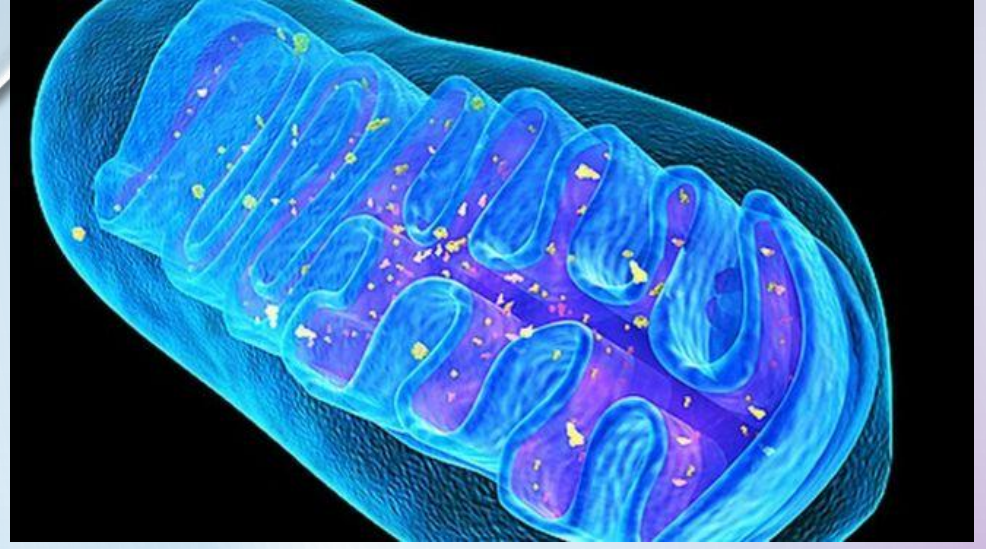


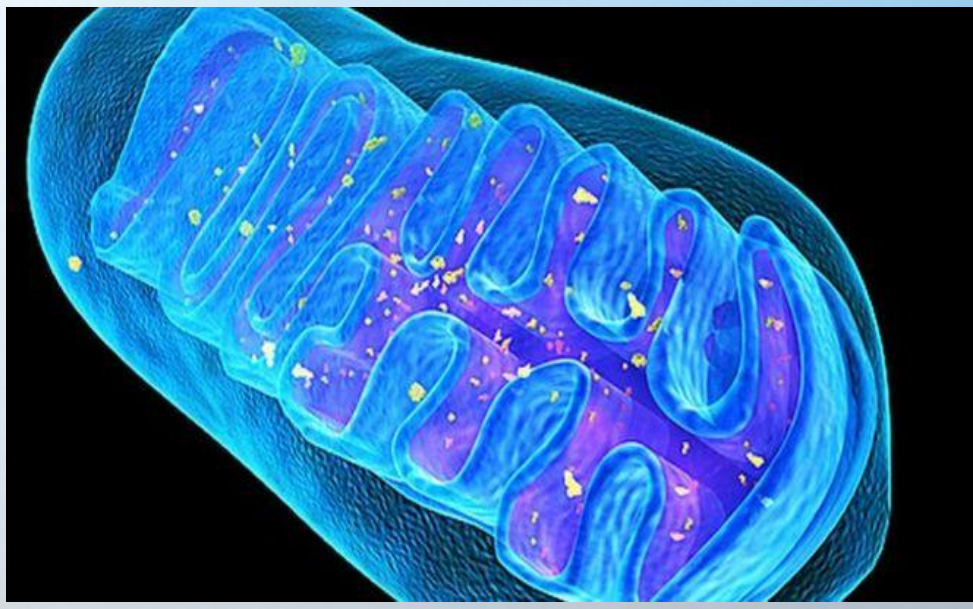
MIGHTY MITOCHONDRIA: THE MUSCLE'S FORGOTTEN POWERHOUSE

TONY BOUTAGY & CLAIRE NORGATE

FILEX 2017

SESSION FOCUS

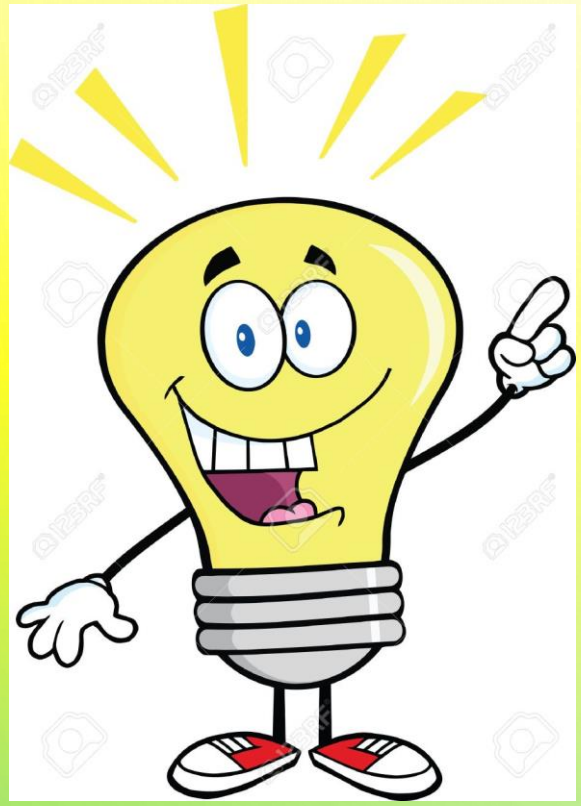
- Energy production
- What happens inside the cell?
- What are mitochondria?
- Physiology – a brief overview



- What regulates their growth?
- How does exercise affect mitochondria?
- Does nutrition impact mitochondria?
- Mitochondrial-boosting workouts

WHAT IS ENERGY?

