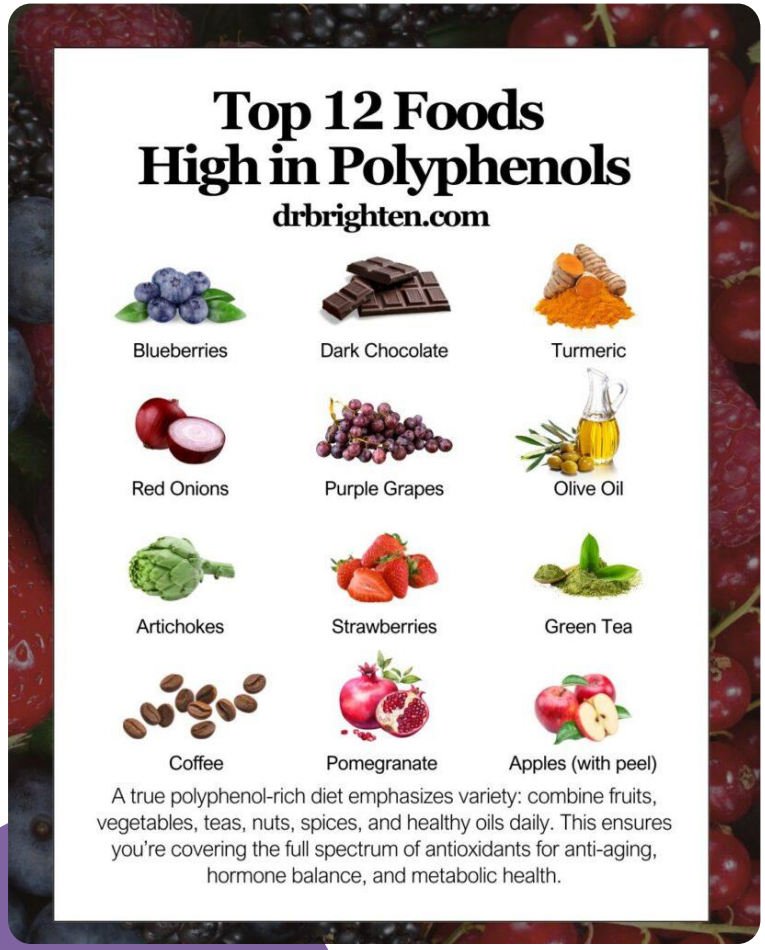
Cell membrane permeability issues

Excess ROS can lead to:

- Fatigue
- Reduced performance
- Slower recovery
- Muscle damage / Injury
- Impaired red blood cell survival



Roadblock #3: Mitochondria



TREATMENT:

- Fix energy availability (adequate calories + carbs)
- Periodize training (avoid chronic high glycolytic load)
- Treat iron deficiency (oxygen mismatch = ↑ ROS)
- Sleep optimization (critical for antioxidant enzyme repair)
- Apply Antioxidants
  - COq10
  - Glutathione
  - Superoxide dismutase
  - Vitamin E
- Nutrition = P+ P (Protein and Polyphenols)

Roadblock #3: Mitochondria

2. Impaired Lactate Oxidation

What is lactate oxidation?

Lactate → transported into mitochondria 2. Converted to pyruvate 3. Enters the TCA cycle → ATP

What does "impaired lactate oxidation" mean?

Lactate production is normal or high
Clearance and reuse are reduced
Lactate accumulates earlier and at lower workloads

Impaired lactate oxidation and mitochondrial dysfunction reinforce each other in a vicious cycle.
When mitochondria cannot oxidize lactate efficiently, lactate accumulates early → acidosis rises → glycolytic stress increases → mitochondrial damage worsens → lactate handling declines further

Glycolytic stress is a metabolic state where the body is **over-reliant on fast glycolysis for ATP** because oxidative (mitochondrial) pathways can't keep up with demand (You are making energy **fast but inefficiently**, creating excess by-products and stress signals)

*Performance depends less on *how much lactate you make* and more on **how well you oxidize it***

Lactate is **not waste** — it is a **preferred fuel.**
Well-trained cells **produce lactate AND immediately reuse it**

Roadblock #3: Mitochondria

2. Impaired Lactate Oxidation

Causes:

- Overtraining
- Under-fueling (low carb, low energy availability)
- Chronic inflammation
- Iron dysregulation
- Viral illness
- Vitamin / Mineral deficiency (B vitamins especially)

Can I fix it with more threshold work? NO!

