Training in hypoxia and performance.

Jean-Paul Richalet

Laboratoire “Réponses cellulaires
et fonctionnelles à l’hypoxie”,
Hôpital Avicenne, Bobigny
Université Paris 13 - FRANCE

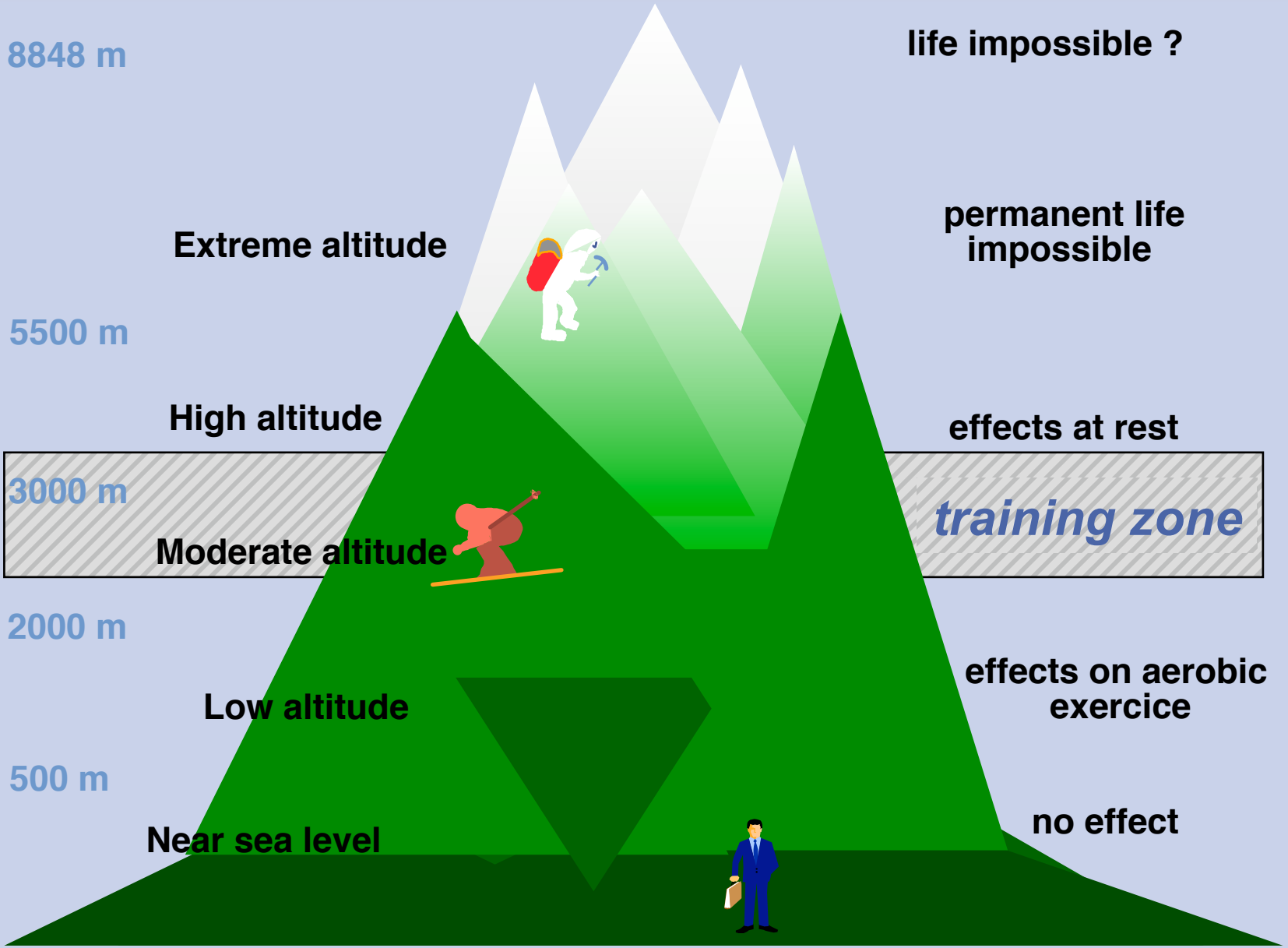
FIMS
Barcelona
Nov. 2008

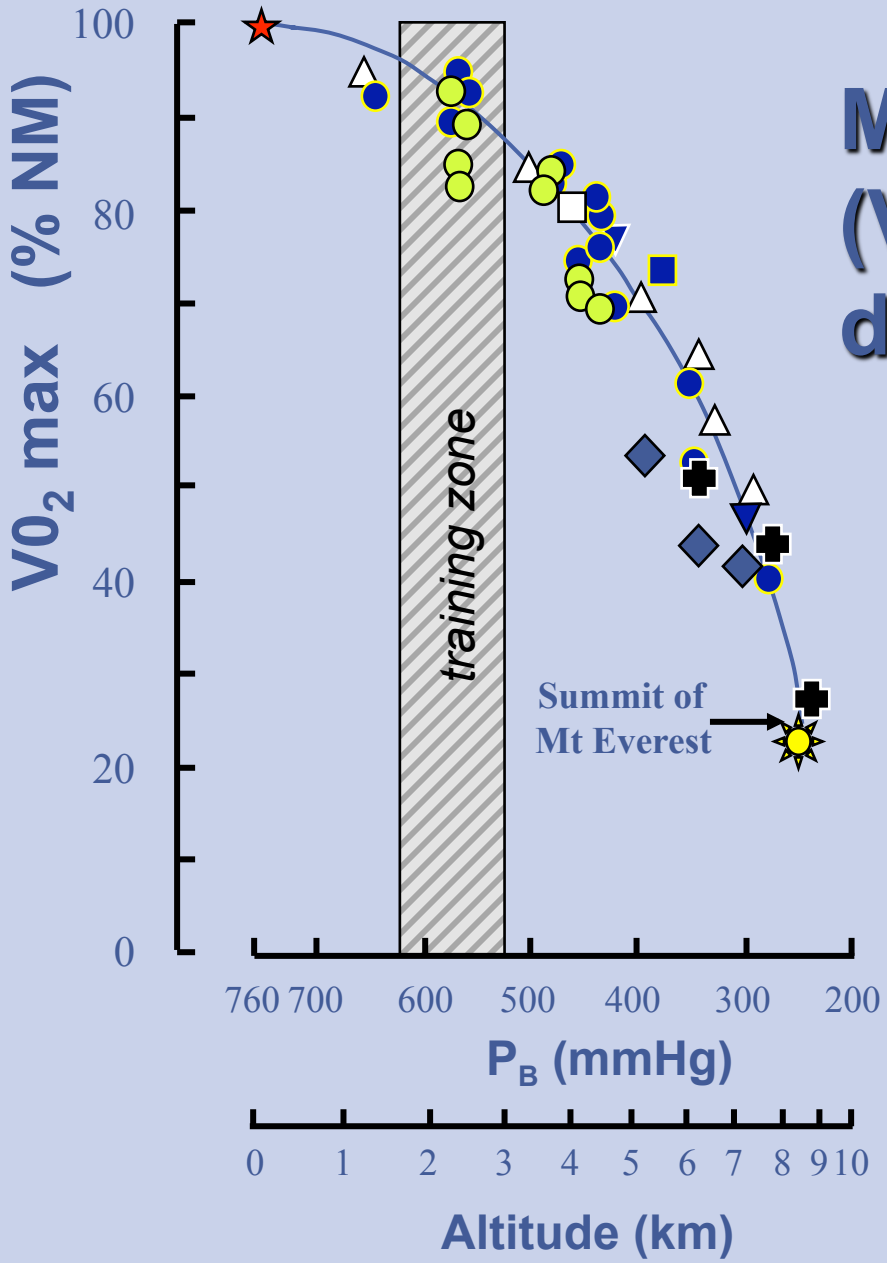
Olympic games and high altitude: MEXICO, 1968