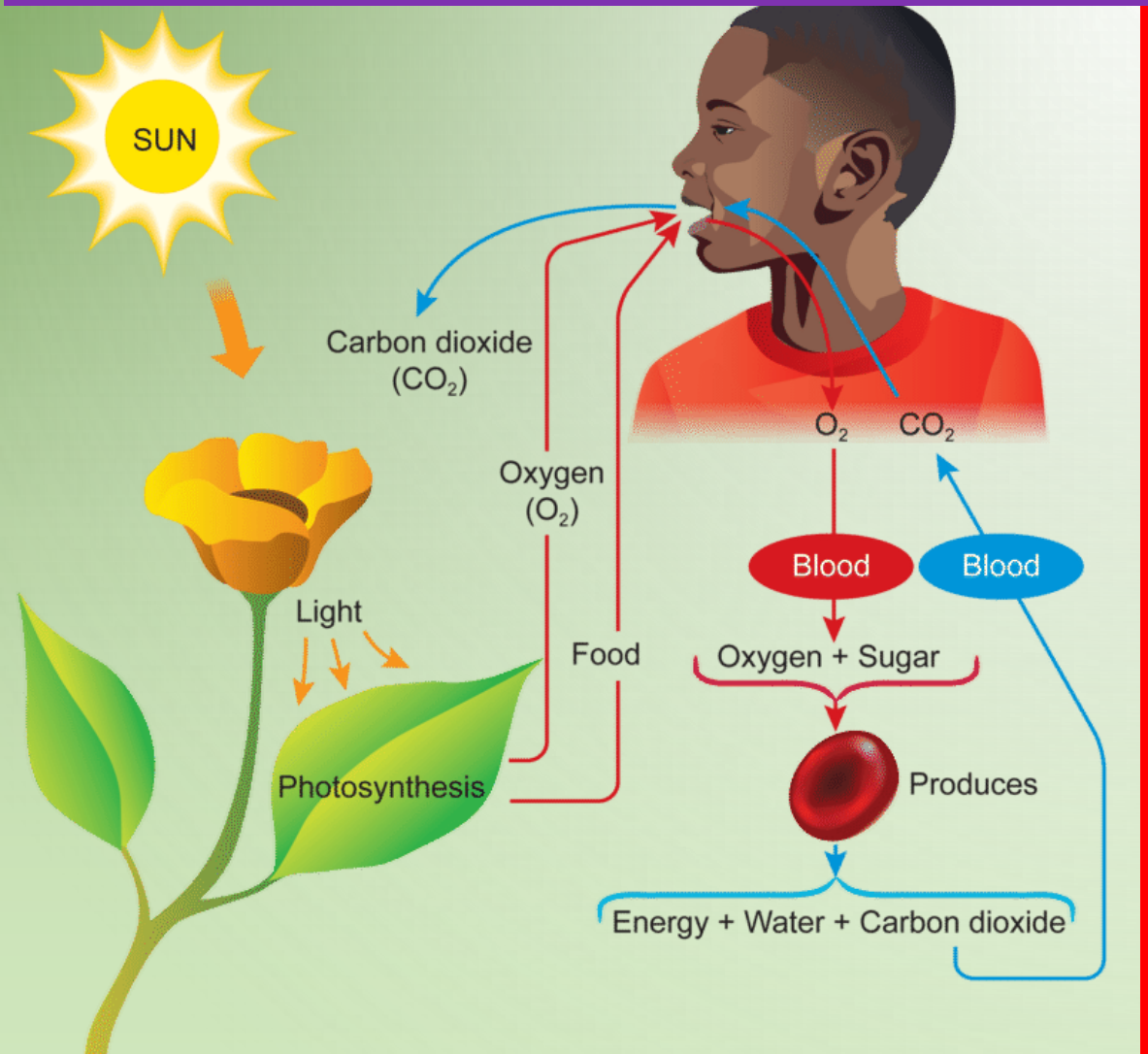
ENERGY IS THE ABILITY TO DO WORK



or . . . POWER DERIVED FROM THE UTILISATION OF PHYSICAL OR CHEMICAL RESOURCES

ENERGY PRODUCTION STARTS WITH

BREATHING & DIGESTING FOOD

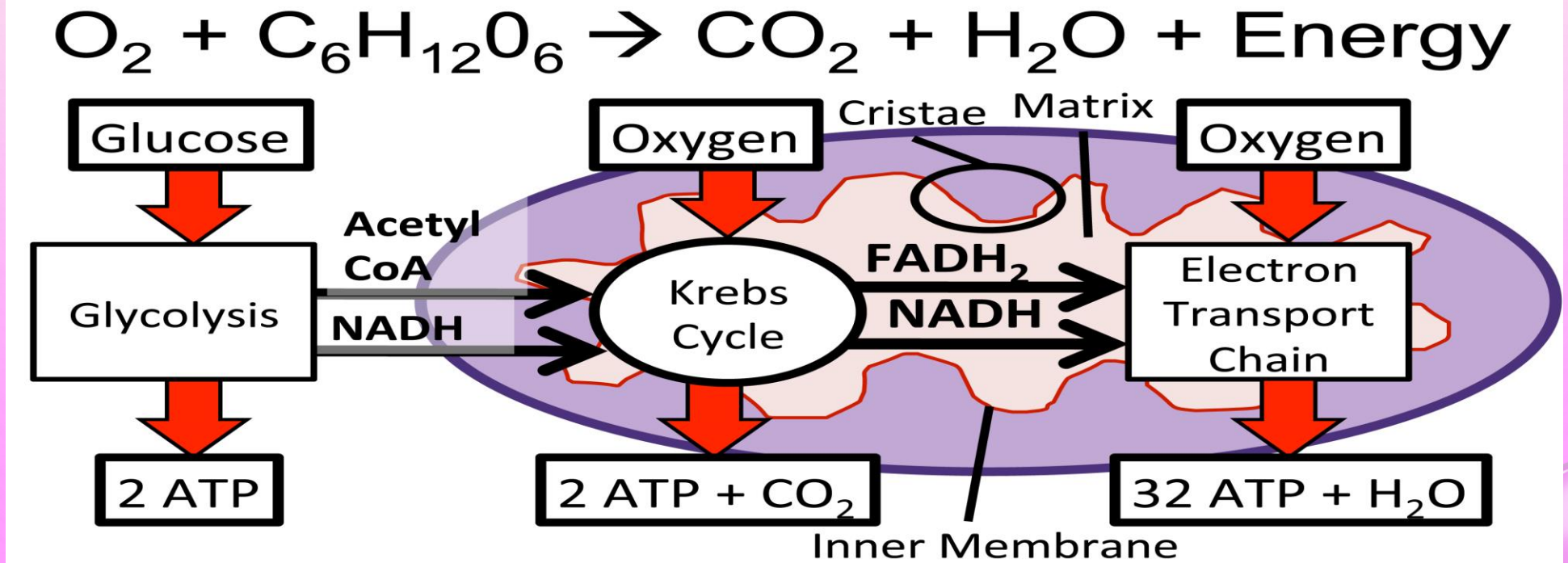


THIS PROVIDES OXYGEN AND GLUCOSE TO ACT AS FUEL

THIS FUEL IS USED IN OUR CELLS TO PRODUCE WORK ENERGY

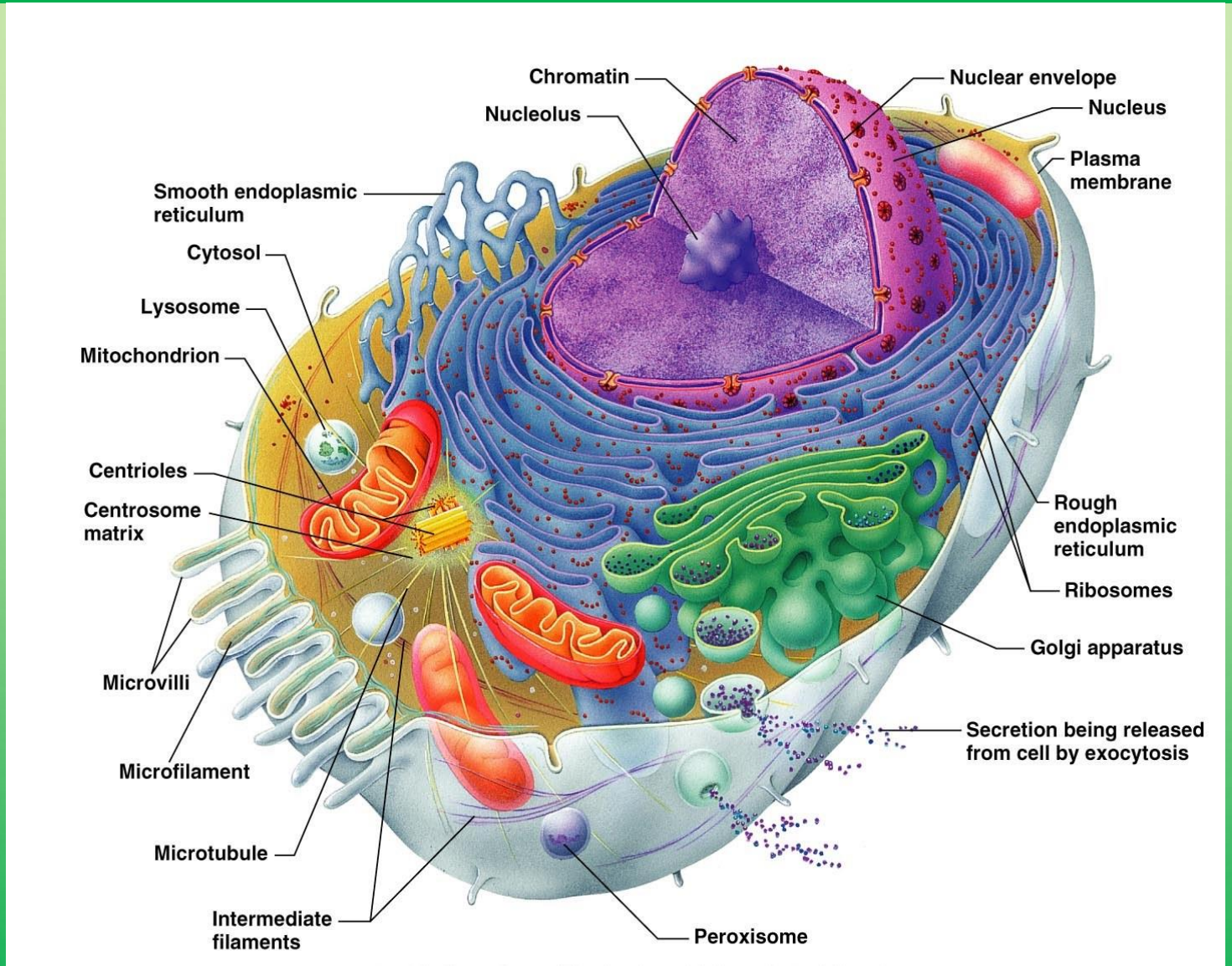
ENERGY PRODUCTION IN THE CELL IS CALLED **CELLULAR/INTERNAL RESPIRATION**

IT TAKES PLACE IN THE CYTOSOL & **MITOCHONDRIA** OF THE CELL



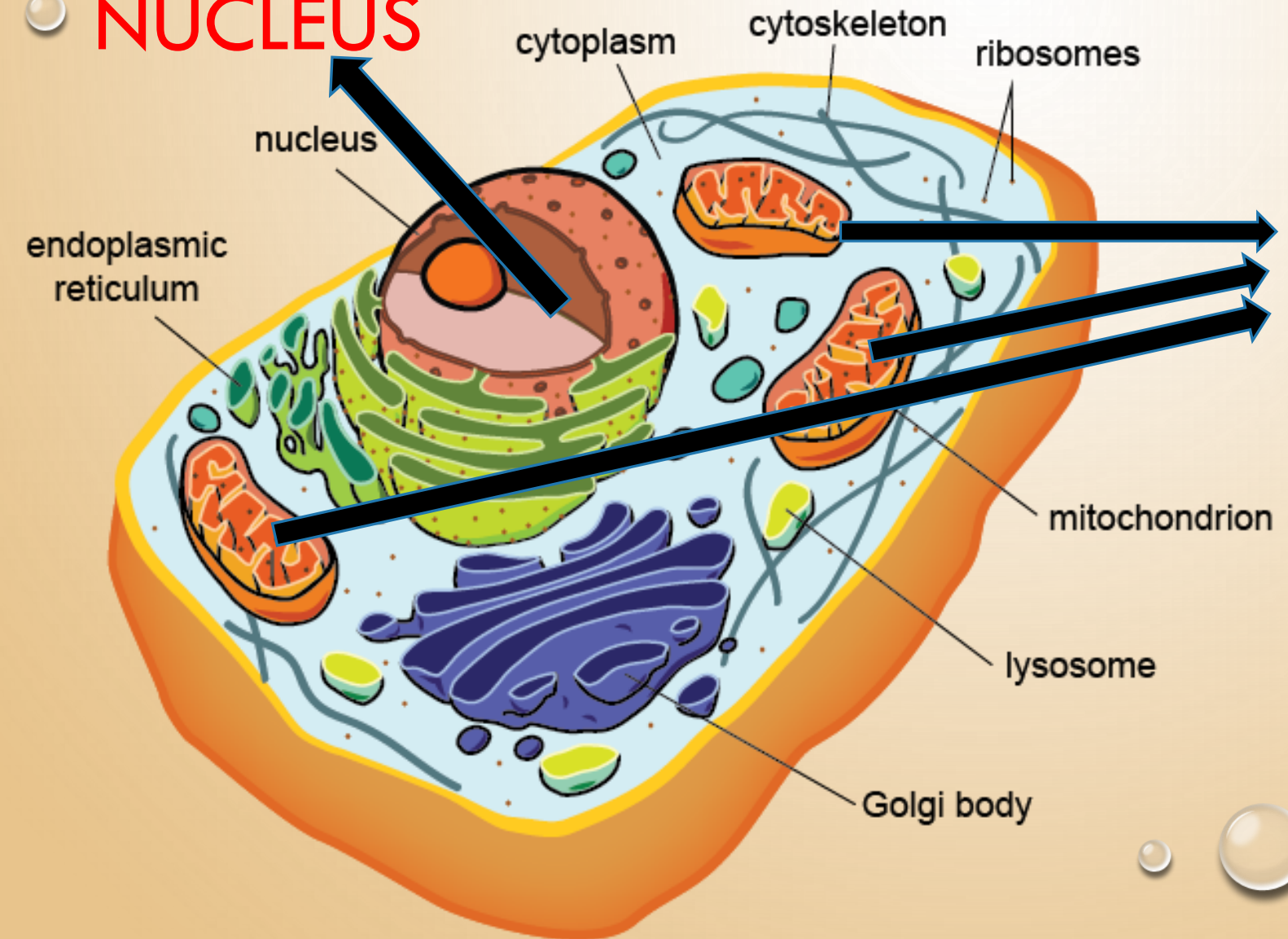
THE BASICS: Lots of mitochondria = lots of energy

Healthy mitochondria contribute to a healthy body



WHAT ARE MITOCHONDRIA?