More threshold work often makes it worse

You increase **lactate production**

But this doesn't fix:

Mitochondria

Transport capacity

Fuel / substrate availability

Result: higher stress, worse clearance

Roadblock #3: Mitochondria

2. Impaired Lactate Oxidation

Treatment

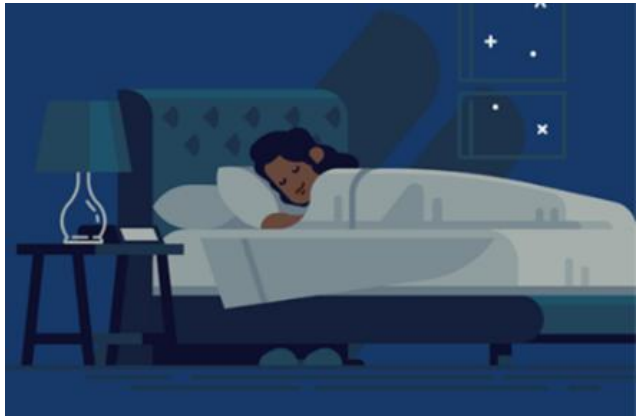
Impaired lactate oxidation is a mitochondrial problem expressed through training.

SUPPLEMENTS

PEA (Palmitoylethanolamide) and **Omega-3 fatty acids (EPA/DHA)**
Stabilize membranes, reduce inflammatory lipid peroxidation
Phosphatidylcholine
Supports mitochondrial and cellular membrane repair
CoQ10 (ubiquinone or ubiquinol)

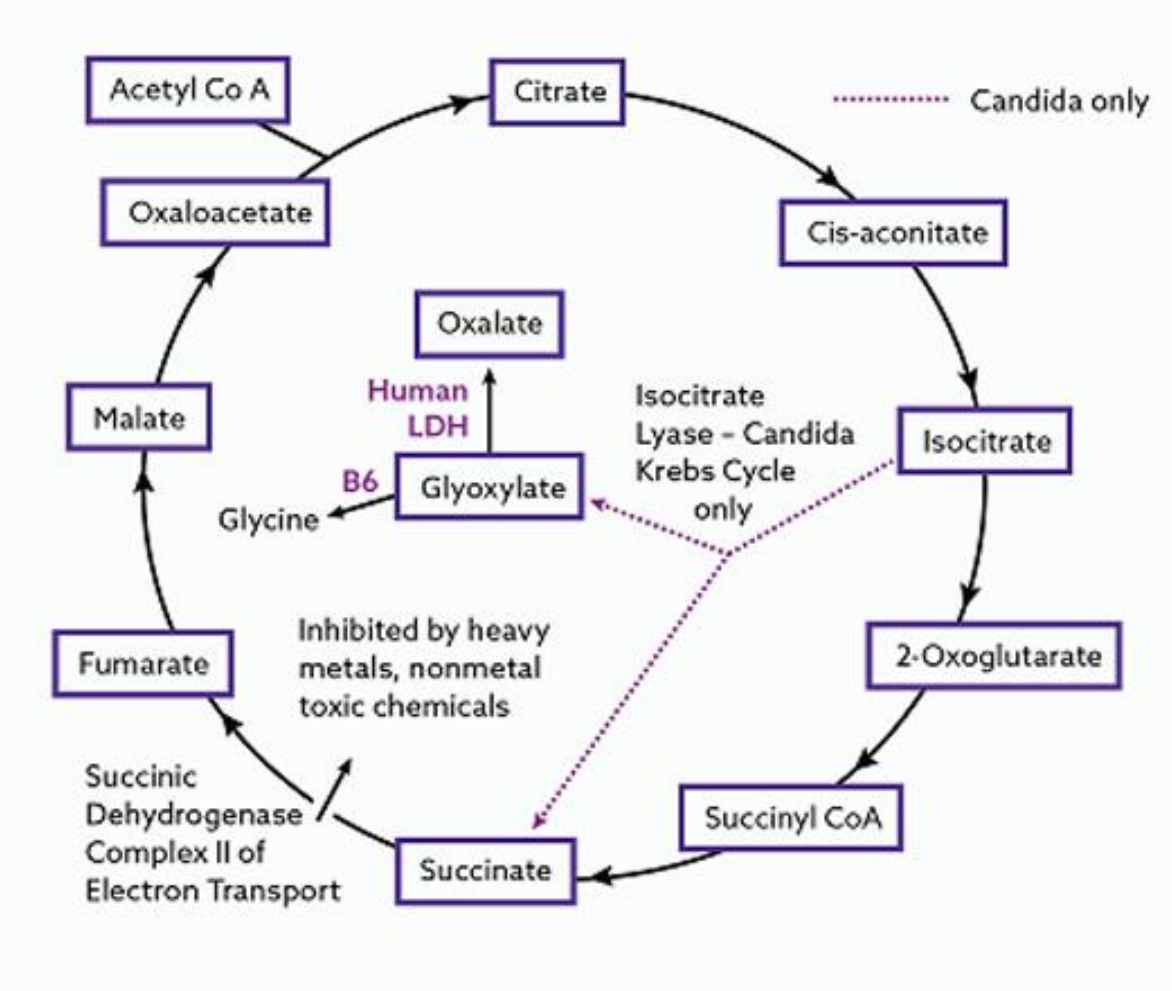
Electron transport chain efficiency

- **Zone 2 aerobic training**
- **Adequate carbohydrate availability**
- **Sleep (7–9 hrs)**