Bob Beamon, long jump: 8.90 m, world record for 22 years

Ron Clarke, 10000m: exhaustion at the end of the race

BIOLOGICAL DEFINITION OF ALTITUDE



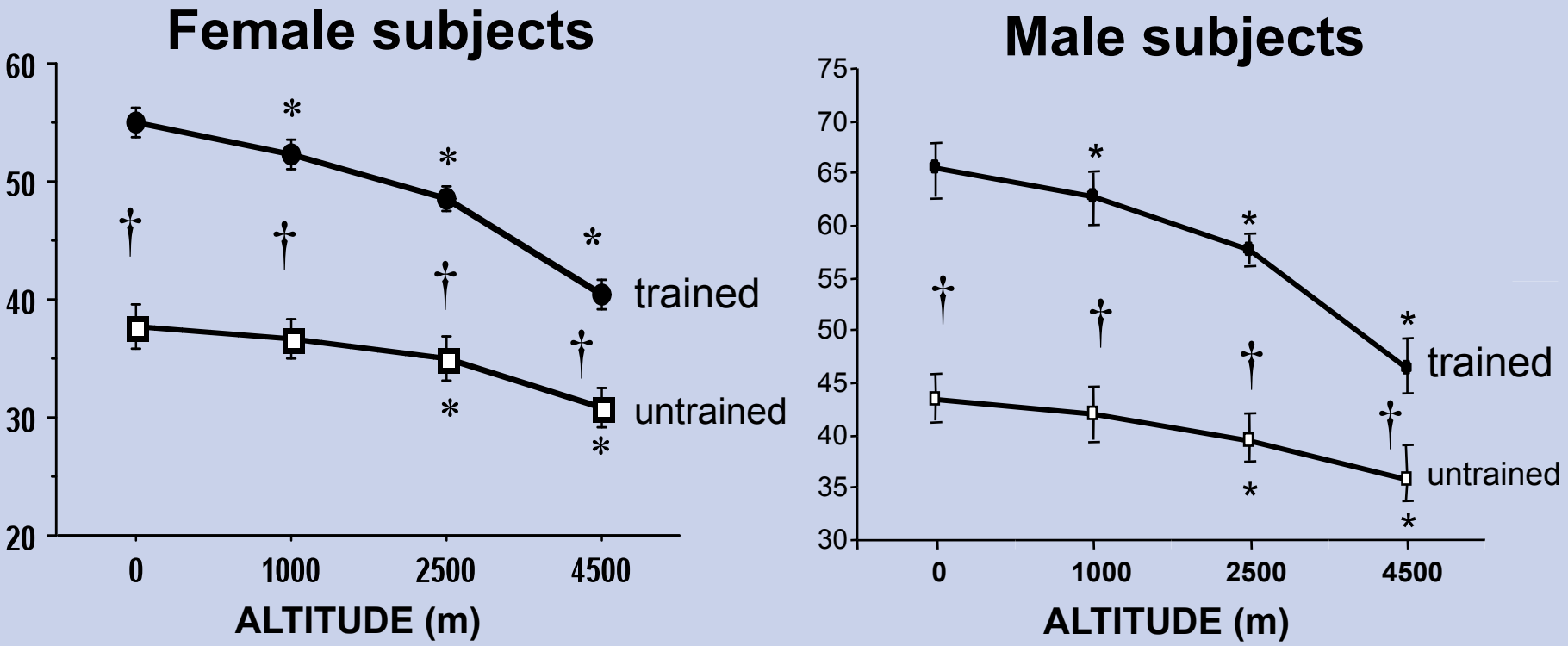


Maximal aerobic power (VO_2max) decreases with altitude

Consequence:
Training load must be lower when training at high altitude

Question ?
What are the limiting factors of performance ?