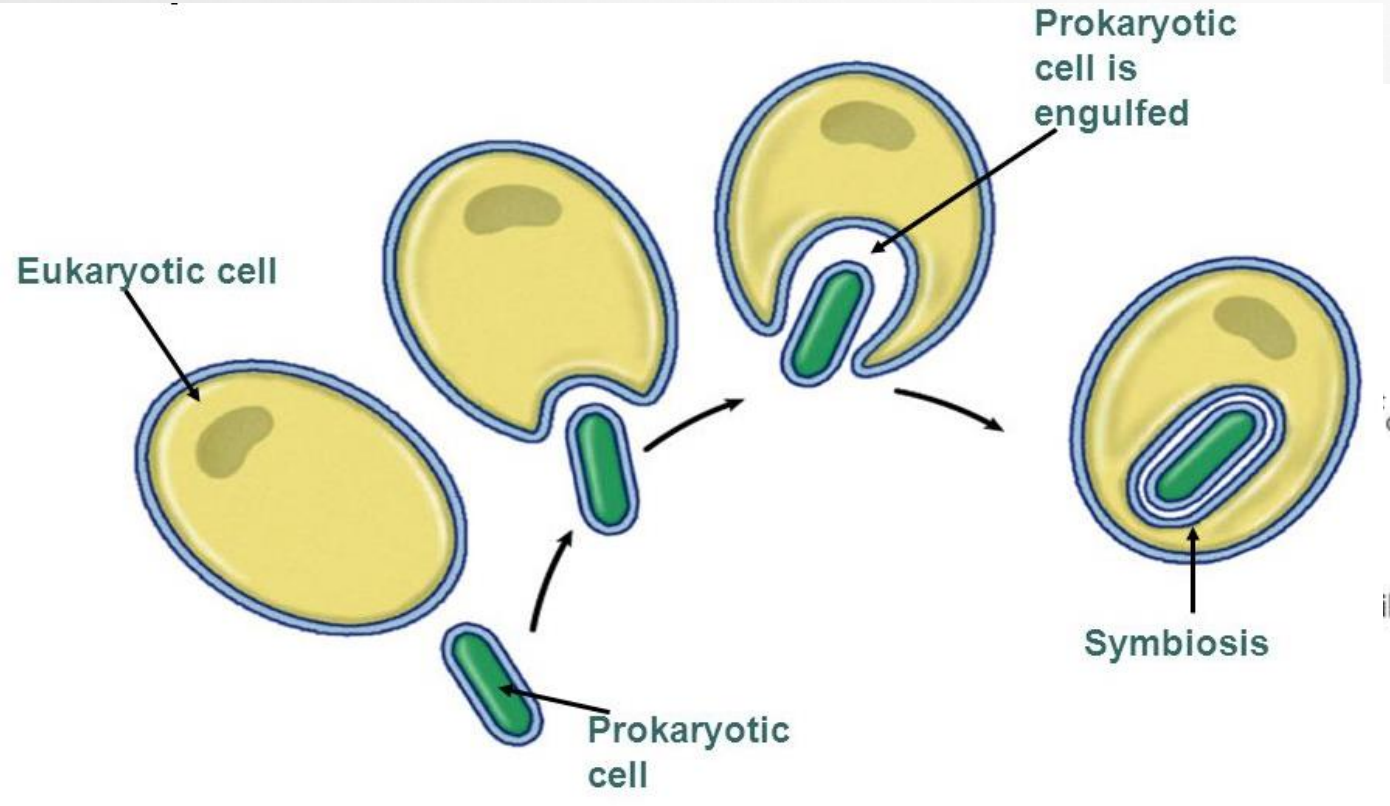
NUCLEUS

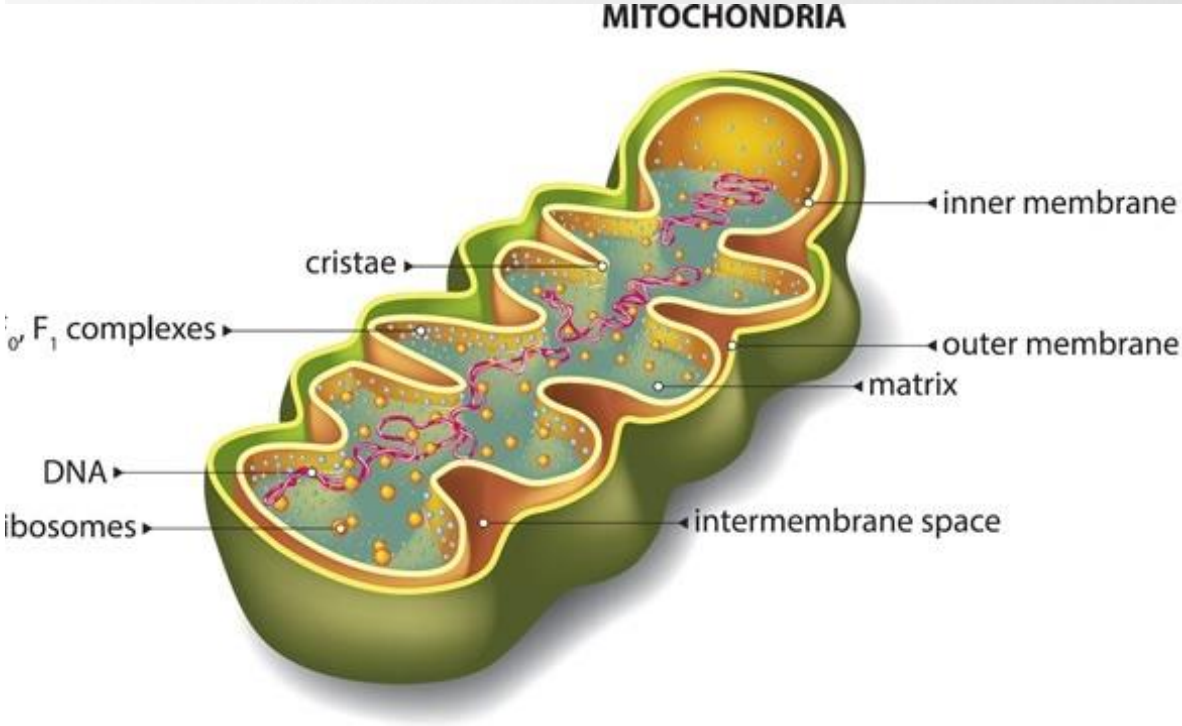


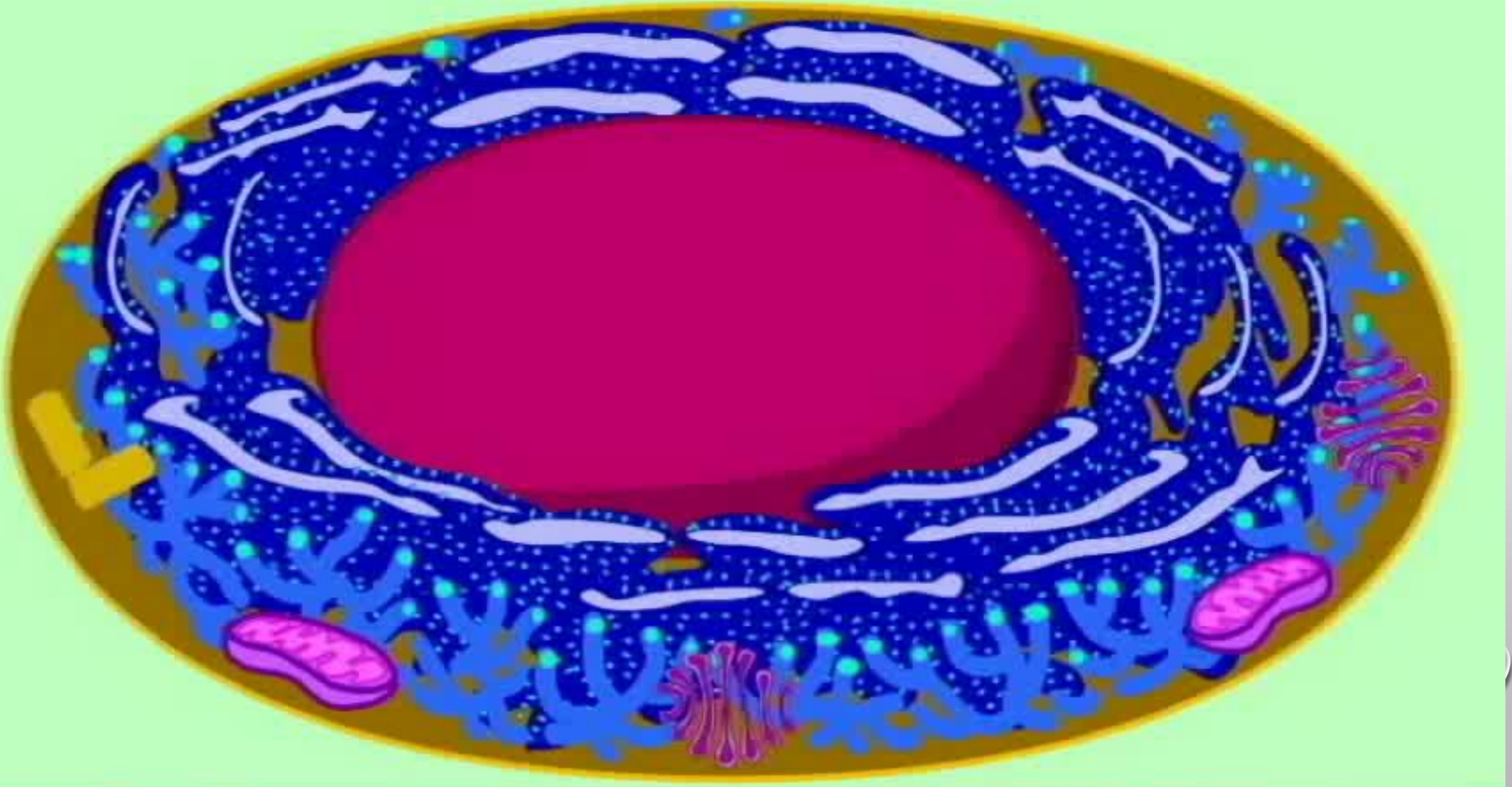
MITOCHONDRIA ARE ORGANELLES (OR PART OF A CELL)

A CLOSER LOOK

They are AMAZING and pre-date human life.
They're considered a prokaryotic cell (no nucleus)
Symbiotic relationship developed
The larger cell swallowed the smaller cell without digesting it!
https://www.youtube.com/watch?v=f2rX-nWDqoU

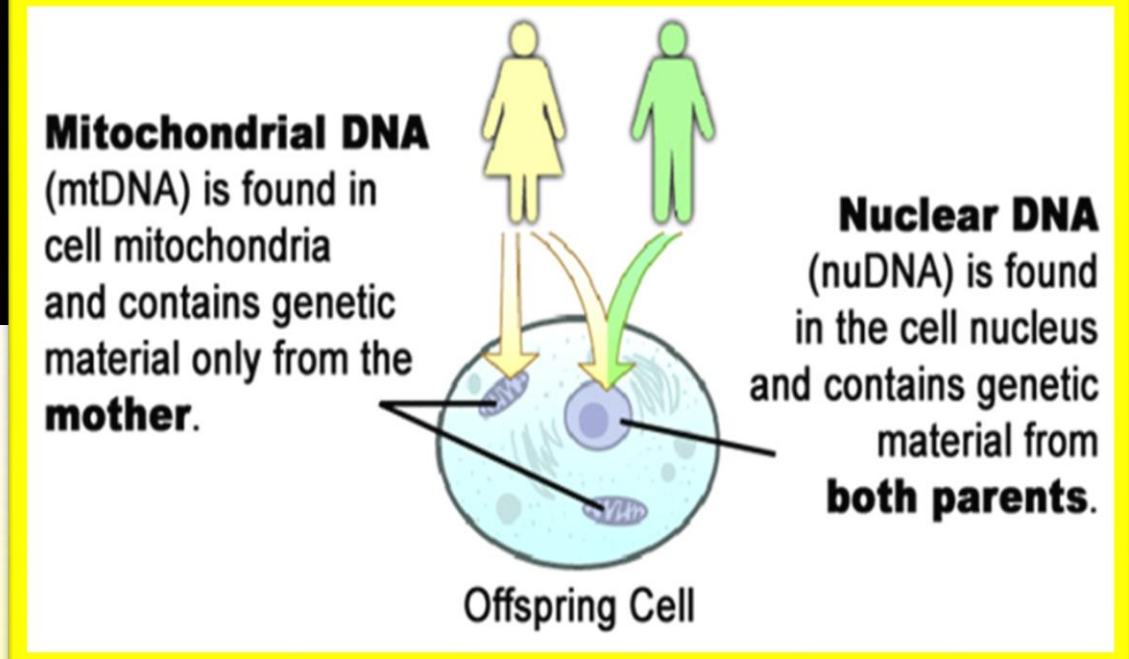


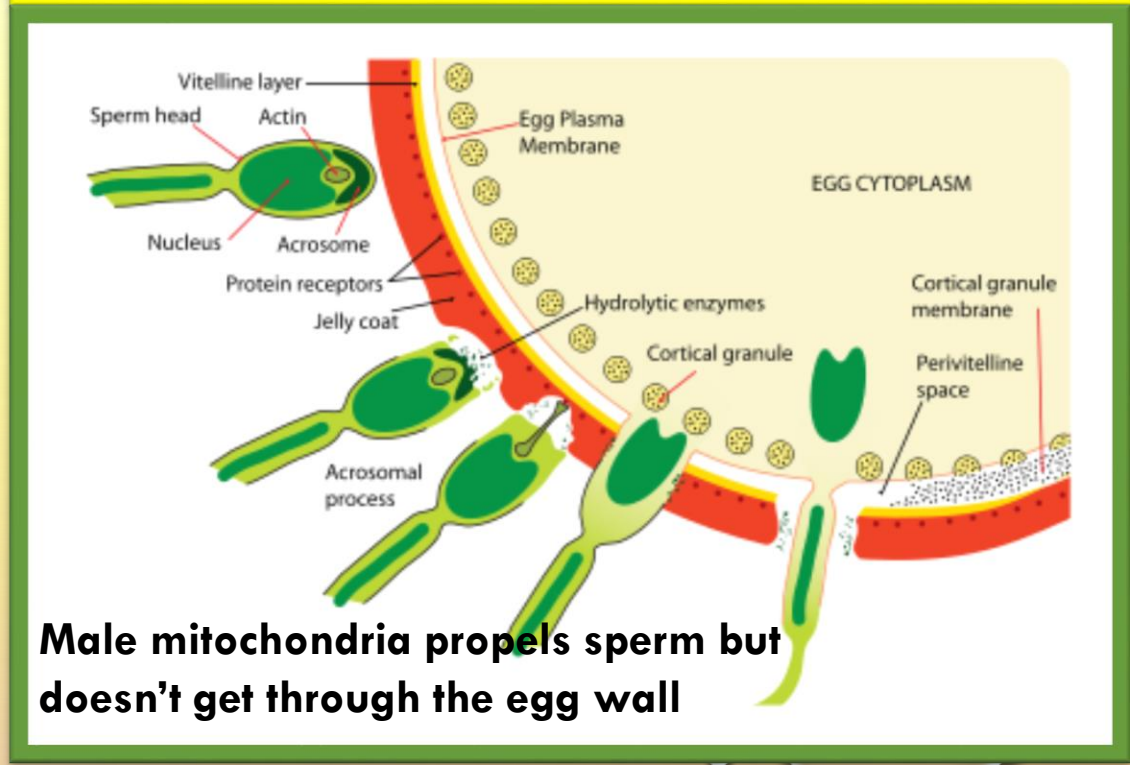




AMAZING MITOCHONDRIA

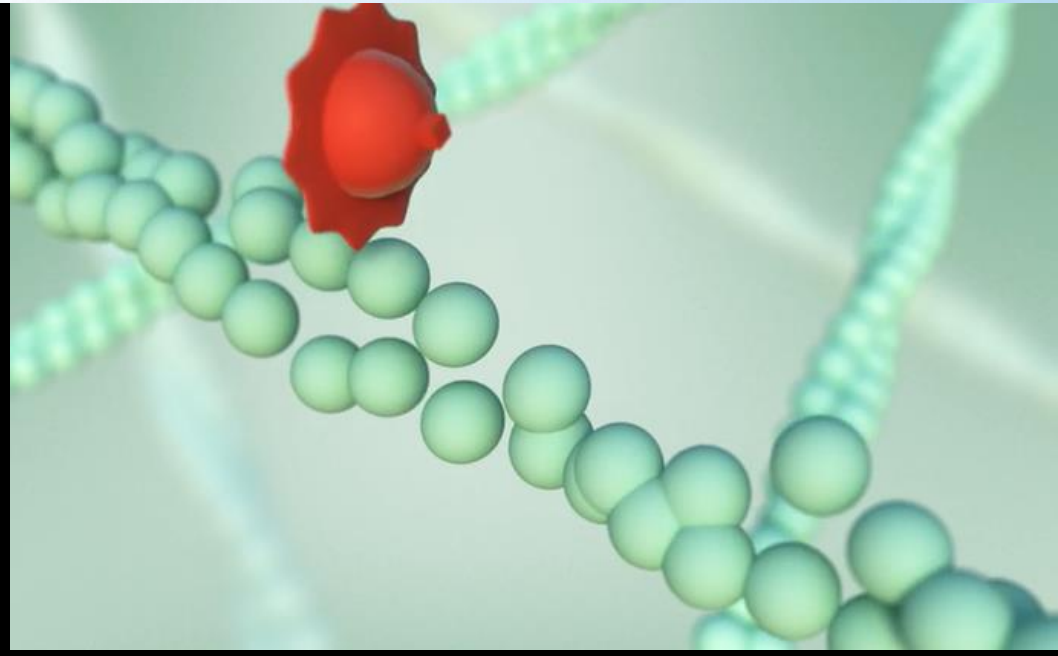
- HAVE THEIR OWN CHROMOSOMES
- HAVE THEIR OWN DNA
- MAKE THEIR OWN PROTEINS
- BEHAVE LIKE BACTERIA
- CAN BE DAMAGED BY ANTIBIOTICS
- ARE INHERITED FROM MATERNAL SIDE
- ARE IN MOST CELLS IN THE BODY
- IMMUNE FUNCTION
- PRODUCE ENERGY VIA AEROBIC METABOLISM