Roadblock #3: Mitochondria

3.) Infection – Clinical Medicine Implications



Roadblock #3: Mitochondria

3.) Infection

Glycolytic Cycle Metabolites				
22	Lactic	≤ 48	9.7	9.7
23	Pyruvic	≤ 9.1	3.0	3.0
Mitochondrial Markers - Krebs Cycle Metabolites				
24	Succinic	≤ 9.3	2.3	2.3
25	Fumaric	≤ 0.94	0.11	0.11
26	Malic	0.06 - 1.8	0.73	0.73
27	2-Oxoglutaric	≤ 35	6.3	6.3
28	Aconitic	6.8 - 28	7.0	7.0
29	Citric	≤ 507	90	90

Roadblock #4: Hormones

- Hormones do not create performance — they *coordinate* it
- They determine whether fuel is accessible, whether mitochondria can function efficiently, and whether tissue repair occurs after training
  - Cortisol regulates fuel
  - Thyroid regulates output
  - Sex hormones support repair (estradiol, progesterone, testosterone).

Roadblock #4: Hormones

Cortisol

- Cortisol is not bad — it is essential. The problem is chronic dysregulation.
- **Both High and Low Cortisol Impair Performance**
  - Chronic elevation → catabolism & poor sleep
  - Suppression → fatigue & low stress tolerance
  - Rhythm matters more than single values
    - Morning cortisol should be high
    - Should start to taper off mid day
    - Nice and low in the evening for relaxation and sleep
    - OBJECTIVE Testing AM and PM
- High cortisol often reflects excessive training stress or under-fueling. Low cortisol often reflects long-term stress exposure where the system has downregulated.
- Both states impair performance, recovery, and immune function.

Roadblock #4: Hormones

Thyroid Hormone

- **TSH Is Not the Whole Story -** "Normal" labs can still impair performance
- Always test TSH, Free T4, Free T3 and anti thyroid antibodies
- T3 drives mitochondrial output
  - T3 directly influences mitochondrial ATP production and carbohydrate utilization.

Athletes may have normal TSH but insufficient T3 signaling due to:

- Low energy availability
- Chronic stress
- Inflammation
- This creates a low-output physiology despite "normal" labs.

Roadblock #4: Hormones

Thyroid Hormone

- **What It Looks Like in Practice**
  - Slower warm-up and reduced intensity tolerance
  - Cold sensitivity or poor thermoregulation
  - Sleep disturbances despite fatigue
  - Prolonged soreness and recovery time

