MUNI
MED

Adaptation

Marie Nováková

ECOLOGICAL PHYSIOLOGY

examines the influence of environment on living systems and their ability to adapt to changed conditions - **ADAPTATION**

(Adaptation or Environmental Physiology)

ADAPTATION STUDIES

animal models
human volunteers

REACTION (REGULATION): direct, immediate response of organism on environmental changes (seconds, minutes)

ADAPTATION = a complex of biochemical, functional and structural changes in organism caused by long-lasting and repeated environmental changes (days, months, years)

Adaptation = adjustment THM

– Long-lasting structural and/or functional change

– Leads to decrease in energetic demands needed for keeping homeostasis under new (changed) conditions

– Functional / Evolutional advantage

ADAPTATION → INDIVIDUAL

ADAPTATION → GENETICALLY FIXED

ACCLIMATION

Reaction of whole organism on change in one environmental factor

ACCLIMATISATION

Reaction of whole organism on change in several environmental factors

MECHANISMS OD ADAPTATION

1. Changed plasticity of nervous system
- changes at molecular level in CNS
- gene expression changes
- regulation of number of neurites
- changes in neuronal nets (cortical fields)

2) Changes of autonomous tonus (athletes)
3) Changes in organ structure (adaptation to exercise)
4) Temporary changes of skin colour (sunbathing)

CIVILISATION DISEASES = maladaptation

- gastric ulcer disease
- hypertension
- CAD
- psychoses
- neuroses

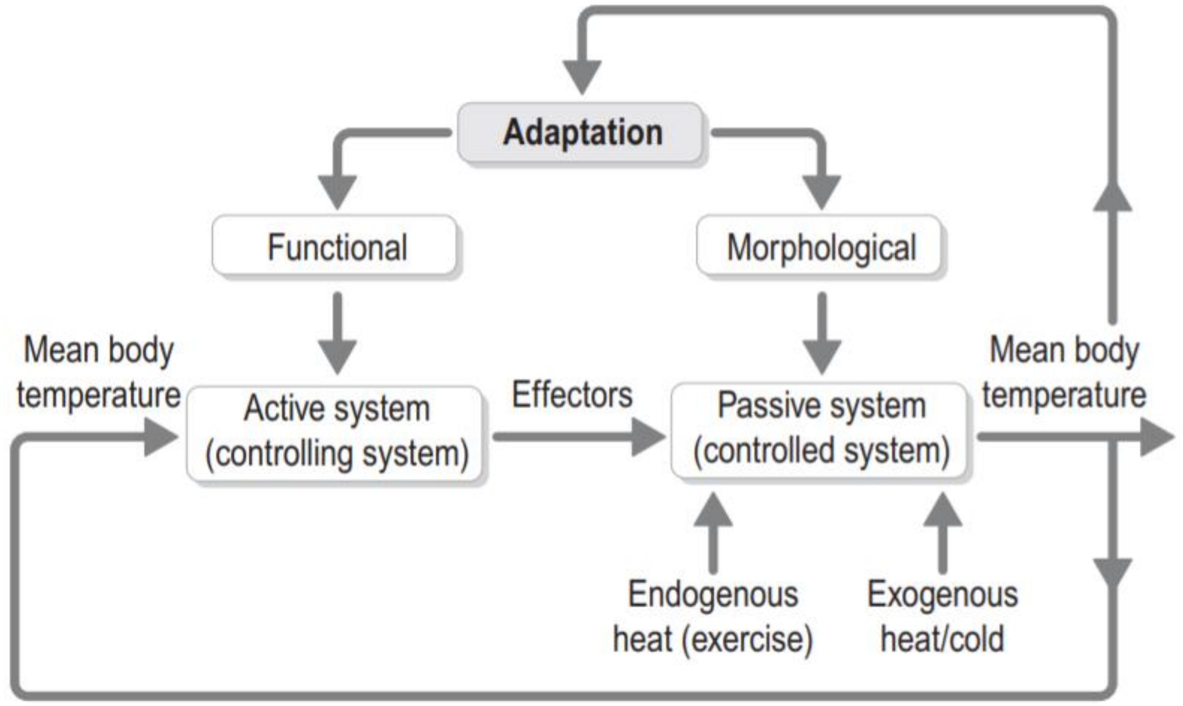
MUNI MED

ADAPTATION MECHANISMS

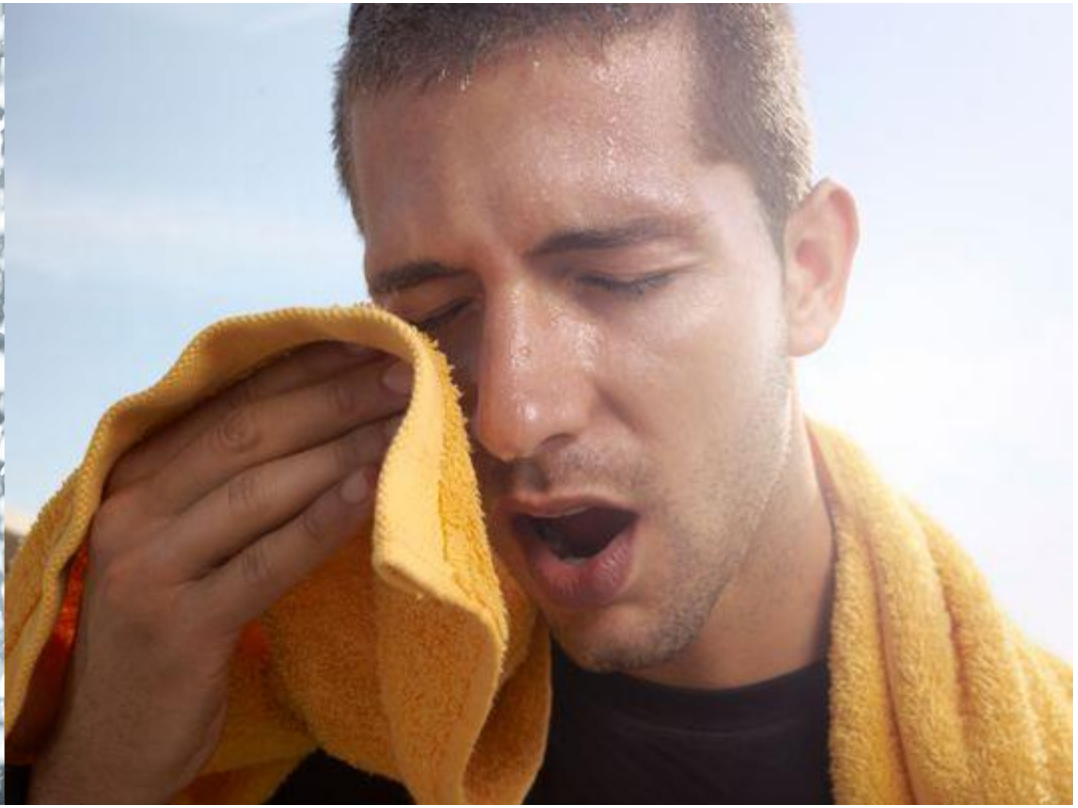
Example: adaptation of thermoregulation

- Sweat glands hypertrophy
- Increased subcutaneous adipose tissue

- Metabolisms/energetic exchange
- Sweating
- Activity



Adaptation to extreme ambient temperature

Source: www.freepik.com

MUNI
MED

ADAPTATION TO COLD

18th century: surviving of shipwrecked sailors in cold water
1887: V. Priesnitz, S. Kneipp
People suffer from low temperatures less in winter than in summer.