WHERE ARE THEY FOUND?: IN CELLS USING O2

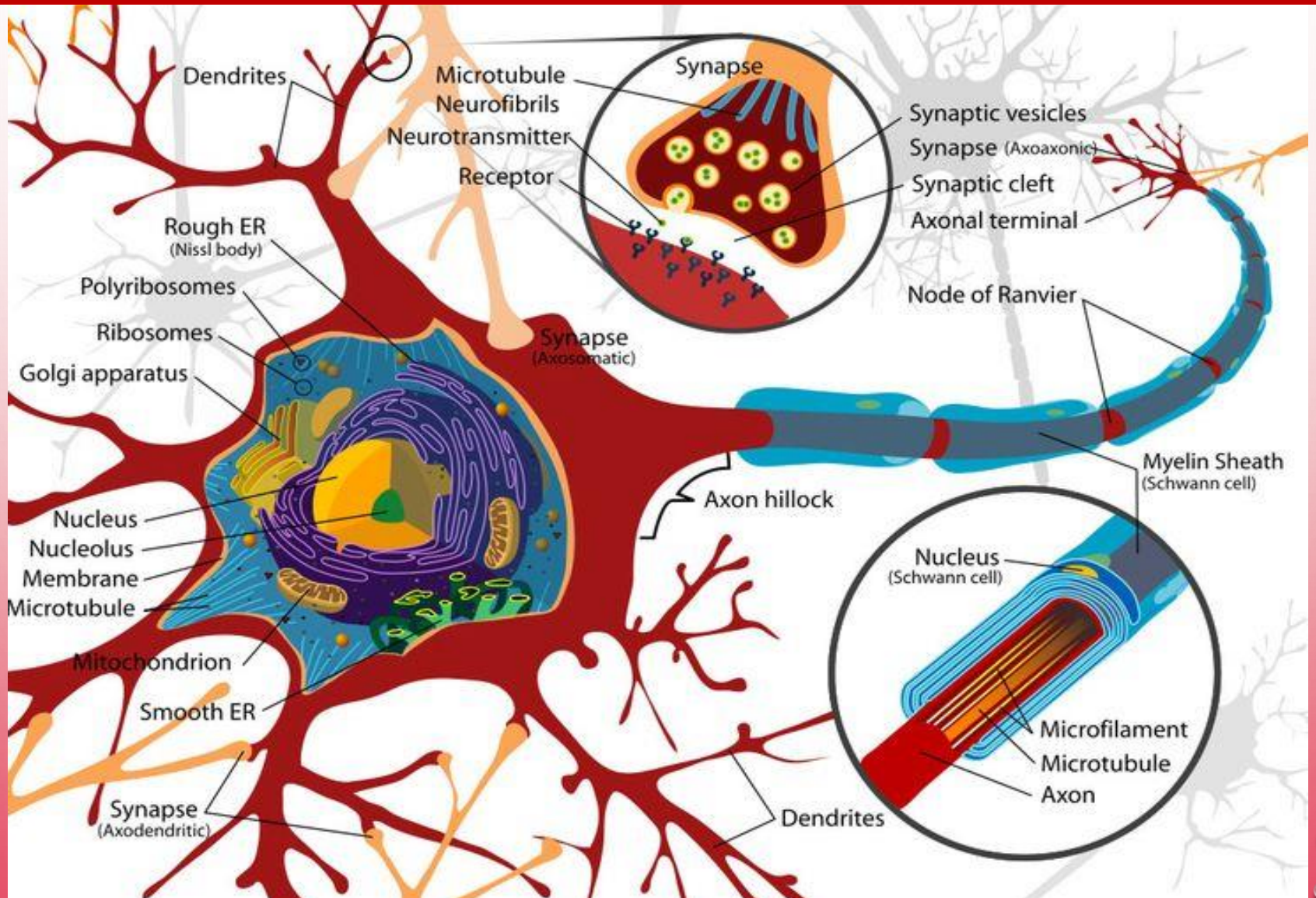
- IN MOST CELLS IN THE BODY
- NOT IN RBC
- LOTS IN LIVER, BRAIN AND MUSCLES
- THEY MULTIPLY ACCORDING TO DEMAND



MITOCHONDRIA PERFORM TWO FUNCTIONS:

1. THEY ARE THE PRIMARY SITES FOR ATP SYNTHESIS IN THE CELL – HENCE CALLED THE POWERHOUSE OF THE CELL
2. THEY HAVE A KEY ROLE IN APOPTOSIS – PROGRAMMED CELL DEATH – INTRINSIC PATHWAY – CANCER LINK

MITOCHONDRIA IN NERVE CELLS



MITOCHONDRIA IN MUSCLE FIBRES

RED MUSCLE

high mitochondrial content

MIXED MUSCLE

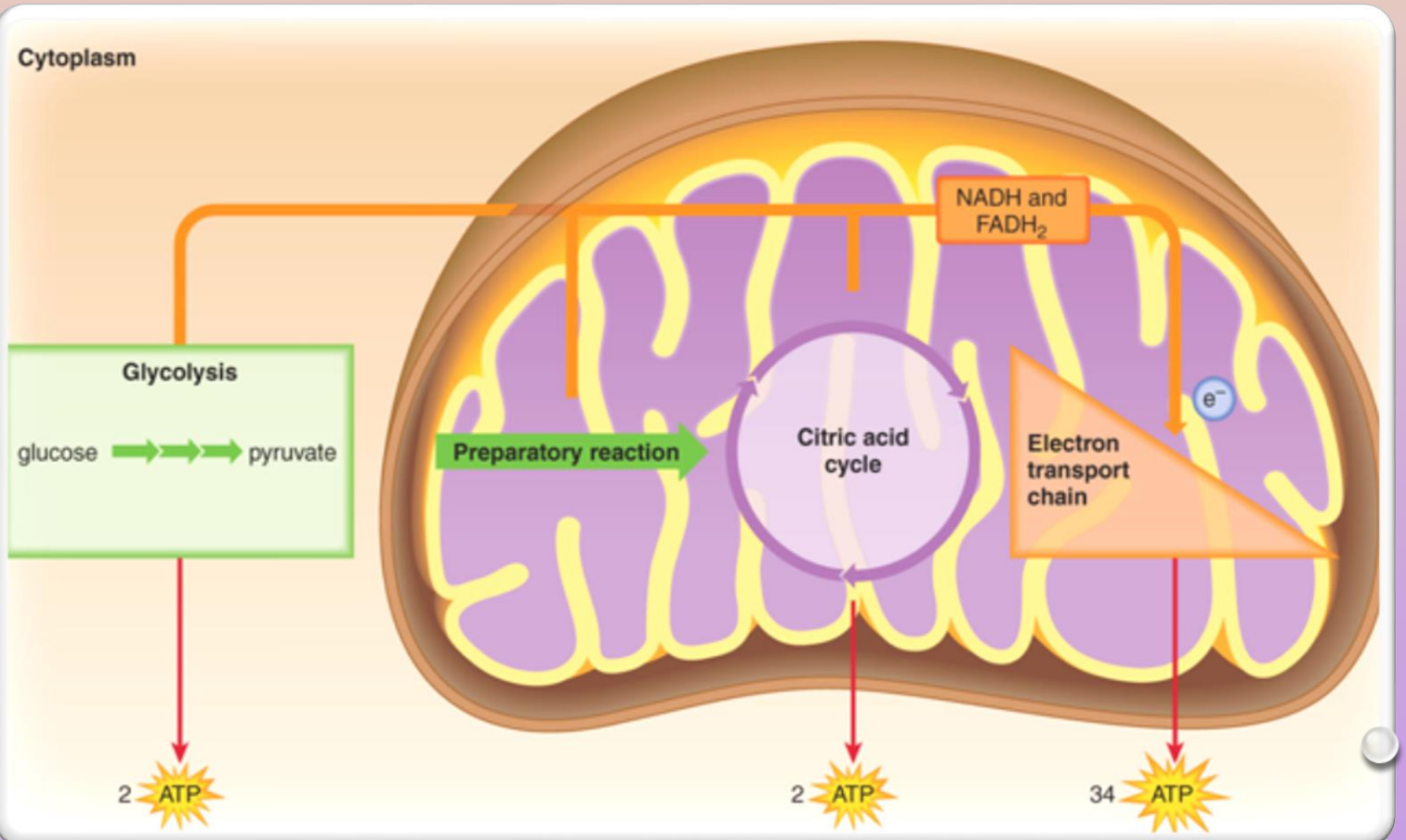
medium mitochondrial content

WHITE MUSCLE

low mitochondrial content

HOW DO CELLS MAKE ENERGY?

1. Glucose via the digestive system into cytosol
2. O2 via lungs into mitochondria together called CELLULAR RESPIRATION



CELLULAR RESPIRATION STEPS:

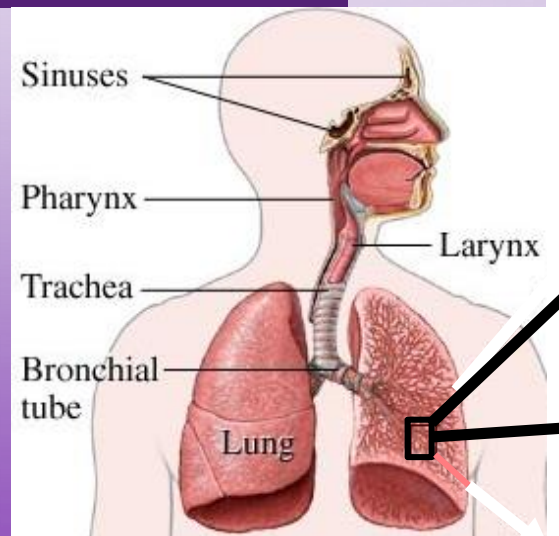
1. GLYCOLYSIS – IN CYTOSOL – **GLUCOSE** (FOOD)
2. KREBS CYCLE – IN MITOCHONDRIA USING **OXYGEN**
3. ELECTRON TRANSPORT CHAIN – IN MITOCHONDRIA USING **OXYGEN**