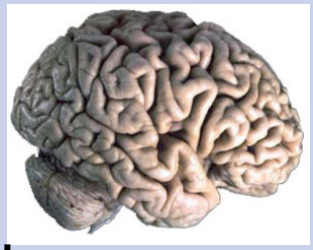
$\dot{V}O_{2max}$ (ml/min/kg)



* hypoxia vs normoxia † trained vs untrained

Trained subjects show a greater decrease in VO_2max with acute hypoxia

Woorons et al. 2005; Mollard et al., 2006; Mollard et al., 2007



O_2 CO_2

Central control

O_2 CO_2

Right heart

Left heart

cell O_2 CO_2

oxygen consumption

Aerobic production of energy

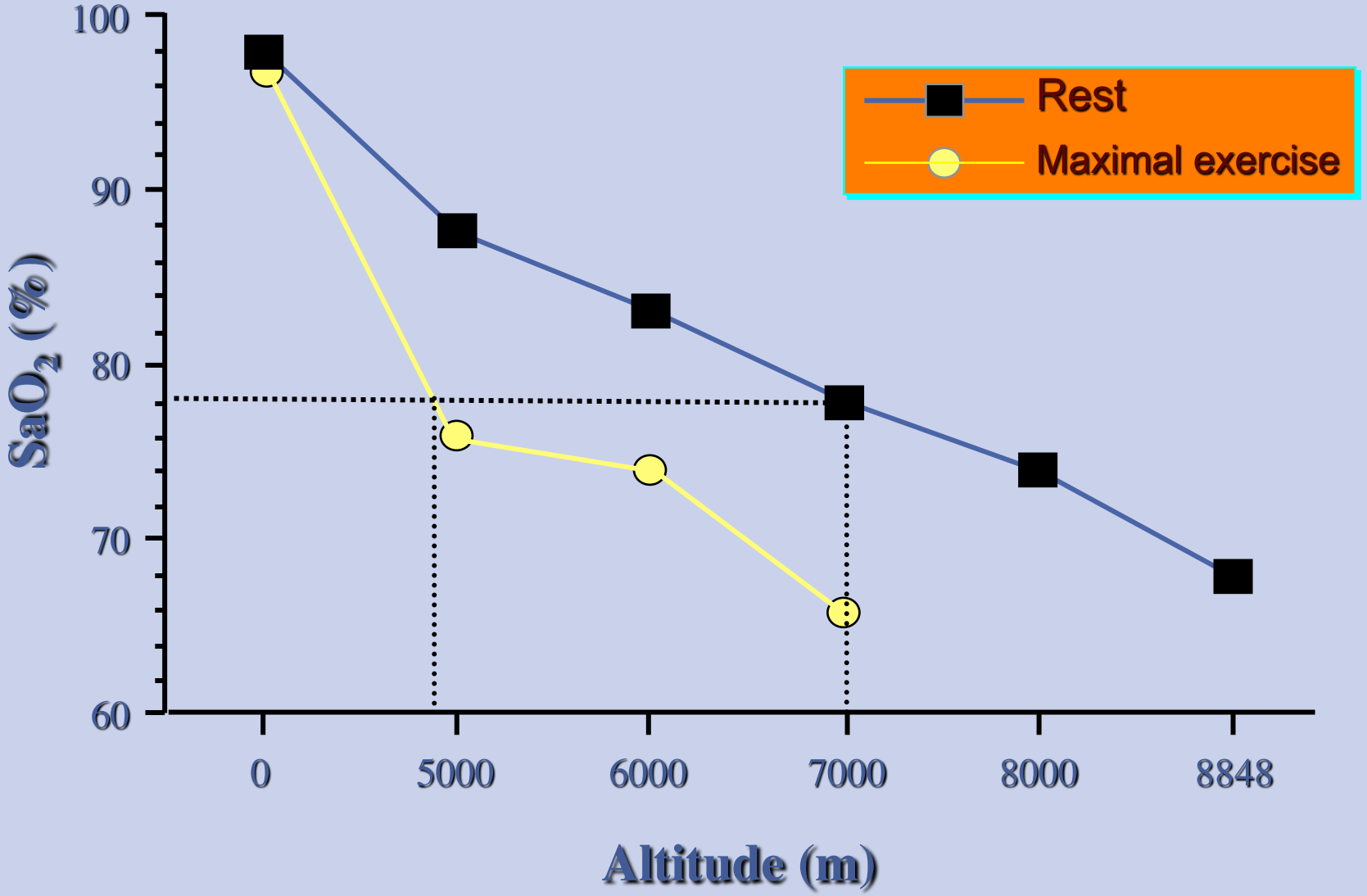
Pulmonary ventilation : CONVECTION

Alveolo-capillary transfer of oxygen : DIFFUSION

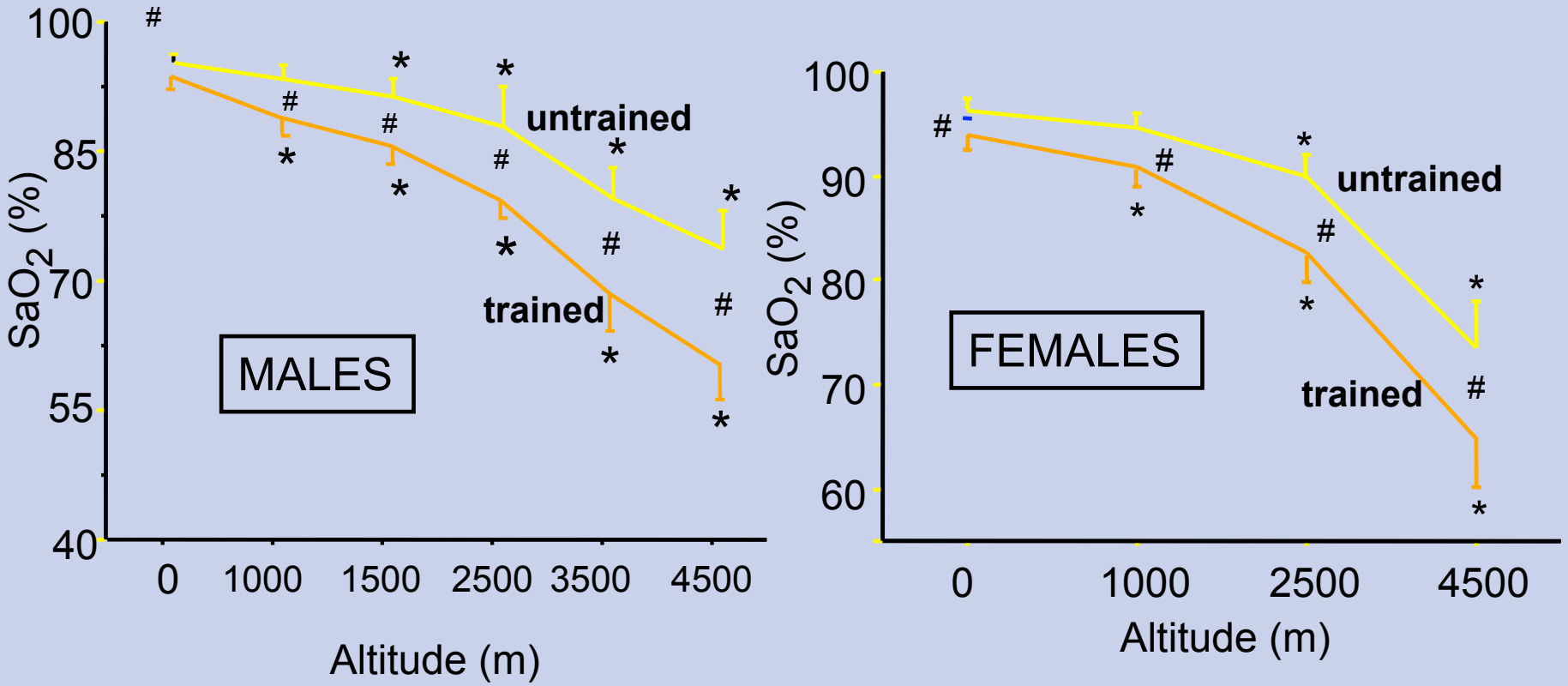
Transport of oxygen by the blood : CONVECTION