ADAPTATION	INSULATIVE METABOLIC HYPOTHERMIC

1. PROTECTION FROM HEAT LOSS (feather, vasoconstriction, increased amount of subcutaneous adipose tissue)
2. INCREASE OF HEAT PRODUCTION (higher metabolic exchange)
3. DOWNWARDS SHIFT OF SET-POINT (opposite to fever, similar to hibernating animals)

Acclimation.

Human: as tropic animals (thermoneutral zone between 27° – 32°C)

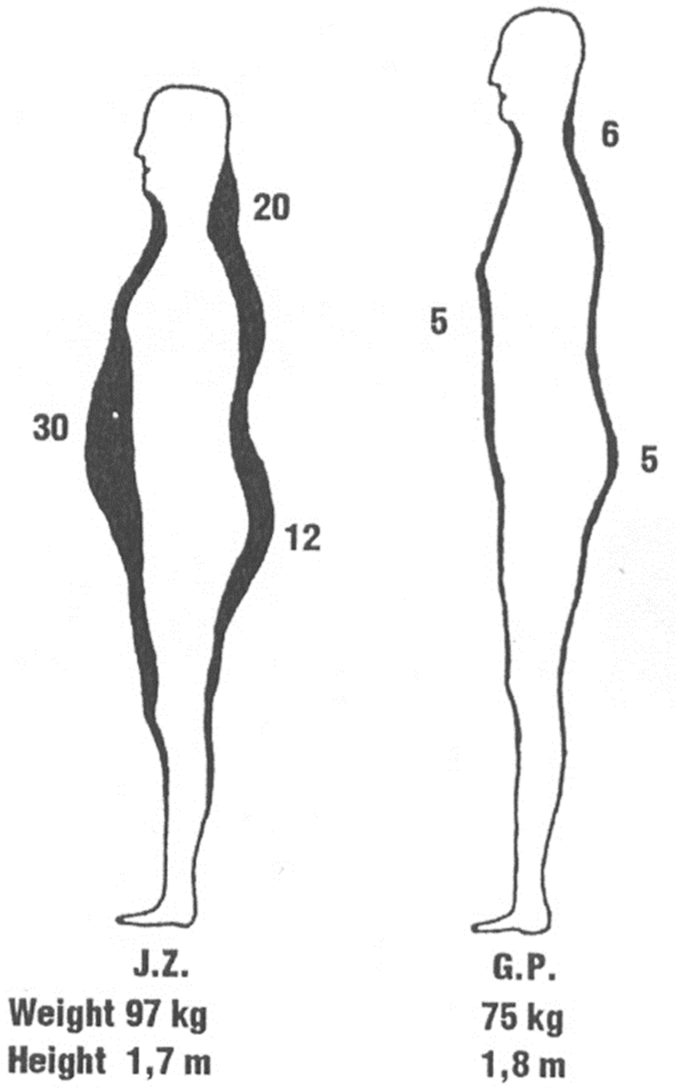
Seal, fog, seagull: arctic animals (thermoneutral zone between 20° – 40°C, thermoregulation starts below 20°C)

In humans always all three mechanisms activated during adaptation.

In adapted subject – O_2 consumption decreases, HR not changed, BP increases (by 20 – 40mmHg), feeling of discomfort is lower (starts at lower temperature), downward shift of set-point (by 0.75°C)

COLD ADAPTATION PROCESS

- Mainly re-setting of set-point (new value)
- Changed diet preferences (higher energy i
mass increase, slow increase in body fat pe
- Cold diuresis (Na^+ and K^+ excretion) – up t
haemoconcentration, increased leucocytes a
- Changed glycaemia: in non-adapted decre
increases (no more stress)
- Decreased skin threshold for pain (total ha
sensitivity); stress analgesia during adaptati
- Decreased threshold for shivering



Adaptation to cold THM

- Strategy: decrease heat loss (+ increase heat production)

- Increased appetite
- Increased subcutaneous adipose tissue
- Re-setting of thermoregulatory centre
  - Decreased temperature for activation of shivering thermogenesis

ADAPTATION TO HEAT

SWEAT PRODUCTION increases (may be even doubled)

THREASHOLD FOR SWEATING decreases to lower temperatures (both core and periphery)

DECREASED CONTENT OF ELECTROLYTES IN SWEAT

PERCEPTION OF THIRST increases

HIDROMEIOSIS (decreased production of sweat in humid hot climate, after the period of profuse sweating; decreases idle dropping of sweat)

ADAPTATION OF TOLERANCE TO HEAT in inhabitants in the tropics, threshold for sweating is increased to higher body temperatures.

ATTENTION must be paid to physical exercise !!!