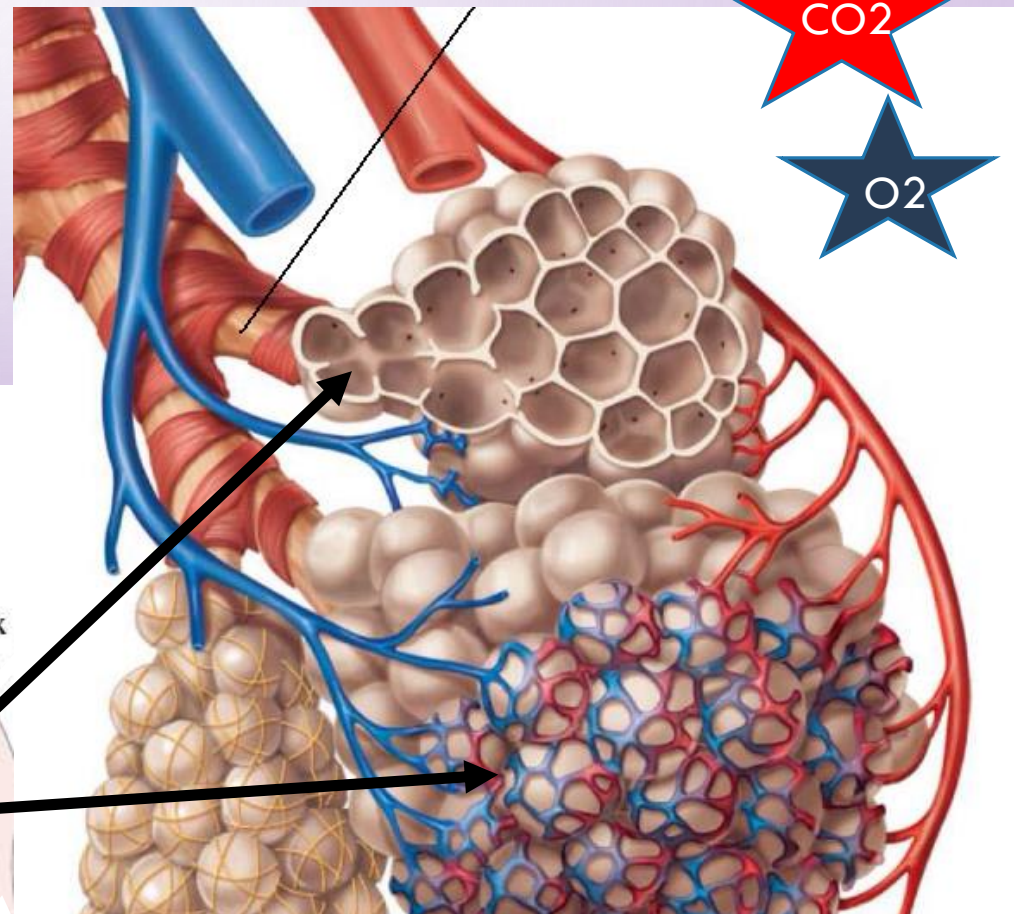
THE MITOCHONDRIA ENERGY CONNECTION

STEP 1

External respiration

O2 movement from environment to the blood





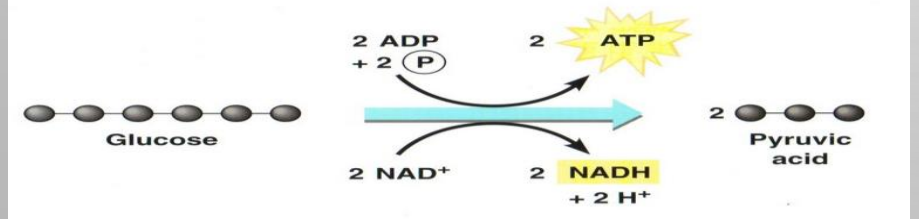
THE MITOCHONDRIA ENERGY CONNECTION

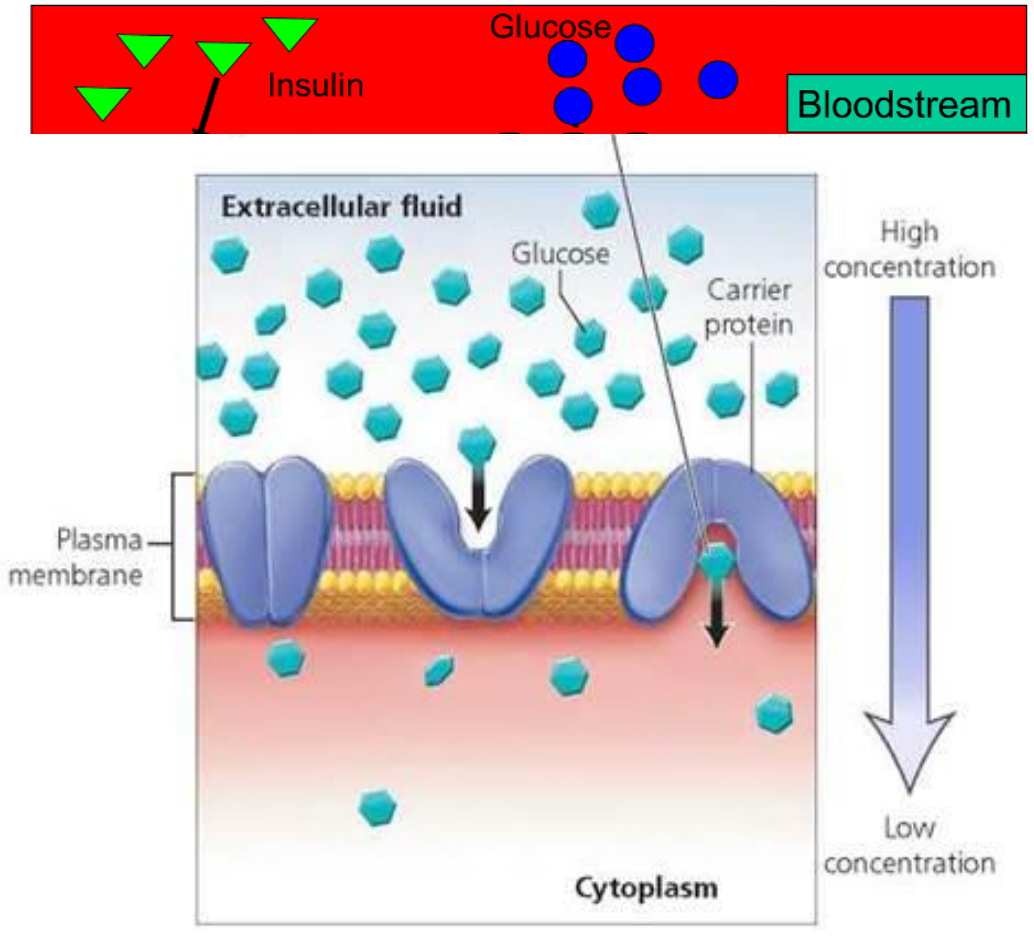
STEP 2

Cellular respiration

Movement of glucose from blood to cell cytosol

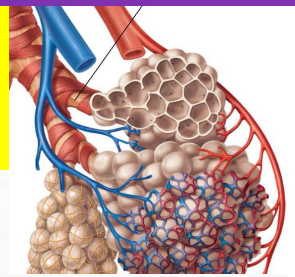
GLYCOLYSIS to feed the mitochondria





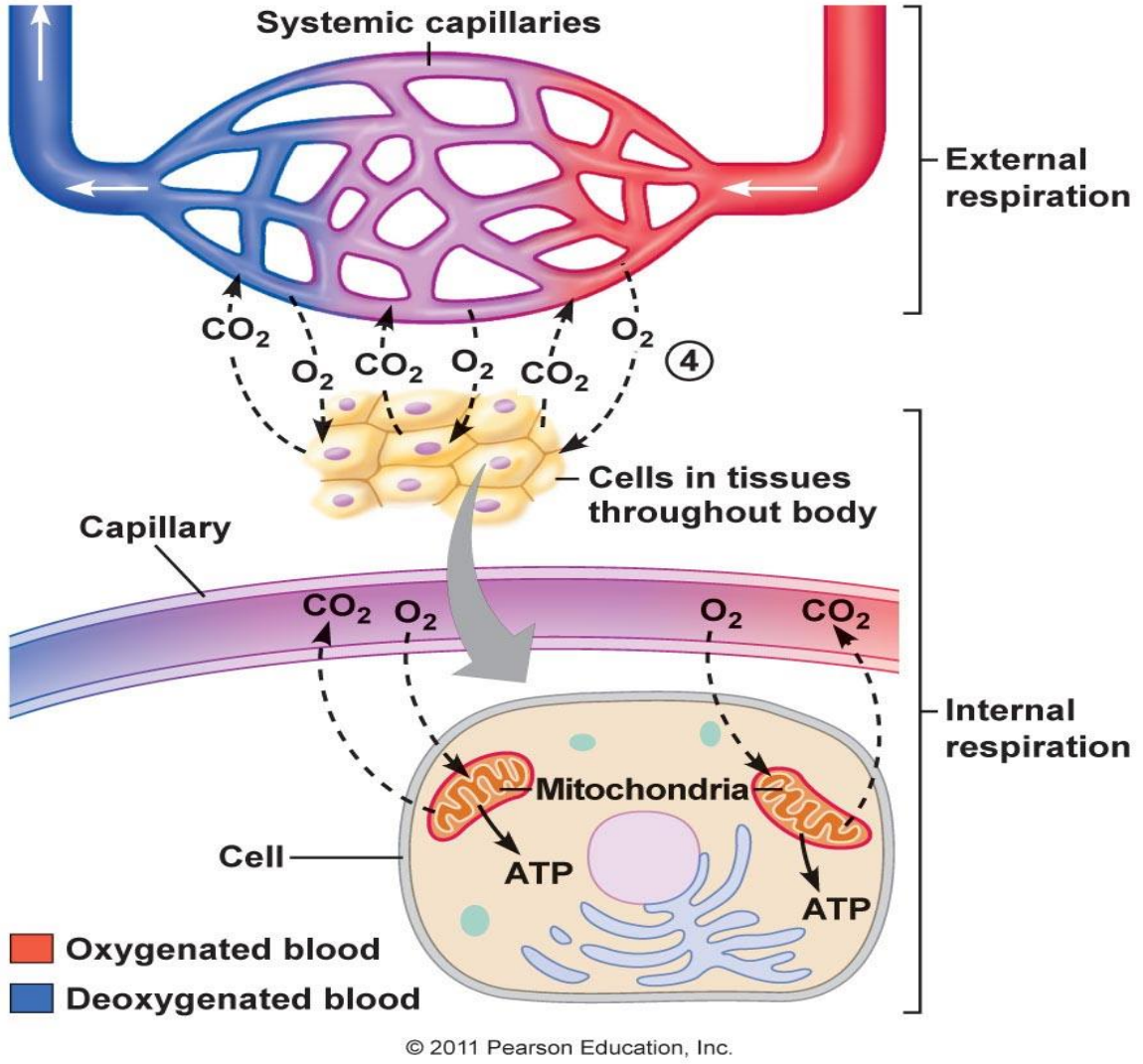
THE MITOCHONDRIA ENERGY CONNECTION

STEP 3



Cellular Respiration

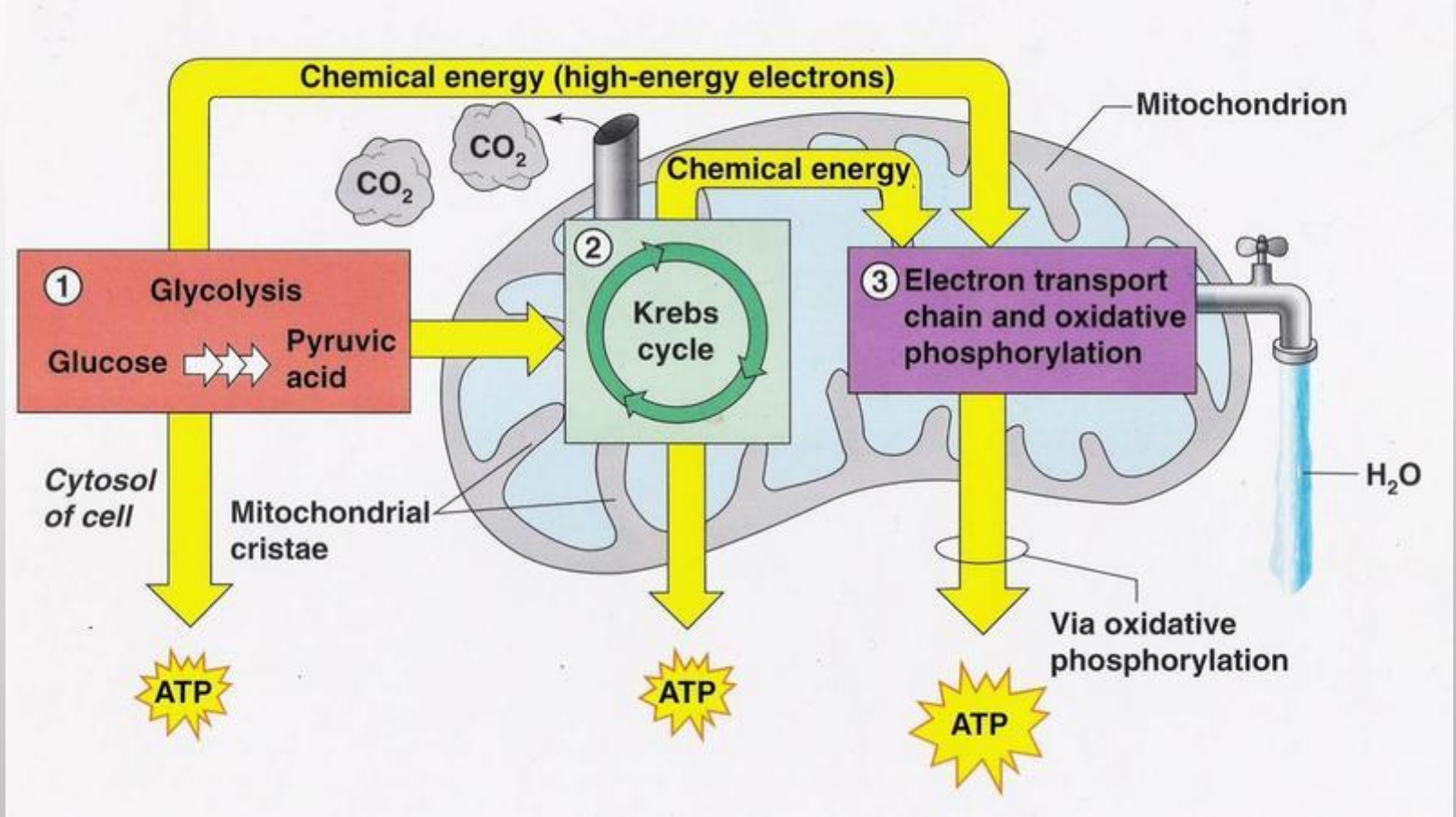
O2 movement from blood into the cell mitochondria in the body's tissues