Diffusion of oxygen to the tissues : DIFFUSION

Decrease in arterial O_2 saturation at rest and exercise with increasing altitude



SaO_2 at maximal exercise

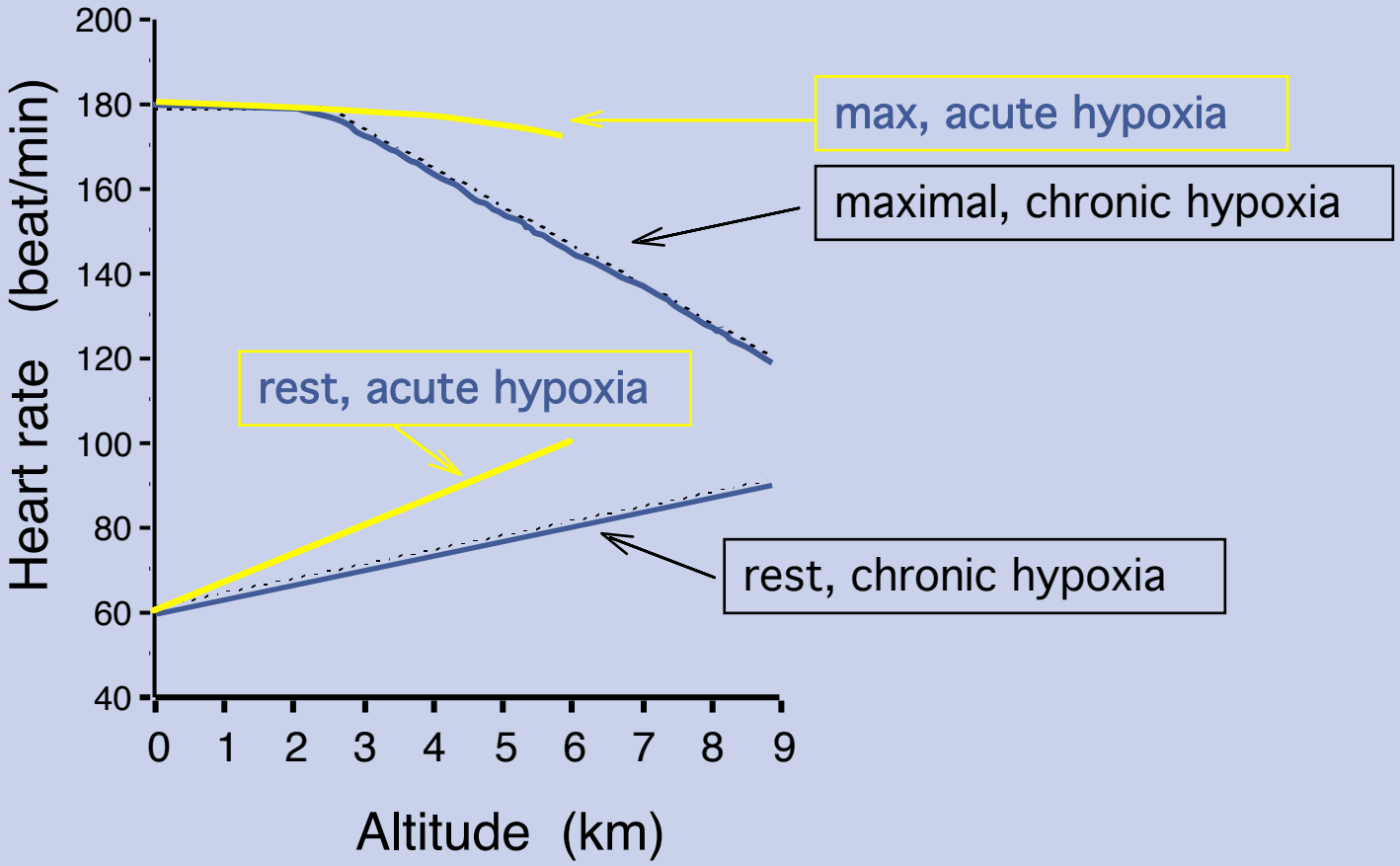


* hypoxia vs normoxia # trained vs untrained