Adaptation to heat THM

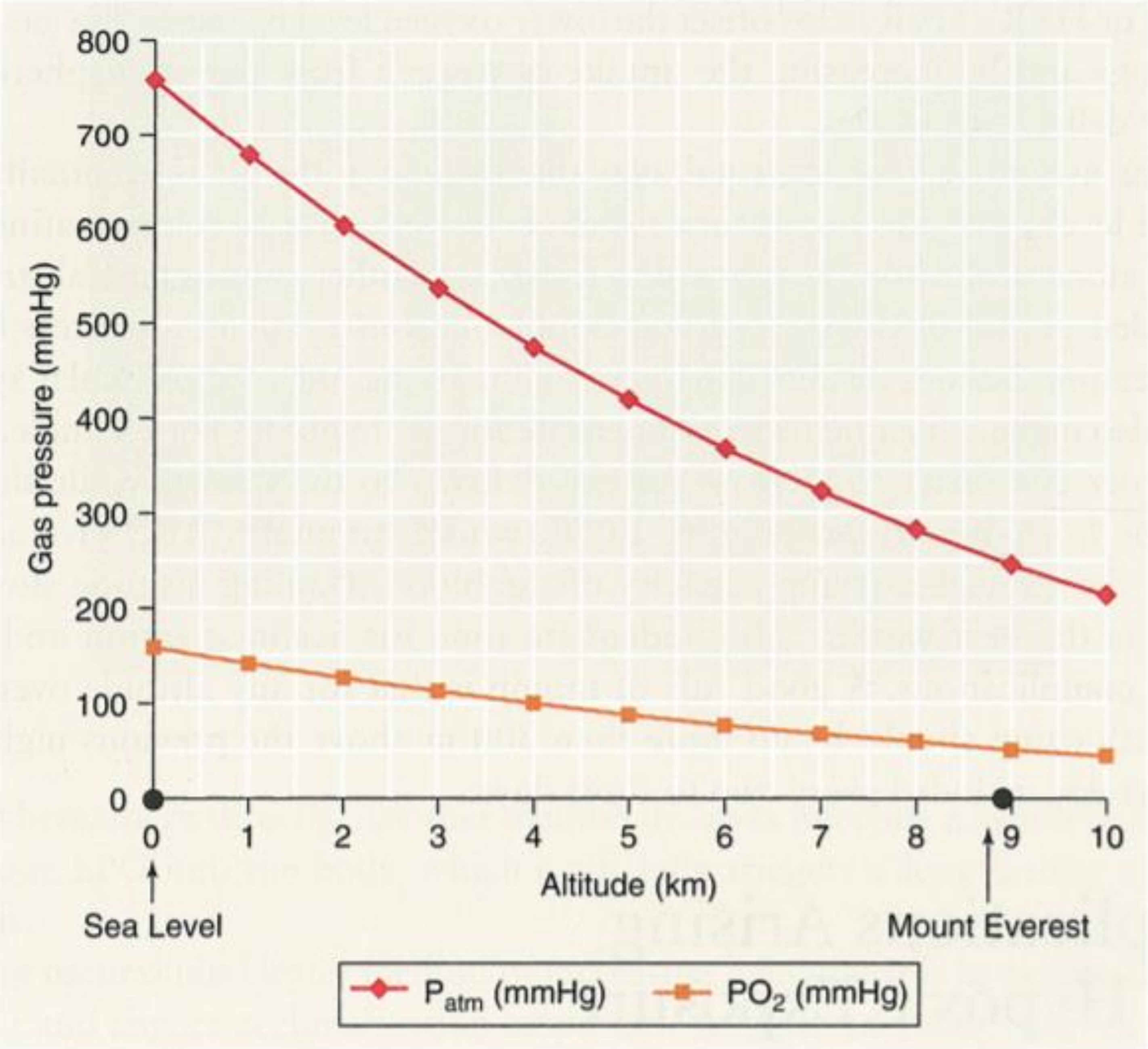
- Strategy: <u>increase heat output</u> + <u>decrease heat production</u>

- Decreased appetite
- Adaptation of sweating
  - Dependent on humidity; decreased sweat production, decreased ionic concentration
- Re-setting of thermoregulatory centre
  - Increased temperature for sweating activation

MUNI MED

ADAPTATION TO HIGH ALTITUDE

PHOTO B. Sir Edmund Hillary and Sherpa Tenzing Norgay on Everest.
This photograph shows Hillary and Norgay summiting Everest for the first time on May 1953. They used supplementary oxygen during their ascent.
Source: © The Kobal Collection.



800
700
600
500
400
300
200
100
0
Gas pressure (mmHg)
0
1
2
3
4
5
6
7
8
9
10
Altitude (km)
Sea Level
Mount Everest
P_{atm} (mmHg)
PO_2 (mmHg)

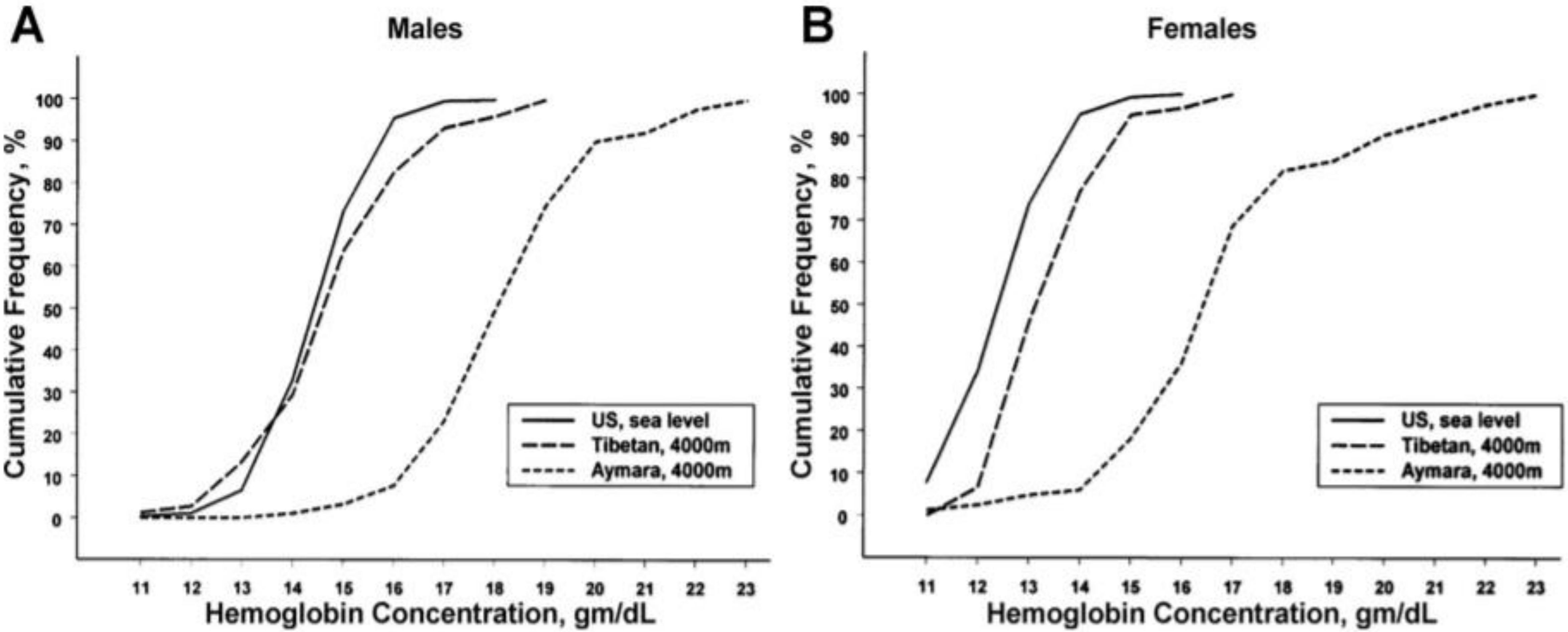
HIGH ALTITUDE ACCLIMATION (long-lasting stay)

At least several weeks, fully developed after several months or years.