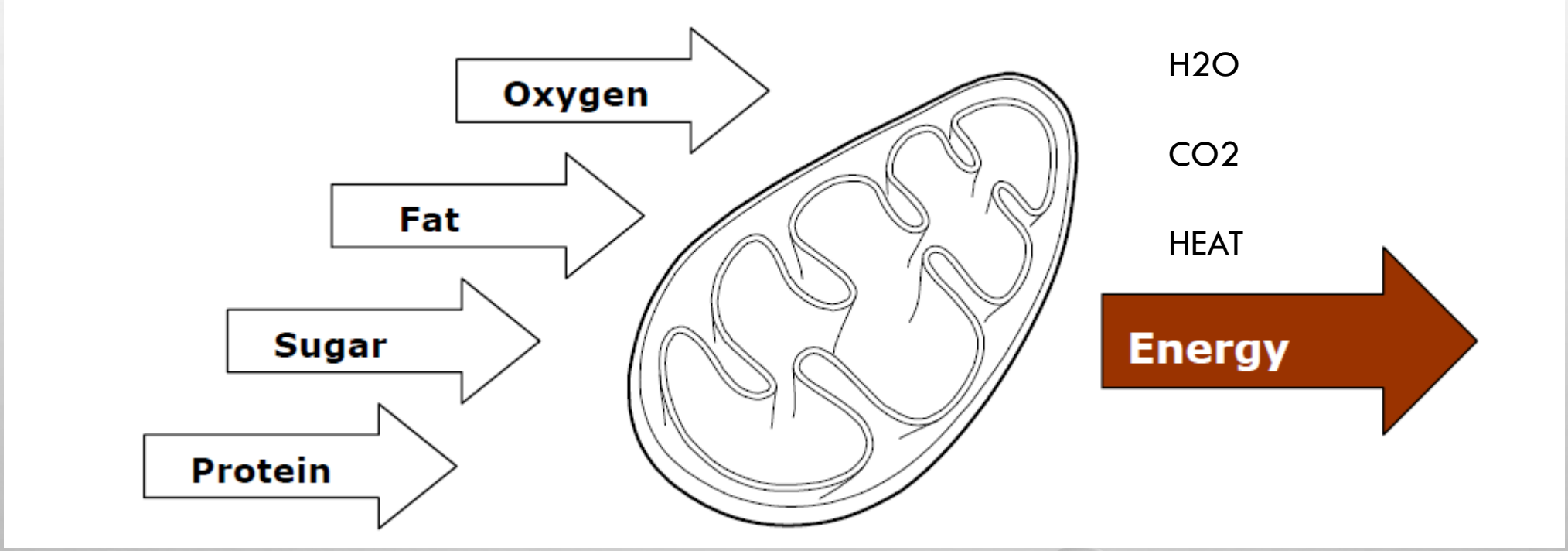
THE MITOCHONDRIA ENERGY CONNECTION

STEP 4

Cellular respiration using energy from glycolysis and oxygen to fuel mitochondria to create energy in form of ATP

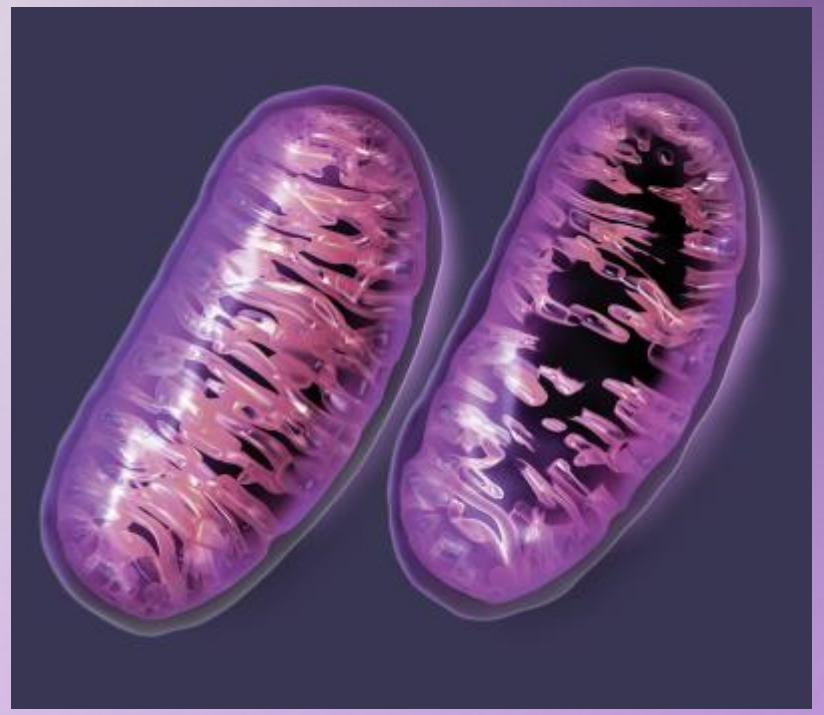


MITOCHONDRIA TRANSFORM ENERGETIC SUBSTRATES. EG, GLUCOSE AND OXYGEN INTO ENERGY

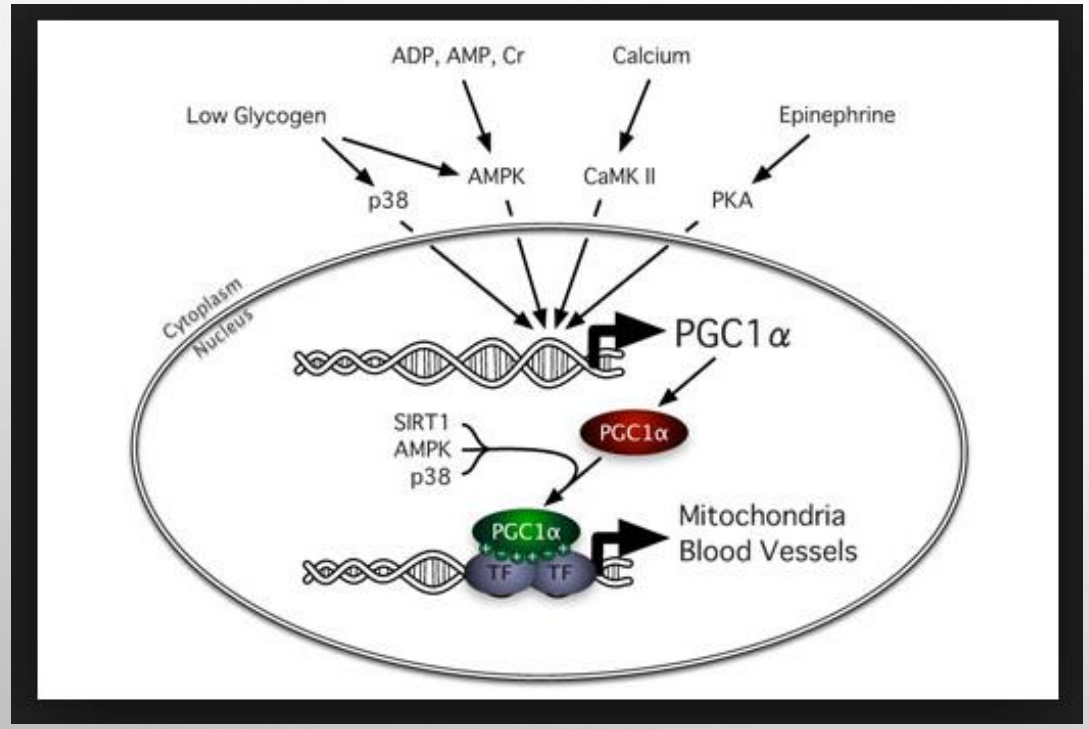


MITOCHONDRIAL DISEASE

- MITOCHONDRIAL DISEASES RANGE IN SEVERITY FROM ASYMPTOMATIC TO FATAL
- BECAUSE CELLS HAVE MULTIPLE MITOCHONDRIA, DIFFERENT MITOCHONDRIA IN THE SAME CELL CAN HAVE DIFFERENT VARIATIONS OF THE MTDNA
- WHEN A CERTAIN TISSUE REACHES A CERTAIN RATIO OF MUTANT VERSUS WILDTYPE MITOCHONDRIA, A DISEASE WILL PRESENT ITSELF.
- DIABETES MELLITUS, FORMS OF CANCER AND CARDIOVASCULAR DISEASE, LACTIC ACIDOSIS, SPECIFIC FORMS OF MYOPATHY, OSTEOPOROSIS, ALZHEIMER'S DISEASE, PARKINSONS'S DISEASE, STROKE, MALE INFERTILITY
- STATINS MAY BE LINKED TO MITOCHONDRIA DYSFUNCTION
- NUTRIENTS CAN PROTECT AGAINST OXIDATIVE DAMAGE TO MITOCHONDRIA. THESE NUTRIENTS INCLUDE Ω3 FATTY ACIDS, ANTIOXIDANTS (VITAMIN C AND ZINC), MEMBERS OF THE VITAMIN B FAMILY (VITAMIN B12 AND FOLIC ACID) AND MAGNESIUM.



WHAT REGULATES MITOCHONDRIAL GROWTH?



AMPK

↓

PGC-1A

↓

NRF 1/2

↓

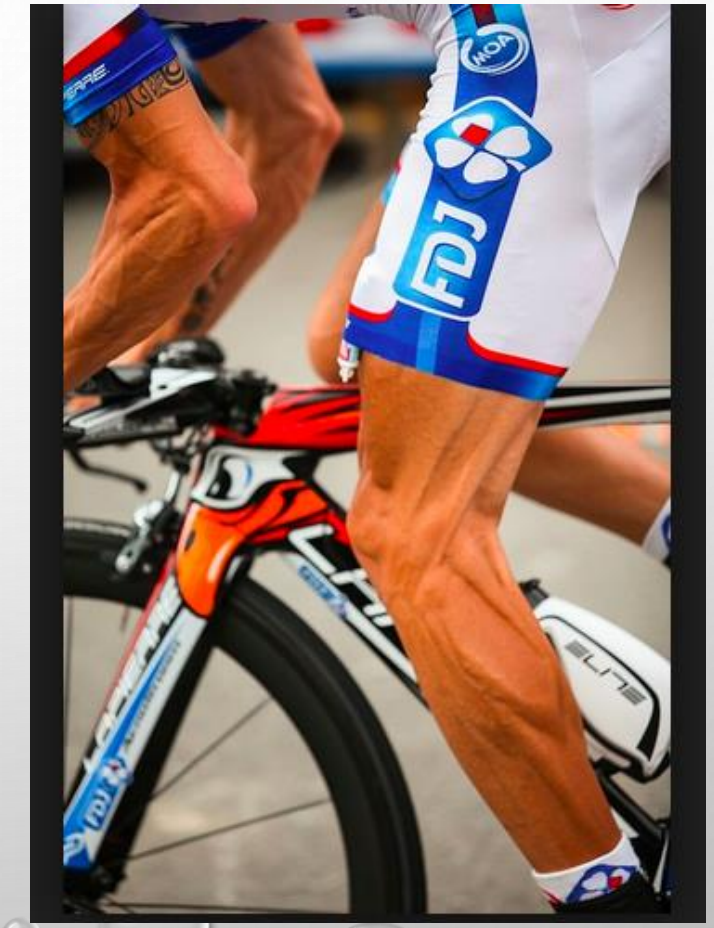
MITOCHONDRIA

CAN MITOCHONDRIA WASTE?

AMPK ACTIVITY HAS BEEN SHOWN TO DECREASE WITH AGE AND SEDENTARY LIFESTYLES, WHICH MAY CONTRIBUTE TO DECREASED MITOCHONDRIAL BIOGENESIS AND FUNCTION WITH AGING AND DISUSE.

HOW DOES EXERCISE EFFECT MITOCHONDRIA?