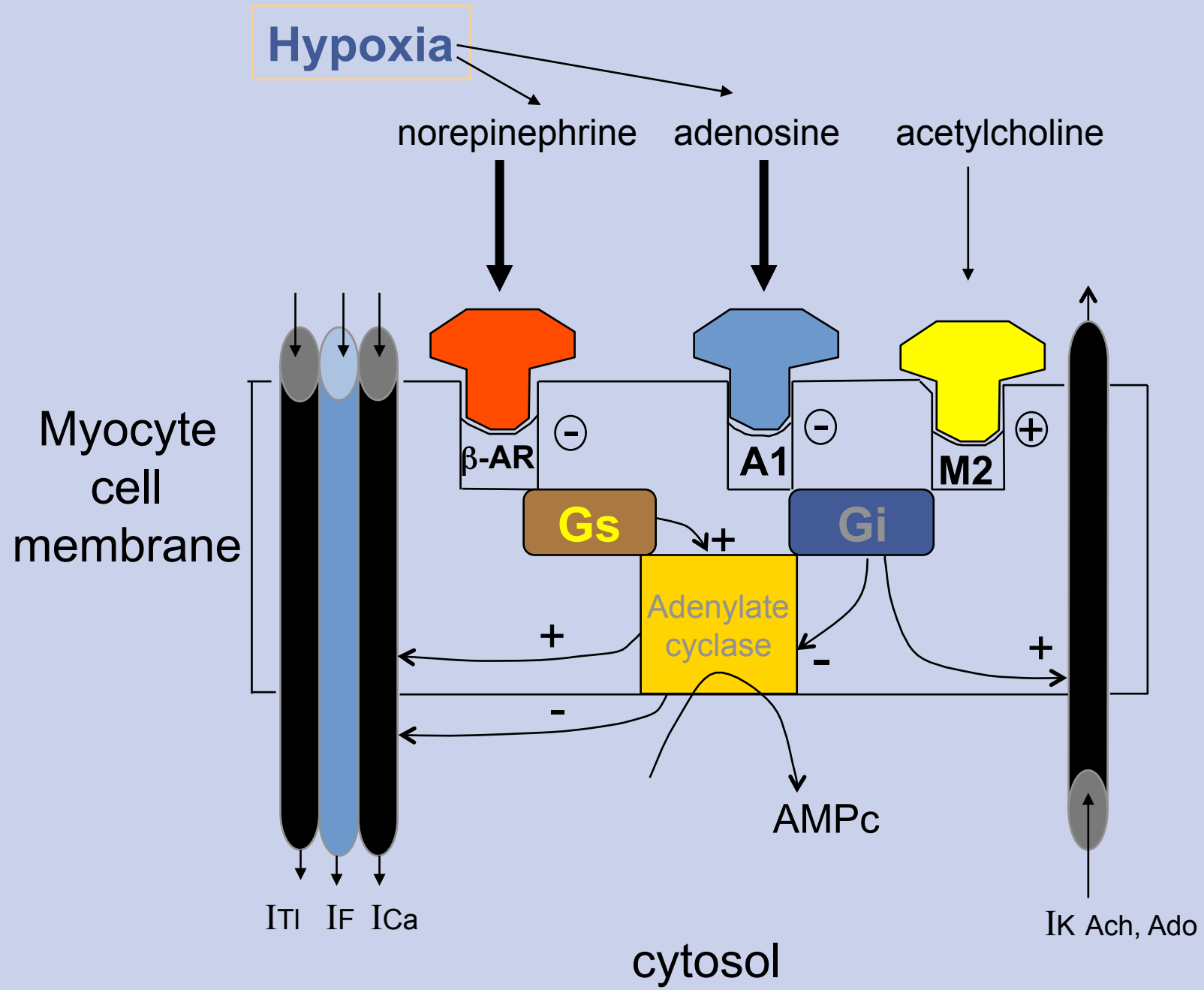
Trained subjects show a greater desaturation at exercise in acute hypoxia

Woorons et al. 2005; Mollard et al., 2006

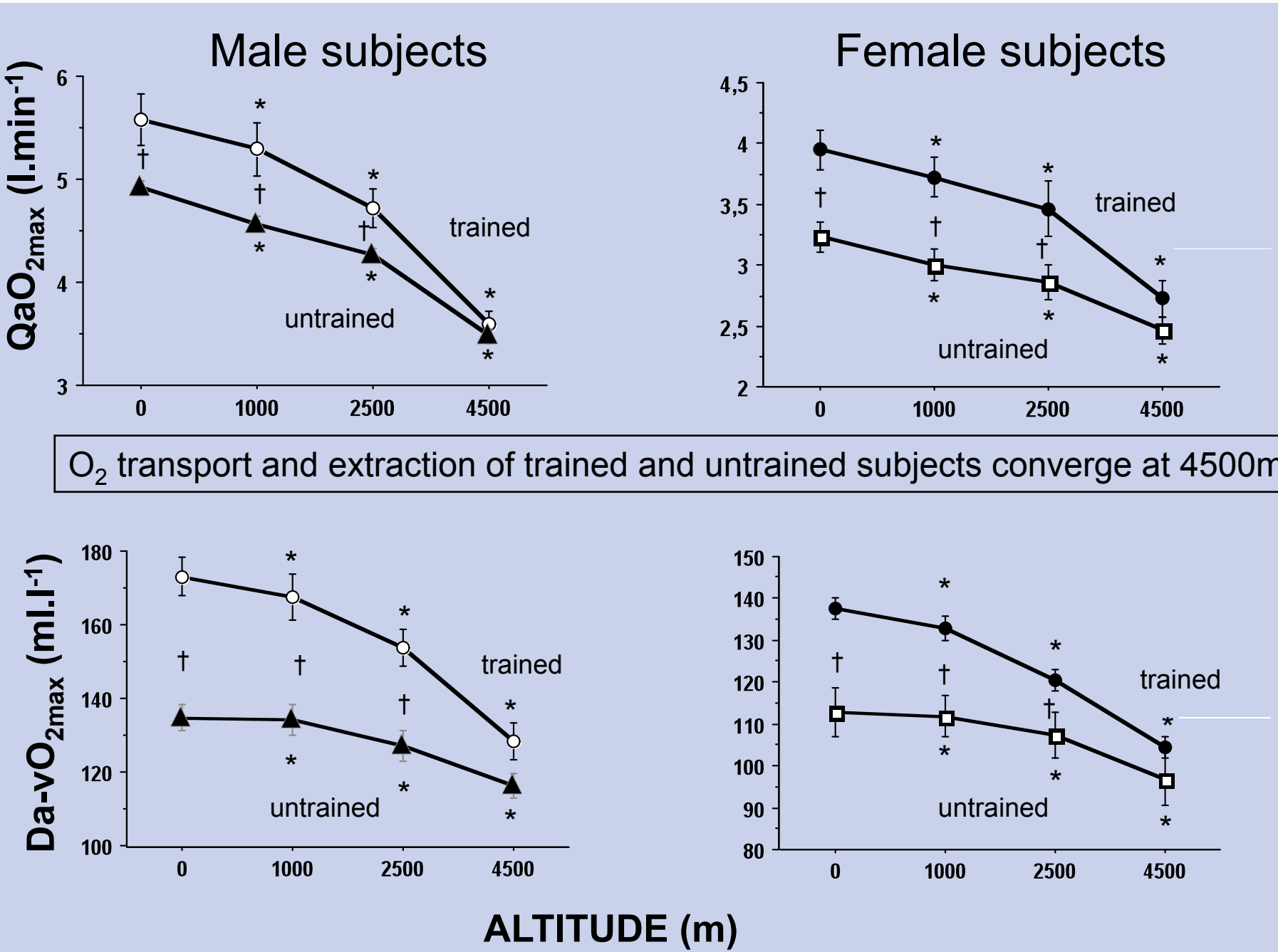
Adaptation of heart rate in acute and chronic hypoxia