CARDIOVASCULAR REACTIONS: HR and SV normalize, pulmonary arterioles constrict – pulmonary hypertension

RESPIRATORY REACTIONS : minute ventilation stabilises (directly proportional to high altitude hypoxia), central chemoreceptors adapt

INCREASED ERYTHROPOETIN SECRETION: polyglobulia, increased transport capacity of blood for O_2, blood viscosity, density of mitochondria, and myoglobin content



A
Males
Cumulative Frequency, %
0
10
20
30
40
50
60
70
80
90
100
11
12
13
14
15
16
17
18
19
20
21
22
23
Hemoglobin Concentration, gm/dL
US, sea level
Tibetan, 4000m
Aymara, 4000m
B
Females
Cumulative Frequency, %
Hemoglobin Concentration, gm/dL
US, sea level
Tibetan, 4000m
Aymara, 4000m

ACCLIMATISATION TO HIGH ALTITUDE - RECOMMENDATIONS

After 3 days: A-B balance restores, Hb concentration increases

After several weeks: exercising is possible

GENETIC ADAPTATION IN ALPINE TRIBES

- Bigger chest
- Higher density of pulmonary capillary net
- Bigger heart (EDV)
- Higher cardiac output
- Higher Hb concentration
- Bigger bone marrow

Adaptation from birth???

MUNI MED

Adaptation to physical exercise:
Static *vs.* Dynamic work

Source: www.freepik.com - photo created by gpointstudio

Source: www.freepik.com - photo created by alexeyzhilkin

Stimuli triggering adaptation

- Overthreshold change of either external and/or internal environment

- Long-lasting and/or repeated stimuli

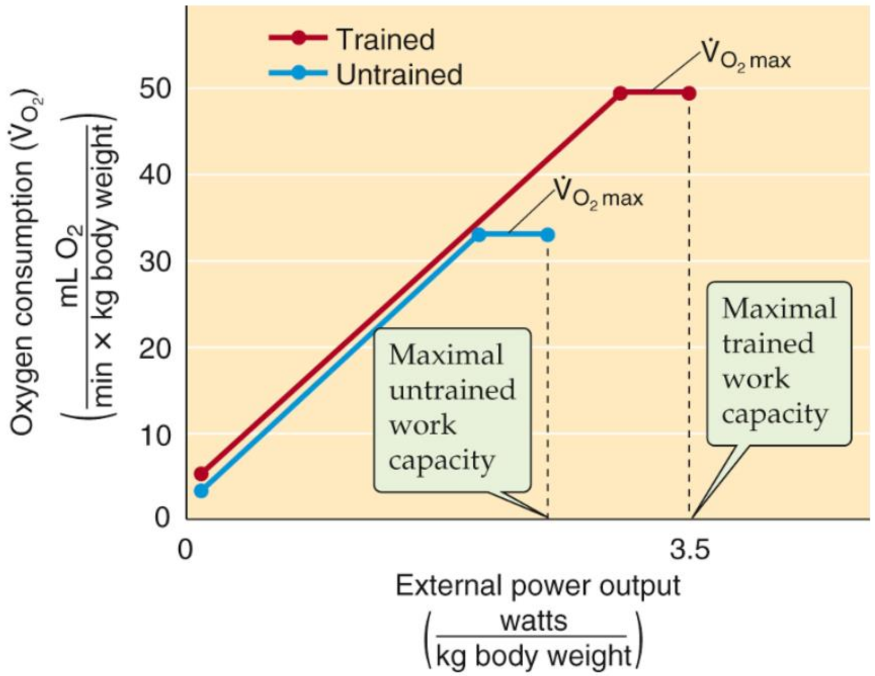
Adaptation to physical exercise THM

- Skeletal muscle
  - Hypertrophy, neovascularisation
- Cardiovascular system
  - Heart adaptation (concentric hypertrophy *vs.* athletes′ heart)
  - Polyglobulia, resp. increased haemoglobin concentration
  - Adaptation of blood pressure and perfusion regulations (skeletal muscle, heart, kidney)
- Respiratory system
  - Lungs growth (event. also chest growth), improved a-c diffusion
- Metabolism

Variable	Sedentary man Pretraining	 Posttraining	Runner
Cardiovascular			
HR at rest (beats • min^{-1})	71	59	36
HR max (beats • min^{-1})	185	183	174
SV rest (ml)	65	80	125
SV max (ml)	120	140	200
$\dot{Q}$ rest (L • min^{-1})	4.6	4.7	4.5
$\dot{Q}$ max (L • min^{-1})	22.2	25.6	32.5
Heart volume (ml)	750	820	1,200
Blood volume (L)	4.7	5.1	6.0
Systolic BP rest (mmHg)	135	130	120
Systolic BP max (mmHg)	210	205	210
Diastolic BP rest (mmHg)	78	76	65
Diastolic BP max (mmHg)	82	80	65
Respiratory			
$\dot{V}_E$ rest (L • min^{-1})	7	6	6
$\dot{V}_E$ rest (L • min^{-1})	110	135	195
TV rest (L)	0.5	0.5	0.5
TV max (L)	2.75	3.0	3.9
RR rest (breaths • min^{-1})	14	12	12
RR max (breaths • min^{-1})	40	45	50
Metabolic			
A-$\bar{v}O_2$ diff rest (ml • 100 ml^{-1})	6.0	6.0	6.0
A-$\bar{v}O_2$ diff max (ml • 100 ml^{-1})	14.5	15.0	16.0
$\dot{V}O_2$ rest (ml • kg^{-1} • min^{-1})	3.5	3.5	3.5
$\dot{V}O_2$ max (ml • kg^{-1} • min^{-1})	40.5	49.8	76.5
Blood lactate rest (mmol • L^{-1})	1.0	1.0	1.0
Blood lactate max (mmol • L^{-1})	7.5	8.5	9.0

Adopted from:
https://studentconsult.inkling.com/read/boron-medical-physiology-3e/chapter-60/figure-60-6

Oxygen uptake by lungs



- Spiroergometry
- Resting V_{O2}: **~3.6** mL O_2 / (min x kg)
- **$V_{O2\ max}$** – objective index for aerobic power
  - untrained middle age person: **30 – 40** mL O_2 / (min x kg)
  - elite endurance athletes: **80 – 90** mL O_2 / (min x kg)
  - HF / COPD patients: **10 – 20** mL O_2 / (min x kg)

Determinants of $V_{O2\,max}$

1. Uptake of O_2 by the lungs
   - pulmonary ventilation
2. O_2 delivery to the muscles
   - blood flow (pressure gradient – cardiac output x resistance)
   - hemoglobin concentration
3. Extraction of O_2 from blood by muscle
   - pO_2 gradient: blood - mitochondria