MUSCLE CONTRACTION INCREASES THE ACTIVITY OF AMPK (WHICH DETECTS FALLING ENERGY AVAILABILITY IN THE MUSCLE), WHICH INCREASES THE EXPRESSION OF PGC-1A & SIGNALS THE GROWTH & PROLIFERATION OF MITOCHONDRIA.



DOES ALL TYPES OF EXERCISE EFFECT MITOCHONDRIAL EQUALLY?

AEROBIC EXERCISE THAT EMPHASIZES VOLUME IS THE MOST POTENT CONTROLLER OF MITOCHONDRIAL GROWTH.

AEROBIC EXERCISE THAT EMPHASIZES INTENSITY REGULATES THE FUNCTION OF MITOCHONDRIA.

.

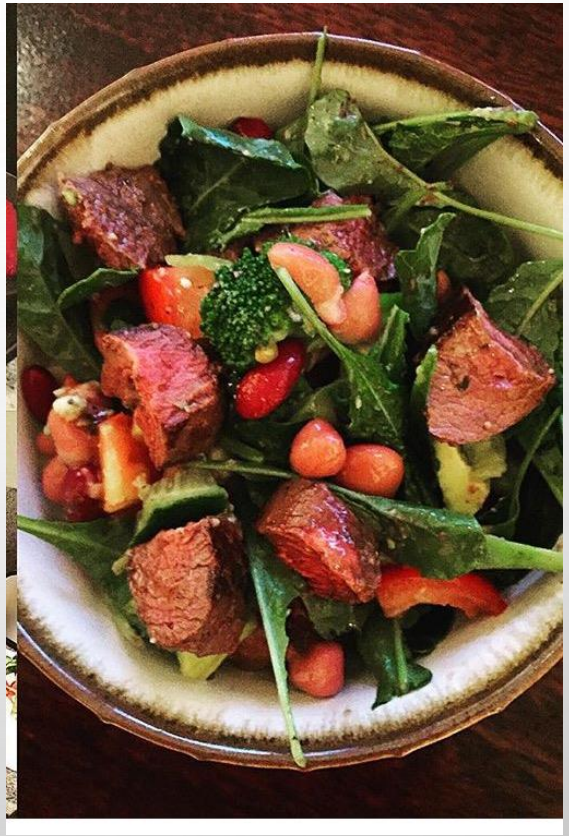
RESISTANCE TRAINING & MITOCHONDRIA?

MUCH LESS IS KNOWN ABOUT THE EFFECT OF LIFTING WEIGHTS ON MITOCHONDRIA.

AVAILABLE EVIDENCE SUGGESTS A ROLE AT INCREASING THE FUNCTION OF MITOCHONDRIA

HOW DOES NUTRITION IMPACT MITOCHONDRIA?

- MACRONUTRIENTS?
- FOOD COMPONENTS?
- TIMING OF MEALS IN THE DAY?
- TIMING OF NUTRITION IN RELATION TO EXERCISE?



HOW DO MACRONUTRIENTS IMPACT MITOCHONDRIA?

DIETS WITH LOW GLYCEMIC RESPONSE, HIGH FIBRE WITH MODERATE PROTEIN INTAKE AND HIGH PLANT VARIETY (MEDITERRANEAN DIET) HAVE BEEN SHOWN TO PROMOTE HEALTHY MITOCHONDRIA

FOOD COMPONENTS & MITOCHONDRIA?

GREEN TEA EXTRACTS,

SODIUM BICARBONATE,

CAFFEINE,

COCOA,

RESVERATROL,

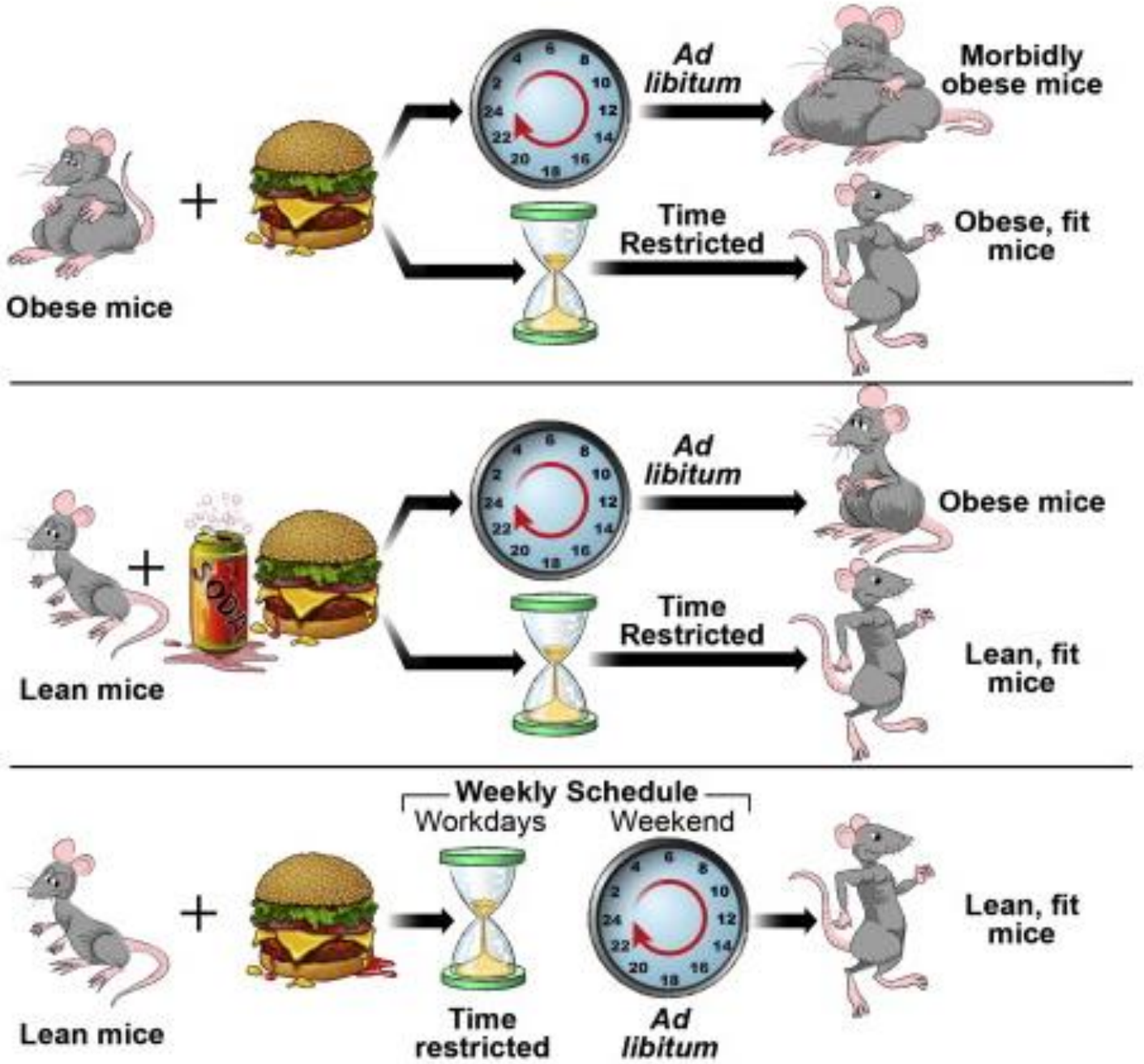
QUERCETIN.

TIMING OF MEALS & MITOCHONDRIA?

FASTING MIMICKING DIETS

KETOSIS

TIME-RESTRICTED FEEDING


Obese mice
Ad libitum
Morbidly obese mice
Time Restricted
Obese, fit mice
Lean mice
SODA
Ad libitum
Obese mice
Time Restricted
Lean, fit mice
Weekly Schedule
Workdays
Weekend
Lean mice
Time restricted
Ad libitum
Lean, fit mice

SUPPLEMENTS & MITOCHONDRIA

CREATINE, L-CARNITINE, A-LIPOIC ACID, COENZYME Q_{10}, REDUCED NICOTINAMIDE ADENINE DINUCLEOTIDE (NADH).

SUPPLEMENTS CONTAINING NICOTINAMIDE RIBOSIDE APPEAR TO HAVE THE MOST LIKELIHOOD AT HAVING A POSITIVE EFFECT ON MITOCHONDRIA

NUTRIENT TIMING INTERACTIONS WITH MITOCHONDRIAL

PERFORMING EXERCISE OR RECOVERING FROM EXERCISE WITHOUT THE PROVISION OF CARBOHYDRATE HAS A FAVOURABLE EFFECT OF MITOCHONDRIAL ADAPTATION.

Sports Med (2017) 47 (Suppl 1):S51–S63
DOI 10.1007/s40279-017-0694-2

CrossMark

REVIEW ARTICLE

Periodized Nutrition for Athletes

Asker E Jeukendrup[1]

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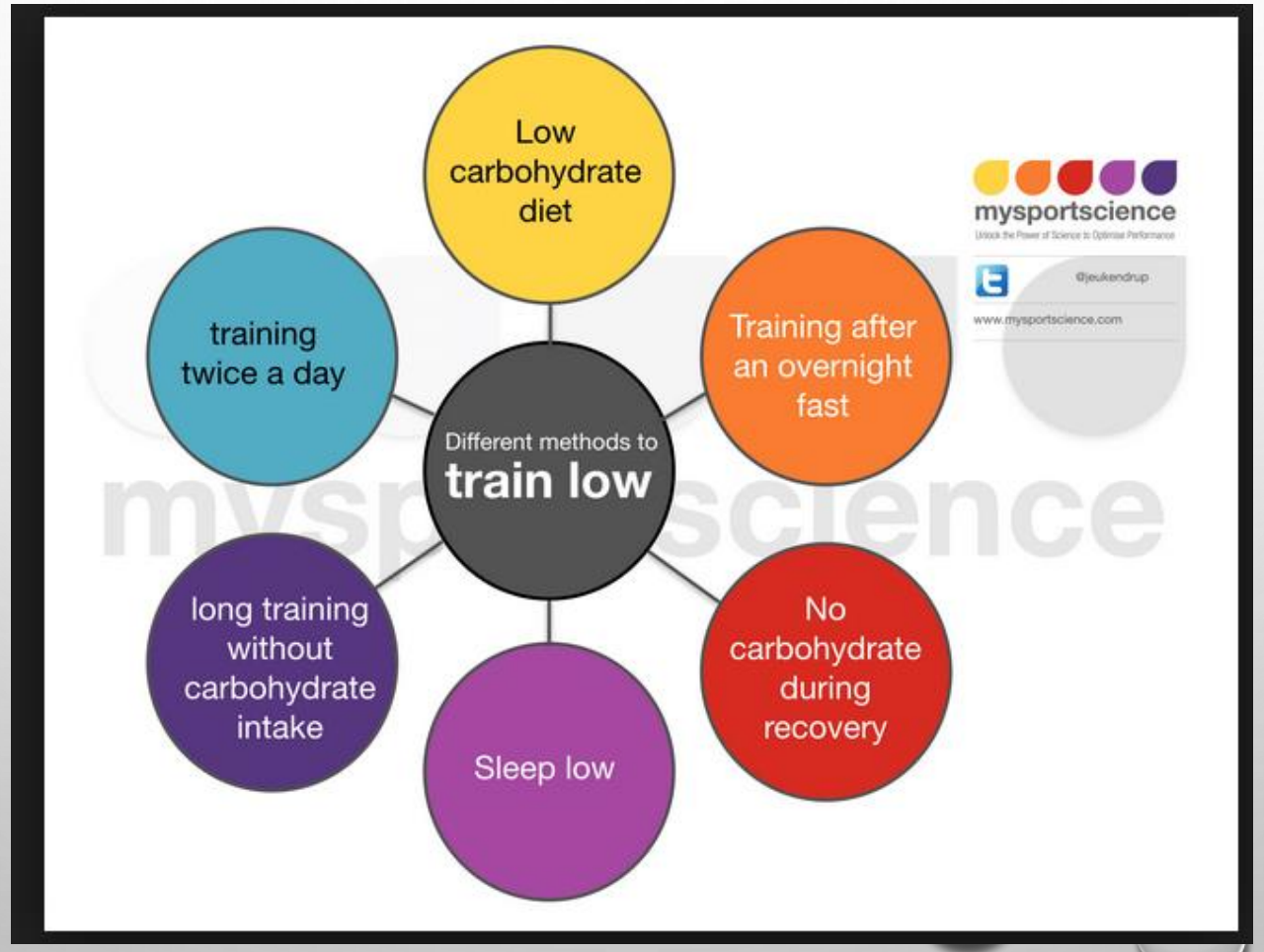
Abstract It is becoming increasingly clear that adaptations, initiated by exercise, can be amplified or reduced by nutrition. Various methods have been discussed to optimize training adaptations and some of these methods have been subject to extensive study. To date, most methods have focused on skeletal muscle, but it is important to note that training effects also include adaptations in other tissues (e.g., brain, vasculature), improvements in the absorptive capacity of the intestine, increases in tolerance to dehydration, and other effects that have received less attention in the literature. The purpose of this review is to define the concept of periodized nutrition (also referred to as nutritional training) and summarize the wide variety of methods available to athletes. The reader is referred to several other recent review articles that have discussed aspects of periodized nutrition in much more detail with primarily a focus on adaptations in the muscle. The purpose of this review is not to discuss the literature in great detail but to clearly define the concept and to give a complete overview of the methods available, with an emphasis on adaptations that are not in the muscle. Whilst there is good evidence for some methods, other proposed methods are mere theories that remain to be tested. 'Periodized nutrition' refers to the strategic combined use of exercise training and nutrition, or nutrition only, with the overall aim to obtain adaptations that support exercise performance. The term nutritional training is sometimes used to describe the same methods and these terms can be used interchangeably. In this review, an overview is given of some of the most common methods of periodized nutrition including 'training low' and 'training high', and training with low- and high-carbohydrate availability, respectively. 'Training low' in particular has received considerable attention and several variations of 'train low' have been proposed. 'Training-low' studies have generally shown beneficial effects in terms of signaling and transcription, but to date, few studies have been able to show any effects on performance. In addition to 'train low' and 'train high', methods have been developed to 'train the gut', train hypohydrated (to reduce the negative effects of dehydration), and train with various supplements that may increase the training adaptations longer term. Which of these methods should be used depends on the specific goals of the individual and there is no method (or diet) that will address all needs of an individual in all situations. Therefore, appropriate practical application lies in the optimal combination of different nutritional training methods. Some of these methods have already found their way into training practices of athletes, even though evidence for their efficacy is sometimes scarce at best. Many pragmatic questions remain unanswered and another goal of this review is to identify some of the remaining questions that may have great practical relevance and should be the focus of future research.