Favret and Richalet, 2007



From: Lerman and Belardinelli, Circulation, 1991 ; Richalet et al. 1990; Favret and Richalet, 2007


Male subjects
Female subjects
QaO2max (l.min-1)
trained
untrained
0
1000
2500
4500
O2 transport and extraction of trained and untrained subjects converge at 4500m
Da-vO2max (ml.l-1)
ALTITUDE (m)

Subjects

5 endurance trained athletes (59.6 ± 2.8 ml/min/kg) and 6 physically active men (46.2 ± 2.8 ml/min/kg).

Protocol

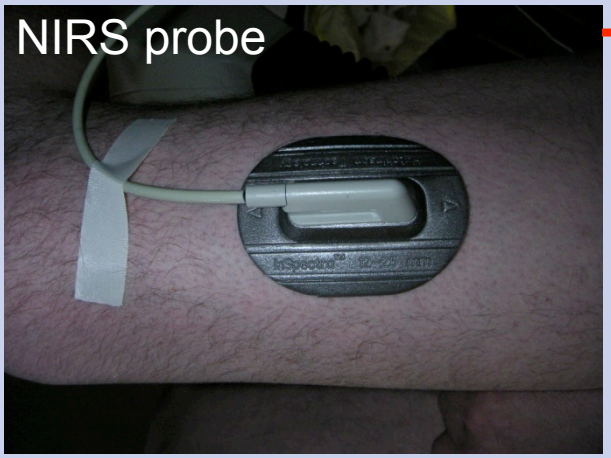
Each subject performed five VO_2 peak tests on a cyclo-ergometer at 4 different simulated altitudes: 0m, 1000m, 2500m and 4000m