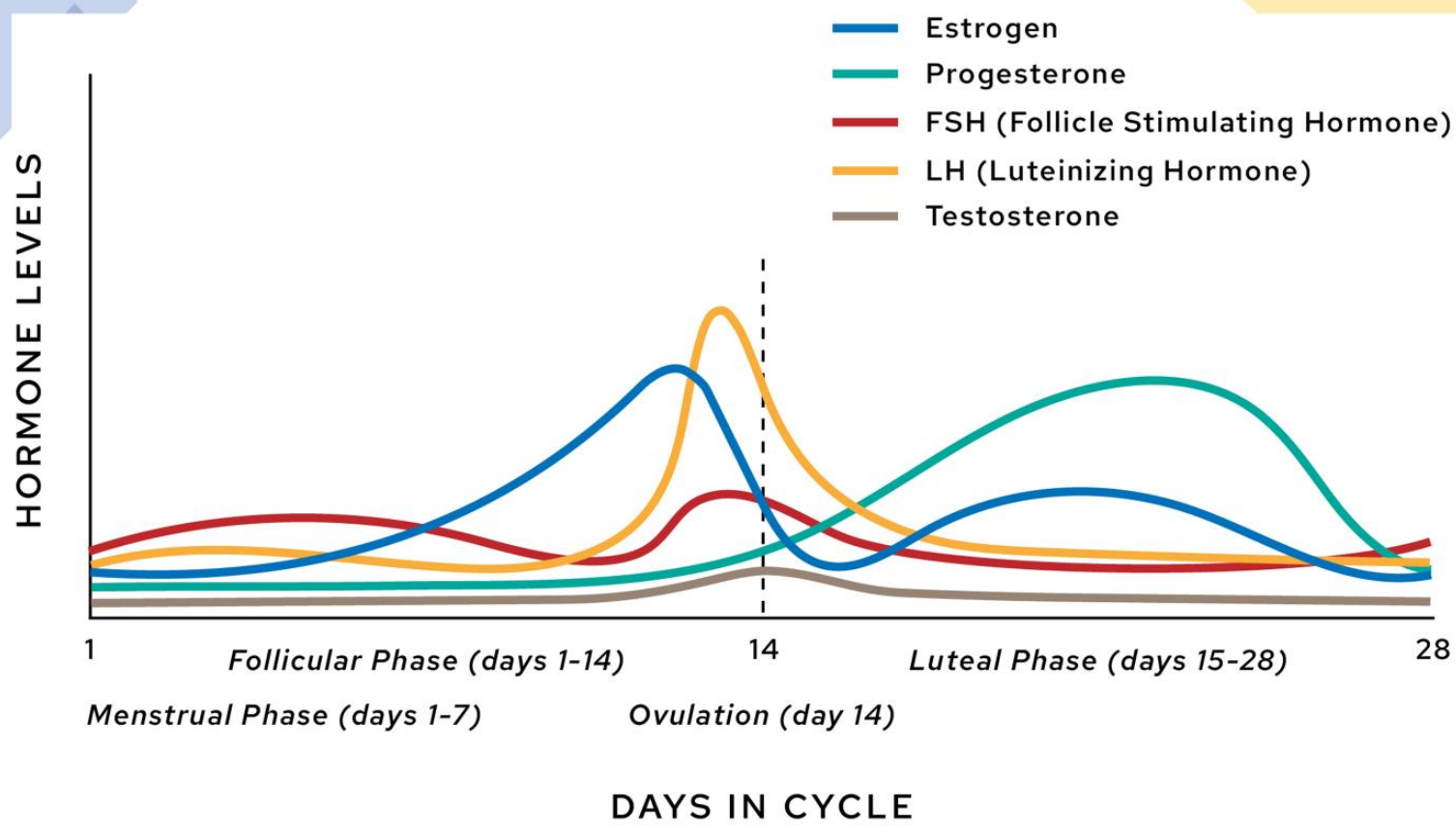
Roadblock #4: Hormones

Sex Hormones

- **Estrogen**
  Improves muscle repair, mitochondrial efficiency, and fat oxidation; stabilizes connective tissue and reduces muscle damage.
- **Progesterone**
  Raises core temperature and ventilation; can increase fatigue, reduce heat tolerance, and impair sleep and recovery when elevated.
- **Testosterone**
  Supports muscle protein synthesis, neuromuscular power, and training adaptation; low levels slow recovery and strength gains.
- **Hormonal fluctuations**
  Change fuel use, thermoregulation, ligament stiffness, and nervous system load—affecting readiness, injury risk, and durability.

- Hormones don't determine *if* you can perform—but they strongly influence **how well you adapt and recover** from training.

Roadblock #4: Hormones



Roadblock #4: Hormones

Training Timing

Training or racing during high hormone phase

- Estrogen will turn down capacity to build muscle
- Progesterone will turn up capacity to breakdown muscle.

Follicular phase

- Hardest Push here!
- Lift heavy, sprint, or do high-intensity workouts

Luteal phase

- During this time, endurance capacity is lower, so steady-state work with low-to-moderate intensity along with more recovery is optimal.
- Work on maintenance and technique

Recovery – Recover Hard!

- Especially in the Luteal Phase

Clinical & Coaching Takeaways

Identify	Identify Roadblocks – Surpass Human Performance can help!
Remove	Remove Roadblocks
Apply	Then apply load

SURPASS

HUMAN PERFORMANCE

Surpass Human Performance bridges sports science and clinical medicine to help athletes train, perform, and recover at their true capacity.

We combine:

- **Comprehensive blood work to identify nutrient, hormonal, inflammatory, and recovery limitations**
- **Performance testing including lactate profiling, VO_2 max, Moxy to define true physiological thresholds**
- **Urine testing to assess metabolism, mitochondrial function, and stress load**
- **One-on-one consulting to integrate data into clear, actionable strategies**

Our focus is simple but powerful:
identify and remove the biochemical barriers that limit performance, adaptation, and recovery—so training actually works.

SURPASS

HUMAN PERFORMANCE

Email: info@surpasshumanperformance.com

Phone: 250-224-7277