1 Introduction

NUTRIENT TIMING INTERACTIONS WITH MITOCHONDRIAL



HIT VS CONTINUOUS EXERCISE?

BOTH INTENSITY & VOLUME APPEAR TO EFFECT MITOCHONDRIA IN DIFFERENT WAYS

(FUNCTION VS GROWTH)

HIT VS CONTINUOUS EXERCISE?

WHEN MATCHED FOR TOTAL VOLUME,

HIT APPEARS TO HAVE A SUPERIOR EFFECT.

Format: Abstract Send to

J Physiol. 2017 May 1;595(9):2955-2968. doi: 10.1113/JP272570. Epub 2016 Aug 3.

Superior mitochondrial adaptations in human skeletal muscle after interval compared to continuous single-leg cycling matched for total work.

MacInnis MJ[1], Zacharewicz E[2], Martin BJ[1], Haikalis ME[3], Skelly LE[1], Tarnopolsky MA[3], Murphy RM[2], Gibala MJ[1].

Author information

Abstract

KEY POINTS: A classic unresolved issue in human integrative physiology involves the role of exercise intensity, duration and volume in regulating skeletal muscle adaptations to training. We employed counterweighted single-leg cycling as a unique within-subject model to investigate the role of exercise intensity in promoting training-induced increases in skeletal muscle mitochondrial content. Six sessions of high-intensity interval training performed over 2 weeks elicited greater increases in citrate synthase maximal activity and mitochondrial respiration compared to moderate-intensity continuous training matched for total work and session duration. These data suggest that exercise intensity, and/or the pattern of contraction, is an important determinant of exercise-induced skeletal muscle remodelling in humans.

ABSTRACT: We employed counterweighted single-leg cycling as a unique model to investigate the role of exercise intensity in human skeletal muscle remodelling. Ten young active men performed unilateral graded-exercise tests to measure single-leg $\dot{V}O2$, peak and peak power (W_{peak}). Each leg was randomly assigned to complete six sessions of high-intensity interval training (HIIT) [4 × (5 min at 65% W_{peak} and 2.5 min at 20% W_{peak})] or moderate-intensity continuous training (MICT) (30 min at 50% W_{peak}), which were performed 10 min apart on each day, in an alternating order. The work performed per session was matched for MICT (143 ± 8.4 kJ) and HIIT (144 ± 8.5 kJ, $P > 0.05$). Post-training, citrate synthase (CS) maximal activity (10.2 ± 0.8 vs. 8.4 ± 0.9 mmol kg protein^{-1} min^{-1}) and mass-specific [pmol O_2 •(s•mg wet weight)$^{-1}$] oxidative phosphorylation capacities (complex I: 23.4 ± 3.2 vs. 17.1 ± 2.8; complexes I and II: 58.2 ± 7.5 vs. 42.2 ± 5.3) were greater in HIIT relative to MICT (interaction effects, $P < 0.05$); however, mitochondrial function [i.e. pmol O_2 •(s•CS maximal activity)$^{-1}$] measured under various conditions was unaffected by training ($P > 0.05$). In whole muscle, the protein content of COXIV (24%), NDUFA9 (11%) and mitofusin 2 (MFN2) (16%) increased similarly across groups (training effects, $P < 0.05$). Cytochrome c oxidase subunit IV (COXIV) and NADH:ubiquinone oxidoreductase subunit A9 (NDUFA9) were more abundant in type I than type II fibres ($P < 0.05$) but training did not increase the content of COXIV, NDUFA9 or MFN2 in either fibre type ($P > 0.05$). Single-leg $\dot{V}O2$, peak was also unaffected by training ($P > 0.05$). In summary, single-leg cycling performed in an interval compared to a continuous manner elicited superior mitochondrial adaptations in human skeletal muscle despite equal total work.

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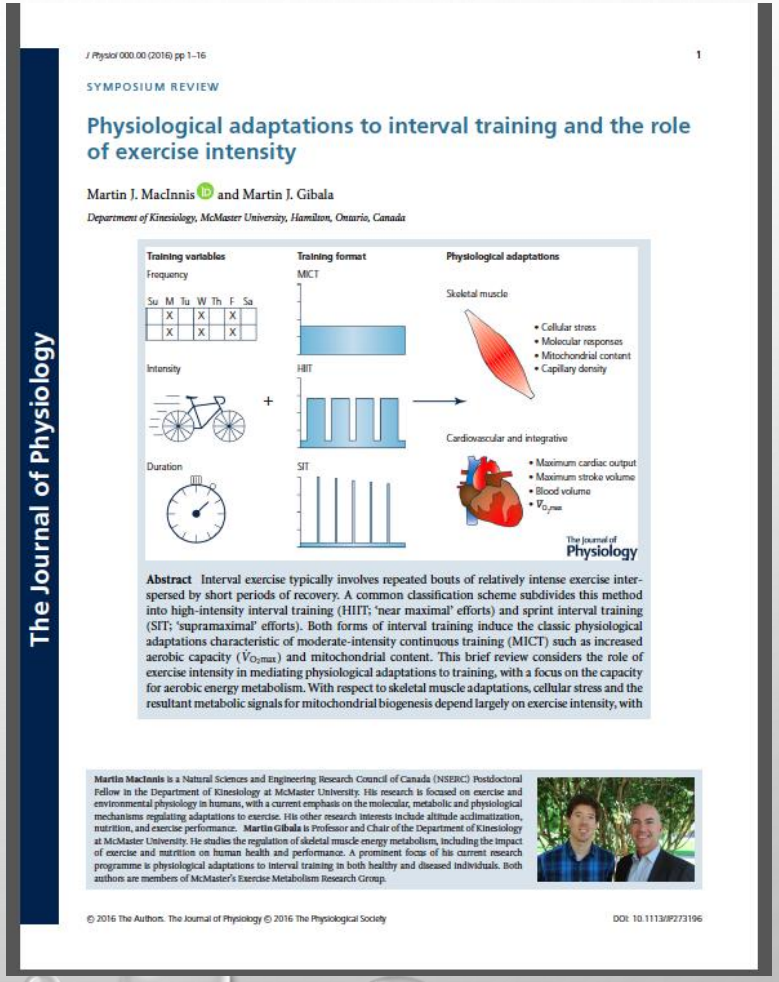
KEYWORDS: exercise intensity; high-intensity interval training; muscle fibre

WHY IS HIT SO EFFECTIVE?

BOTH HIGH-INTENSITY MUSCLE CONTRACTION

&

THE RATE AT WHICH SUBSTRATES ARE DEPLETED IN MUSCLE ACTIVATE AMPK SIGNIFICANTLY.



J Physiol 000.00 (2016) pp 1–16

SYMPOSIUM REVIEW

Physiological adaptations to interval training and the role of exercise intensity

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Abstract Interval exercise typically involves repeated bouts of relatively intense exercise interspersed by short periods of recovery. A common classification scheme subdivides this method into high-intensity interval training (HIIT; 'near maximal' efforts) and sprint interval training (SIT; 'supramaximal' efforts). Both forms of interval training induce the classic physiological adaptations characteristic of moderate-intensity continuous training (MICT) such as increased aerobic capacity ($\dot{V}_{O_2max}$) and mitochondrial content. This brief review considers the role of exercise intensity in mediating physiological adaptations to training, with a focus on the capacity for aerobic energy metabolism. With respect to skeletal muscle adaptations, cellular stress and the resultant metabolic signals for mitochondrial biogenesis depend largely on exercise intensity, with

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TONY'S FAVORITE MITOCHONDRIAL BOOSTING WORKOUTS

Olympic Gold Medalist Sir Chris Hoy falling off his bike in pain after an interval session

CONTINUOUS ENDURANCE TRAINING

- 45+ MINUTES
- INTENSITY 180-AGE
- 3-7 DAYS PER WEEK
- USING SLEEP LOW AND FASTED STATE

SPRINT INTERVAL TRAINING

4-6 X 30 SECONDS ‘ALL OUT’ WITH 4.5 MINUTES EASY

4-6 X 20 SECONDS ‘ALL OUT’ WITH 2:10 MINUTES EASY

4-6 X 60 SECONDS ‘BEST HARDEST PACE’ WITH 4 MINUTES EASY

CONTINUOUS HIGH-INTENSITY INTERVAL TRAINING

60 SECONDS HARD WITH 60 SECONDS EASY REPEATED 10 TIMES

4 MINUTES HARD WITH 1 MINUTE EASY REPEATED 4-6 TIMES

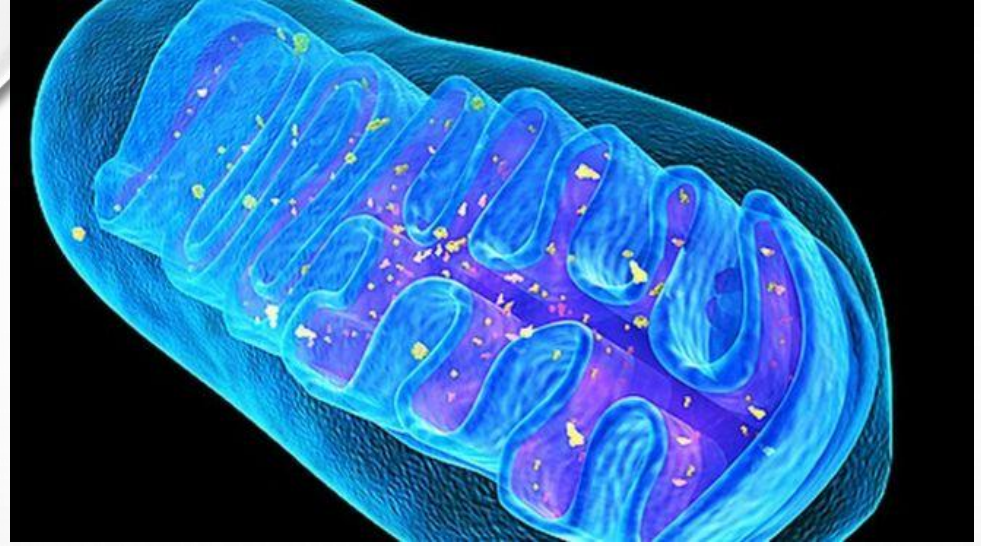
3 MINUTES HARD WITH 2 MINUTES EASY REPEATED 4-6 TIMES

INTERMITTENT HIGH-INTENSITY INTERVAL TRAINING

(20S WITH 40S REST X 8, REST 2 MINUTES) X 3

(30S WITH 30S REST X 8, REST 2 MINUTES) X 3

(40S WITH 20S REST X 8, REST 2 MINUTES) X 3



MIGHTY MITOCHONDRIA: THE MUSCLE'S FORGOTTEN POWERHOUSE

TONY BOUTAGY & CLAIRE NORGATE

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