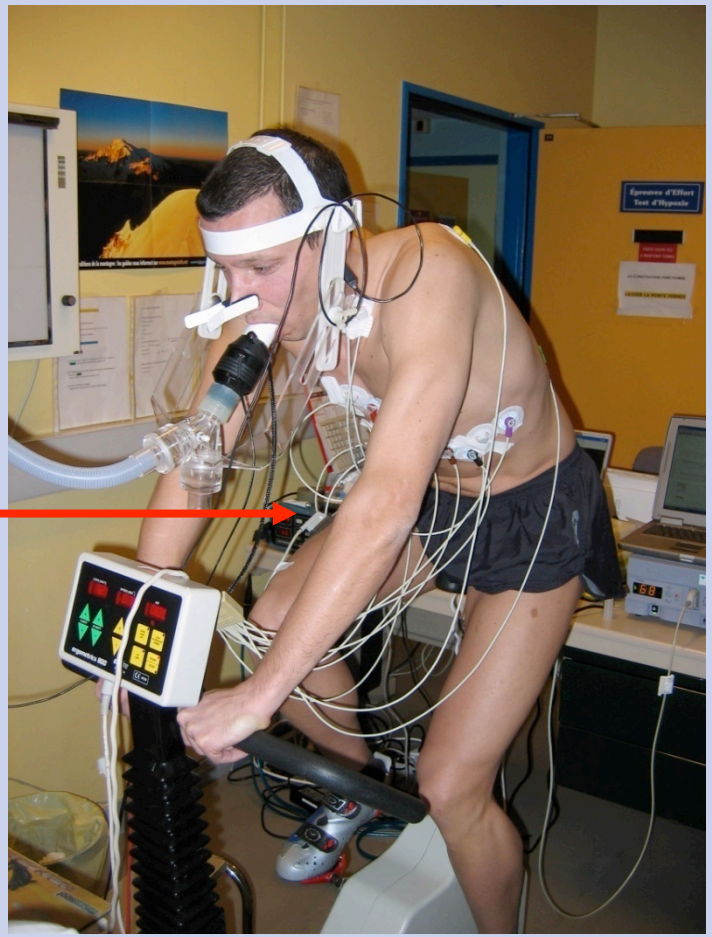
Measurements

Usual ventilatory and cardio-vascular parameters

Cardiac output using *transthoracic bio-impedance*

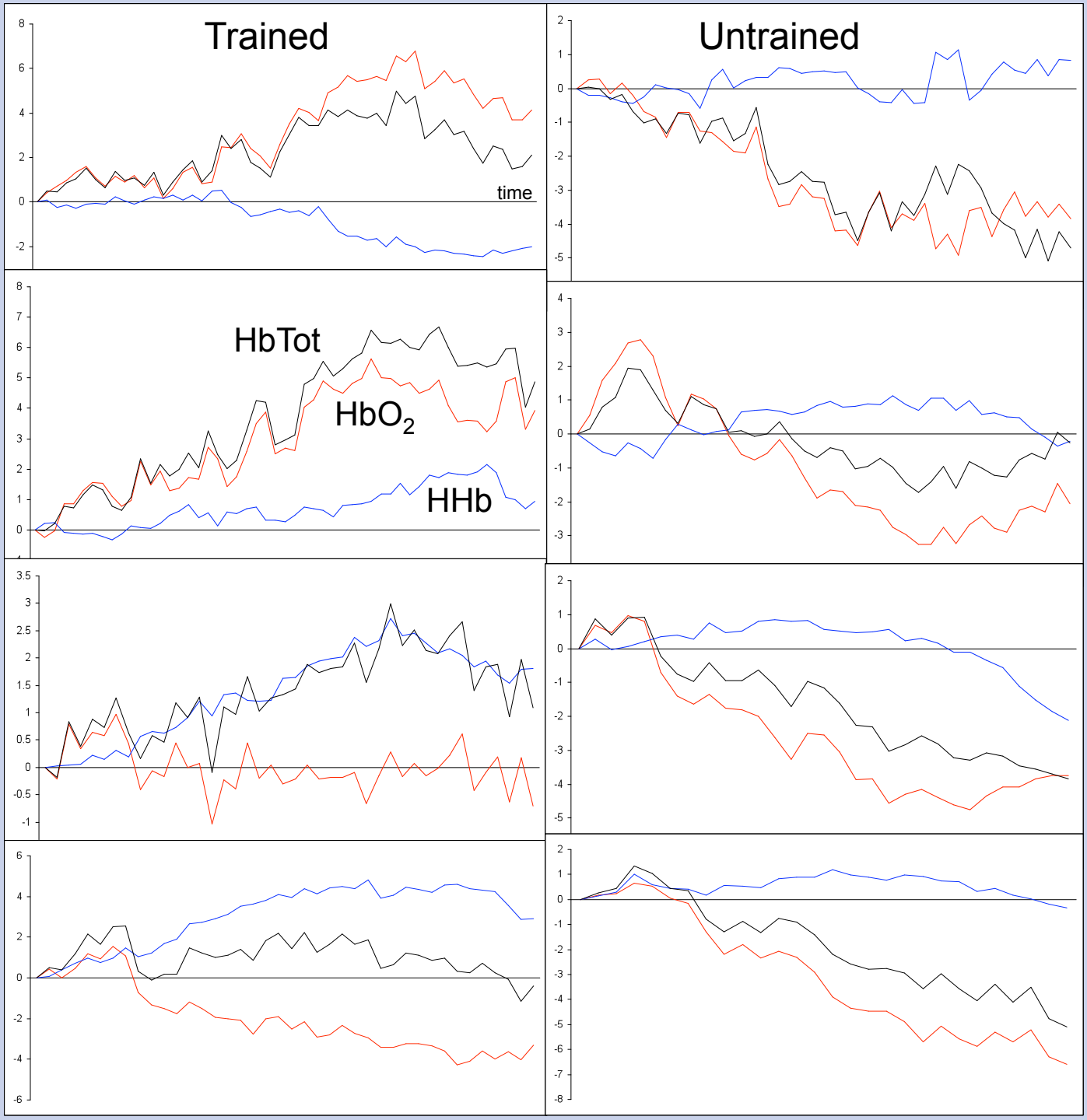
Muscle tissue HbO_2 and HHb using *Near InfraRed Spectroscopy* (NIRS; InSpectra Tissue Spectrometer Model 325, Hutchinson Technology, MN, USA).





NIRS

Vastus lateralis



Conclusions

Maximal aerobic performance decreases with altitude

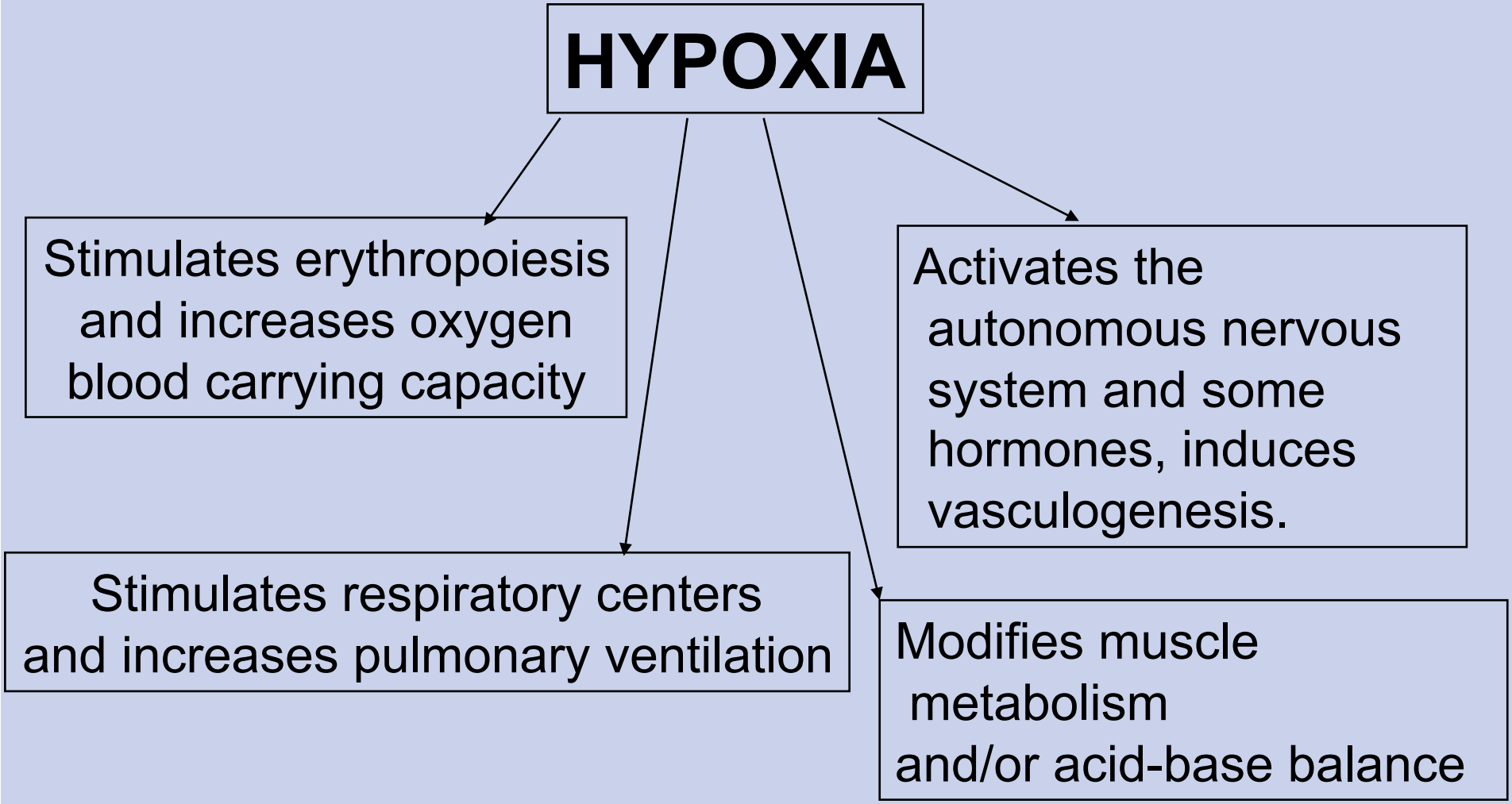
This decrease is more pronounced in aerobically trained subjects.