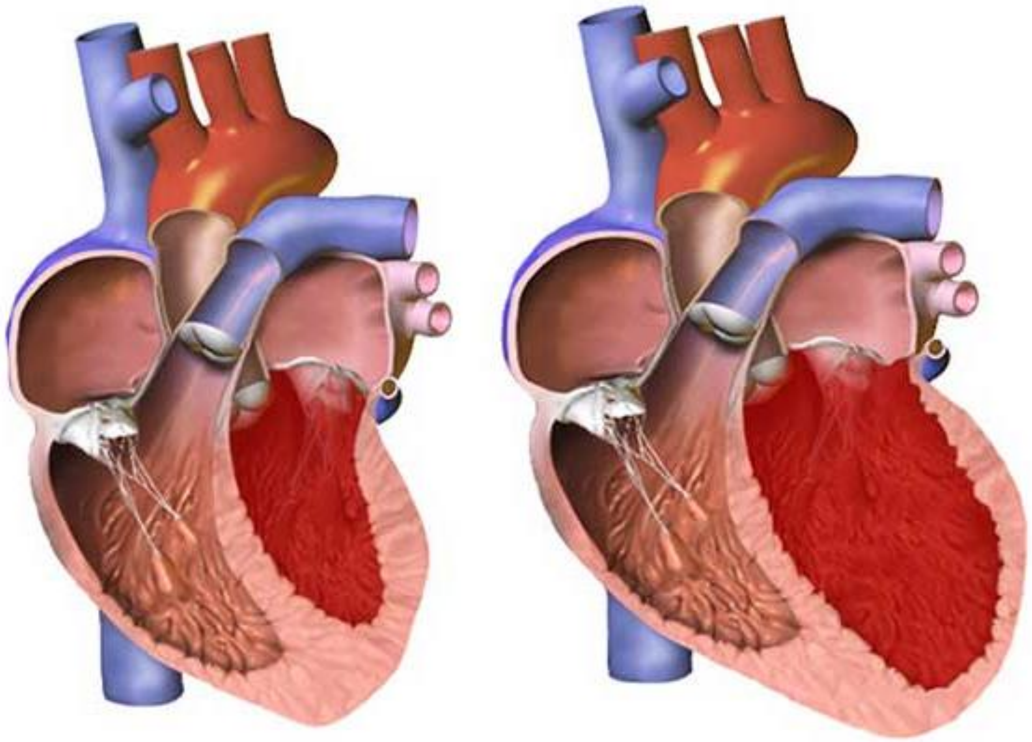
Athletes´ heart

– Adaptation to dynamic exercise

– ↑ LVEDV - ↑ SV - (baroreflex) ↓ HR

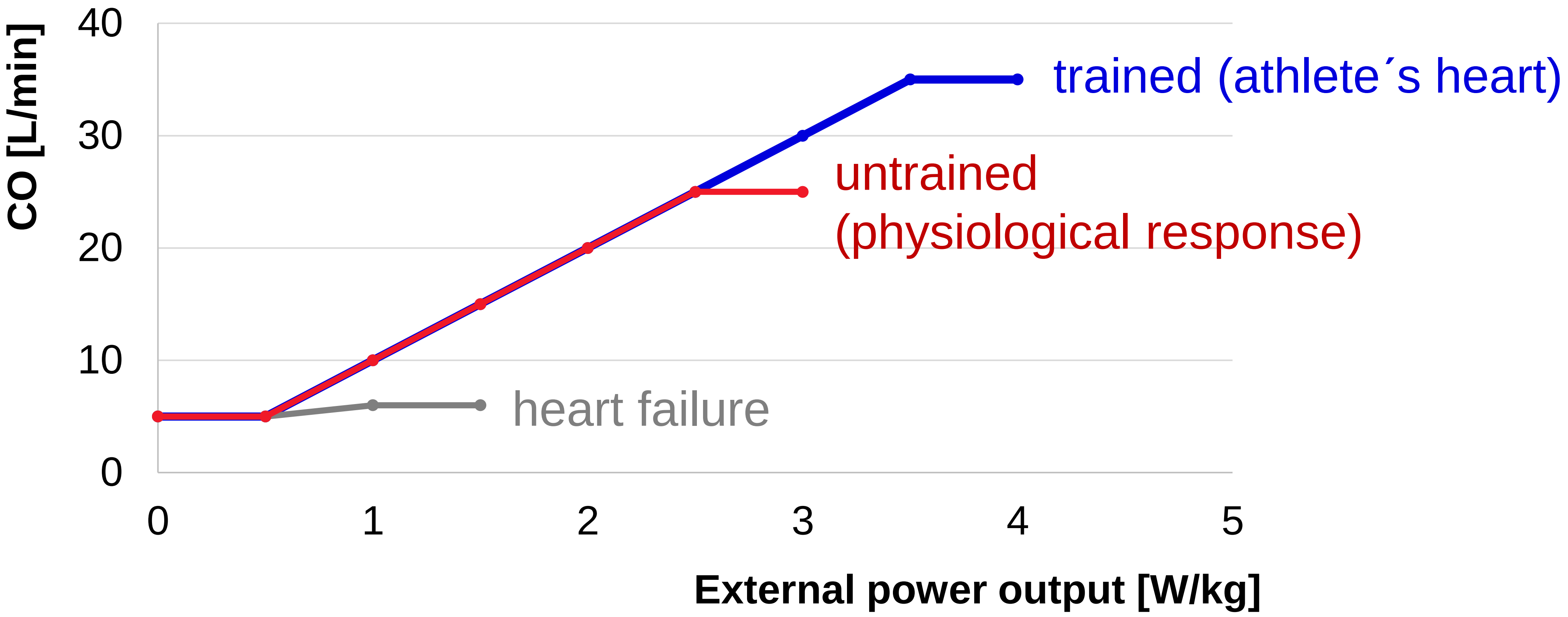
– ~ CO at rest

– ↑ cardiac reserve

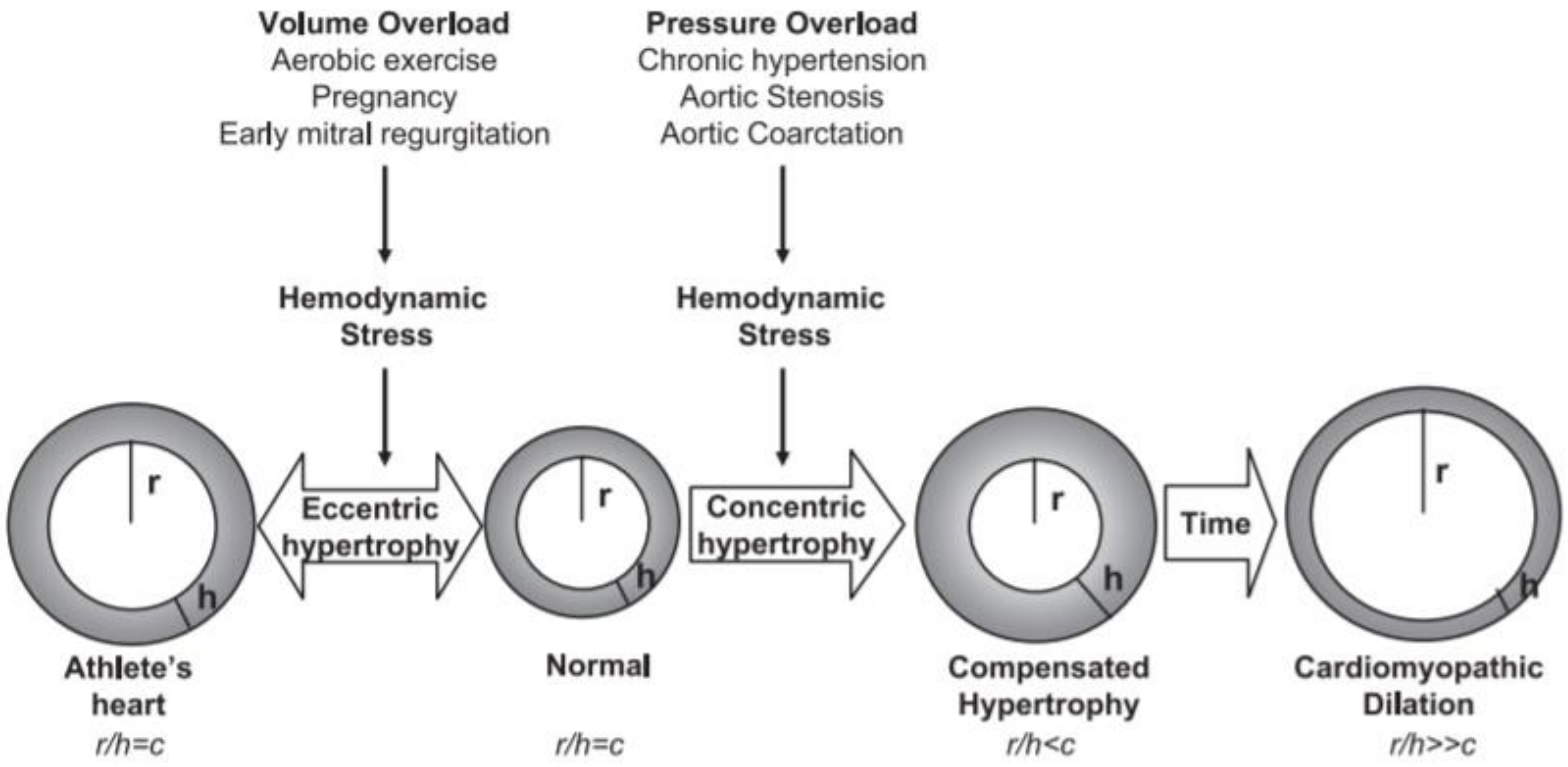


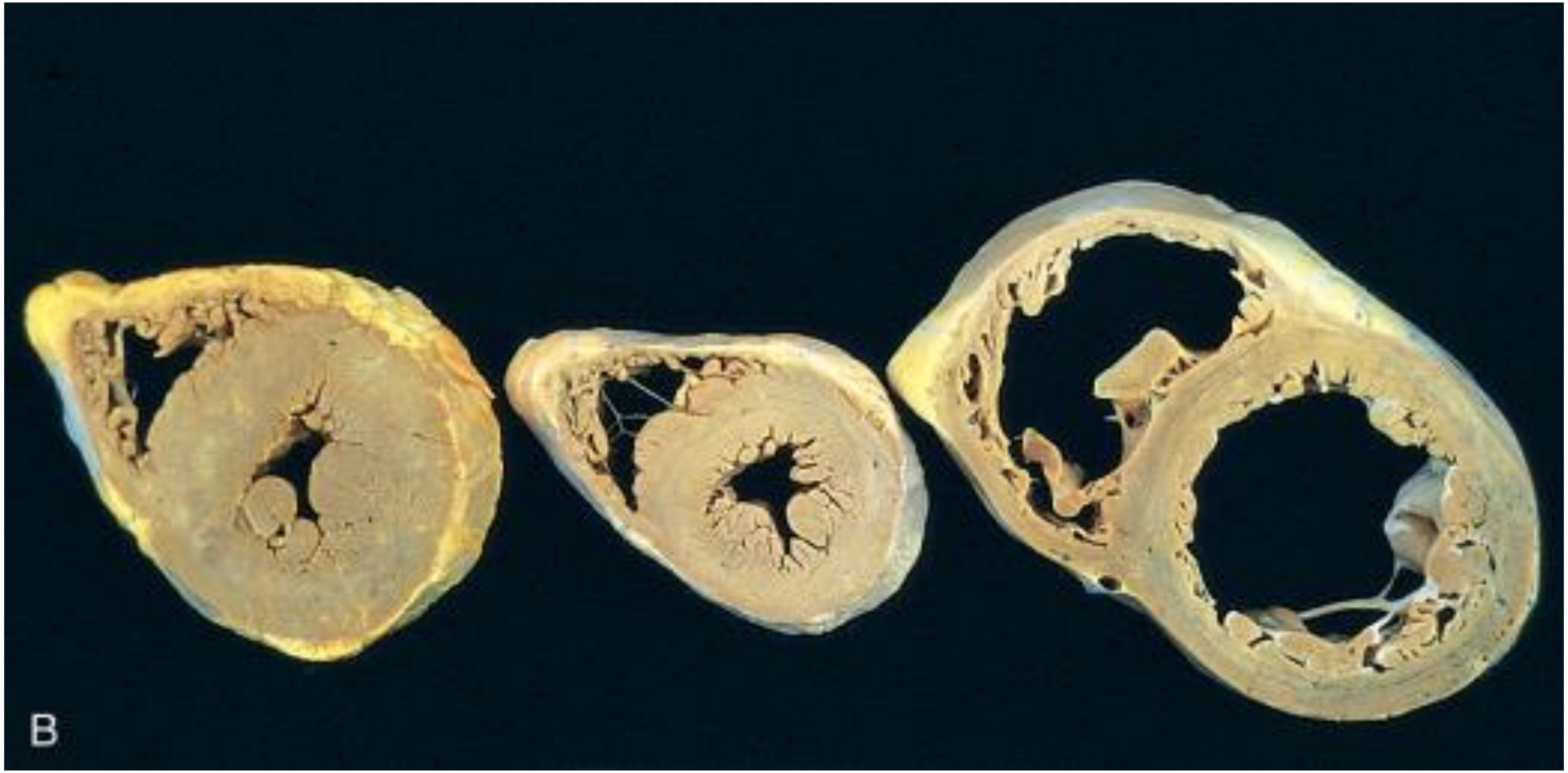
Source: https://assets.beta.meta.org/discover/thematic-feed/83-athletic-heart-syndrome.jpg

Cardiac reserve in healthy and failing heart



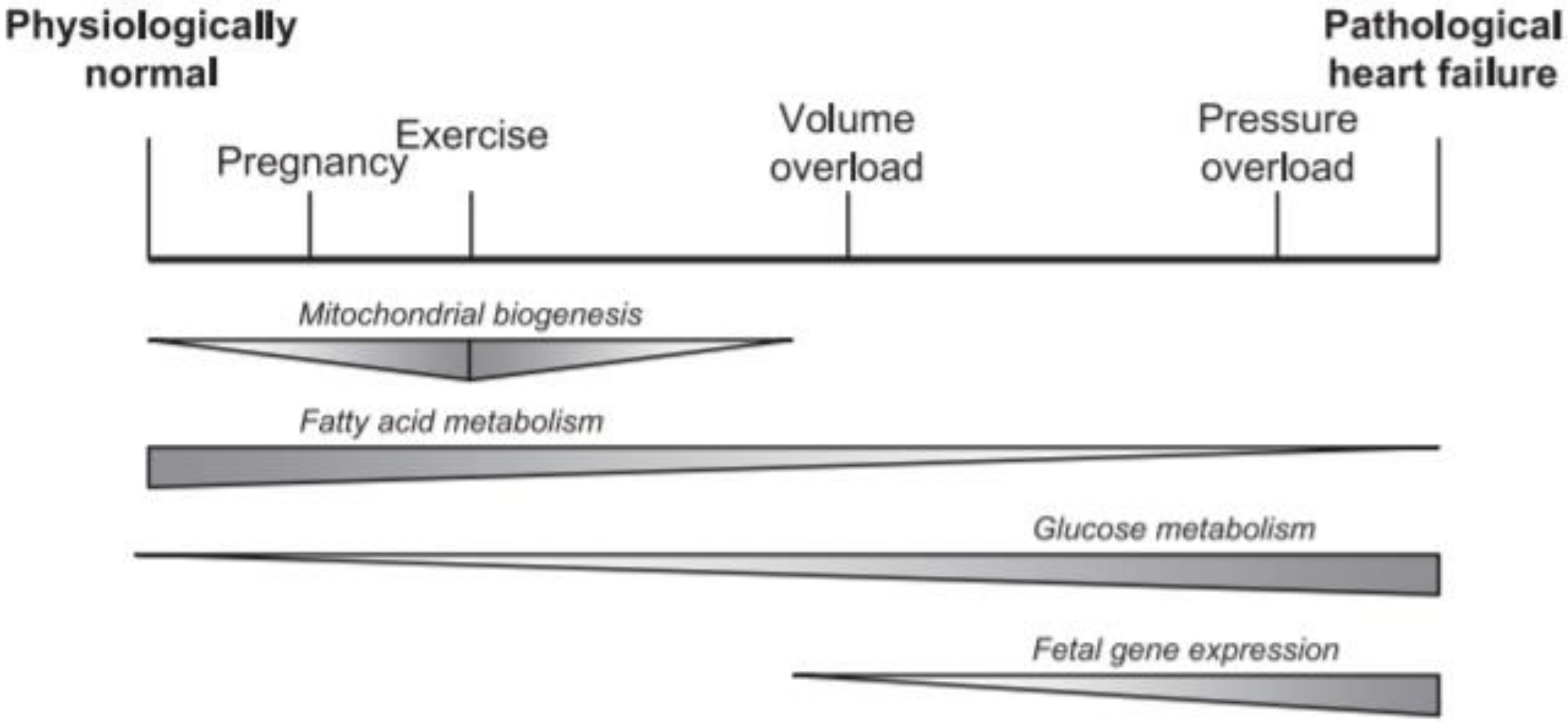
MUNI MED


Volume Overload
Aerobic exercise
Pregnancy
Early mitral regurgitation
Pressure Overload
Chronic hypertension
Aortic Stenosis
Aortic Coarctation
Hemodynamic Stress
Hemodynamic Stress
r
h
Eccentric hypertrophy
r
h
Concentric hypertrophy
r
h
Time
r
h
Athlete's heart
r/h=c
Normal
r/h=c
Compensated Hypertrophy
r/h<c
Cardiomyopathic Dilation
r/h>>c



Transversal heart sections:

heart with concentric hypertrophy (left)
healthy heart (centre)
heart with excentric hypertrophy = hypertrophy + dilation (right)


Physiologically normal
Pathological heart failure
Pregnancy
Exercise
Volume overload
Pressure overload
Mitochondrial biogenesis
Fatty acid metabolism
Glucose metabolism
Fetal gene expression

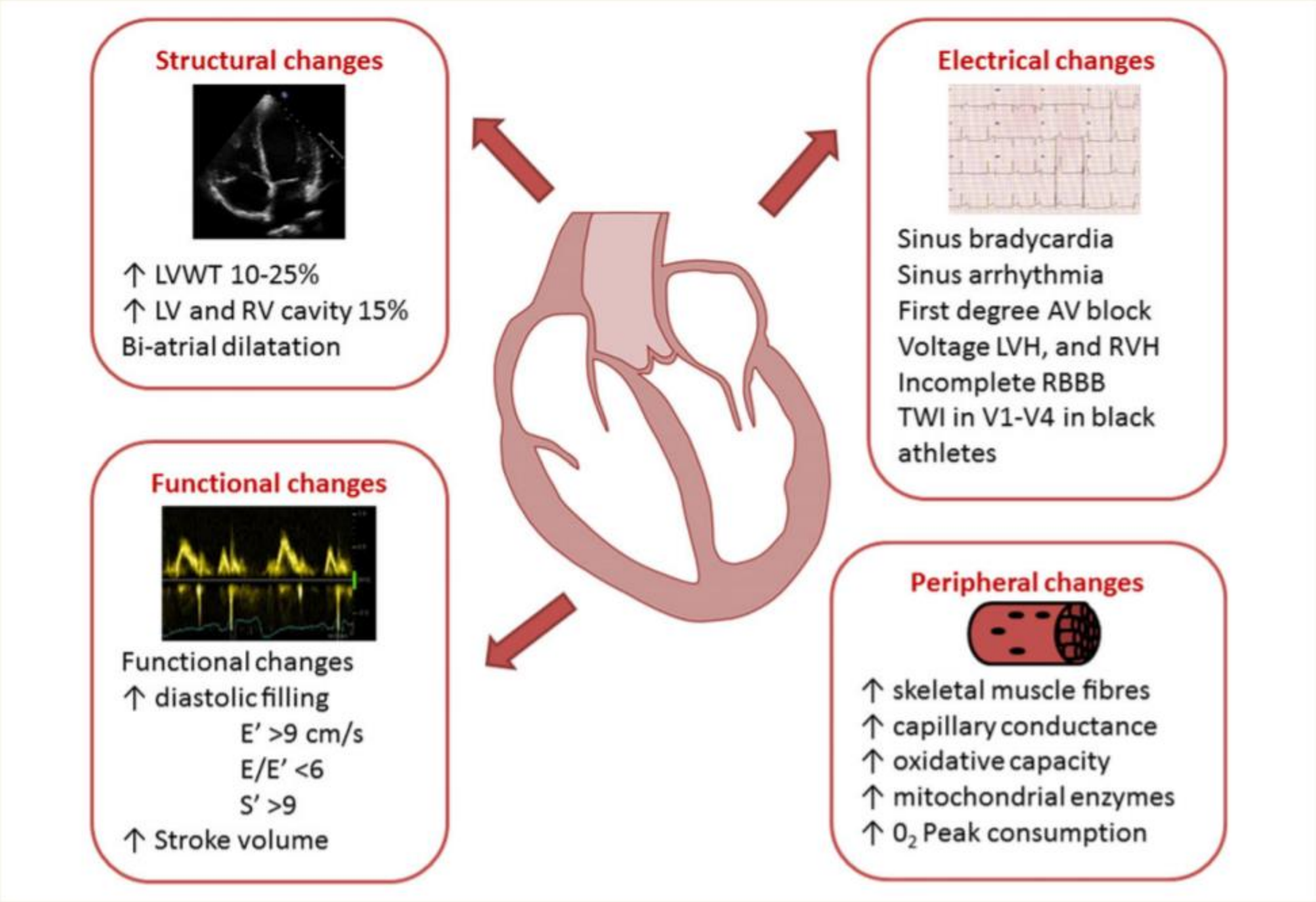


Figure 2 Cardiovascular and peripheral adaptation to exercise in athletes. AV, atrioventricular; LV, left ventricular; LVH, left ventricular hypertrophy; LVWT, left ventricular wall thickness; RV, right ventricle; RVH, right ventricular hypertrophy; TWI, T-wave inversion.

EXERCISE AND HEART – GOOD, BAD, HARMFUL ???

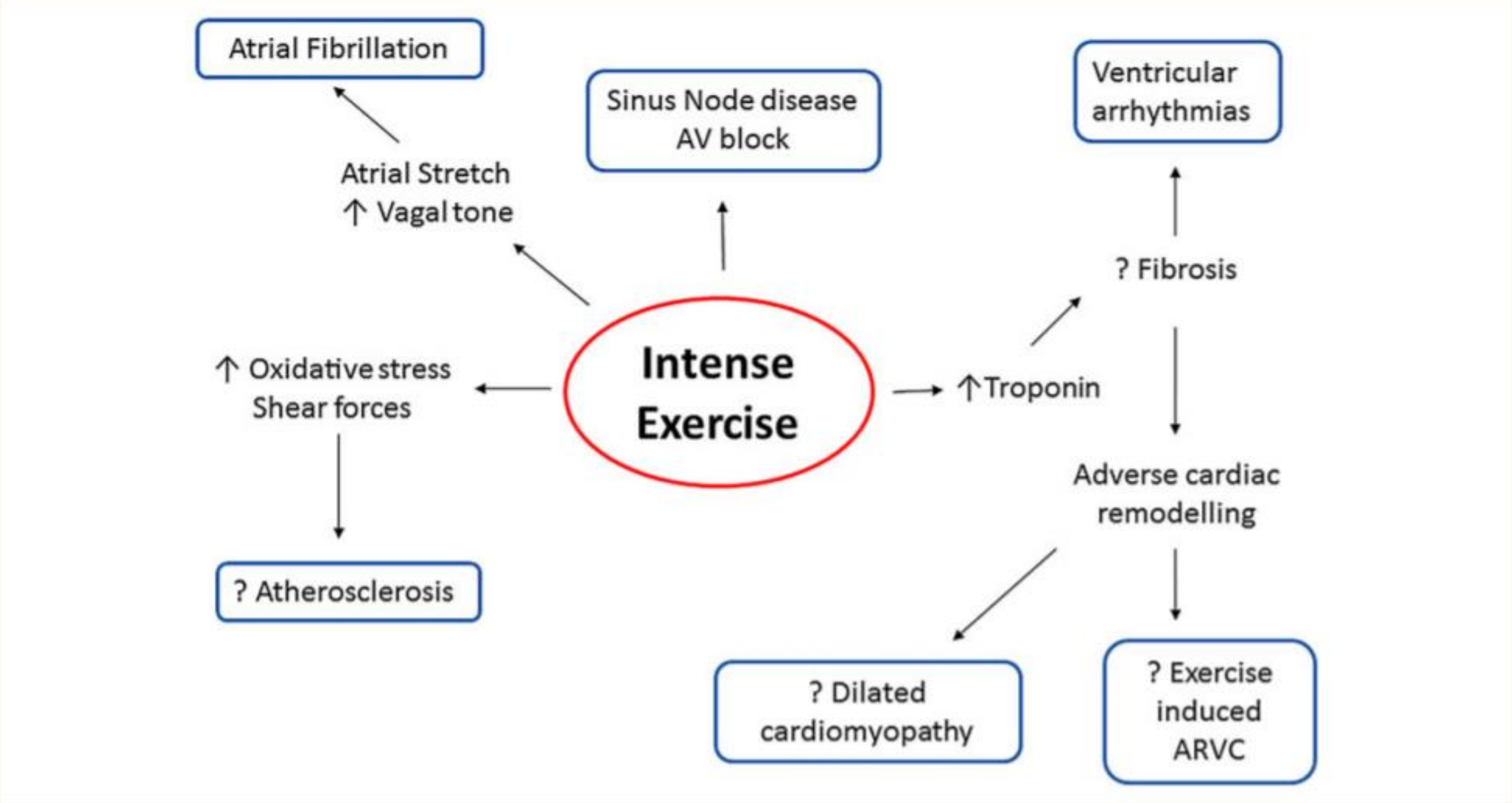


Figure 6 Speculated mechanisms for the detrimental effects of exercise. ARVC, arrhythmogenic right ventricular cardiomyopathy; AV, atrioventricular; DCM, dilated cardiomyopathy.