The decrease in arterial oxygen content is not compensated by an increase in cardiac output so that with increasing altitude, trained subjects loose their advantage of higher cardiac output.

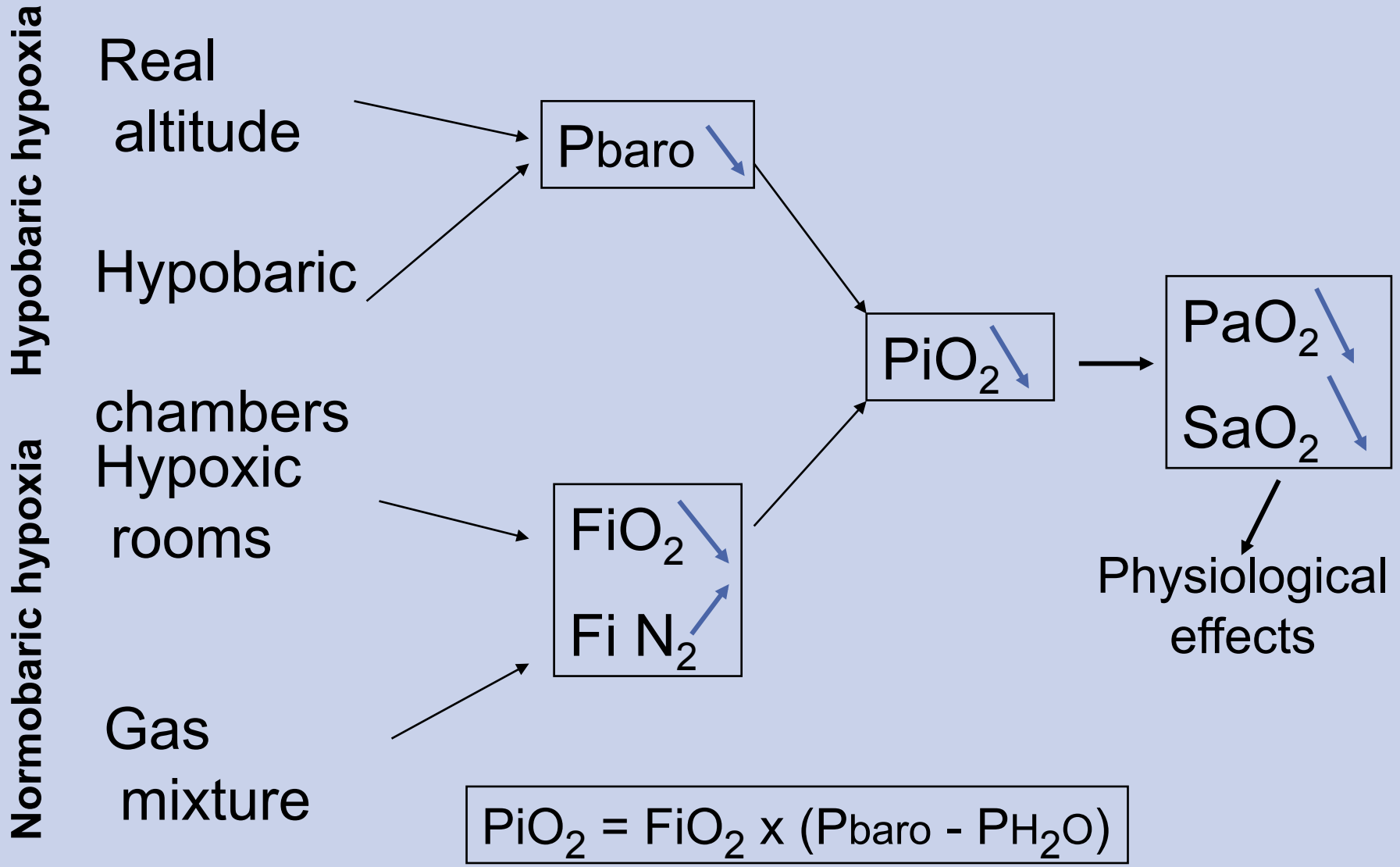
The capacity of the muscle to increase O_2 extraction to compensate the decrease in O_2 transport is compromised, especially in trained subjects.

The observed changes in VO_2max are fully explained by the physical constraints upon convective and diffusive processes involved in the O_2 transport cascade.

Why hypoxia for training / living ?

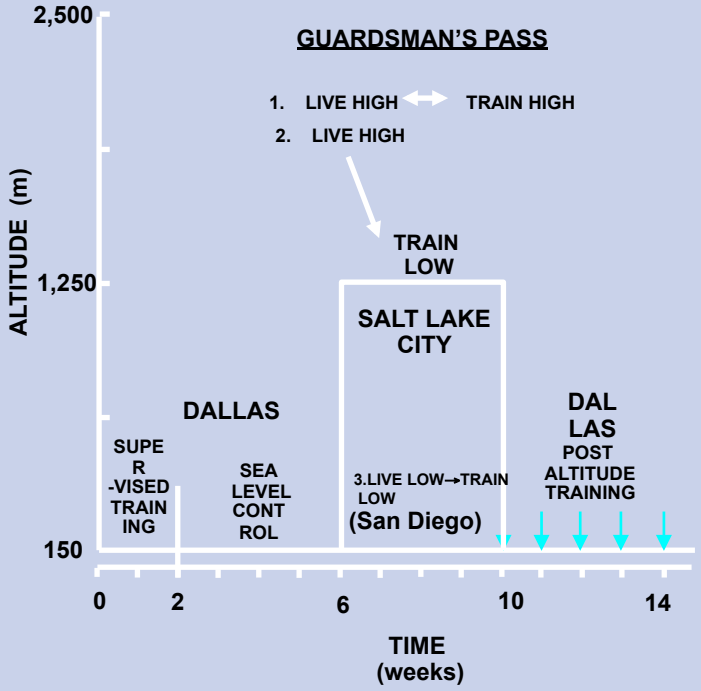


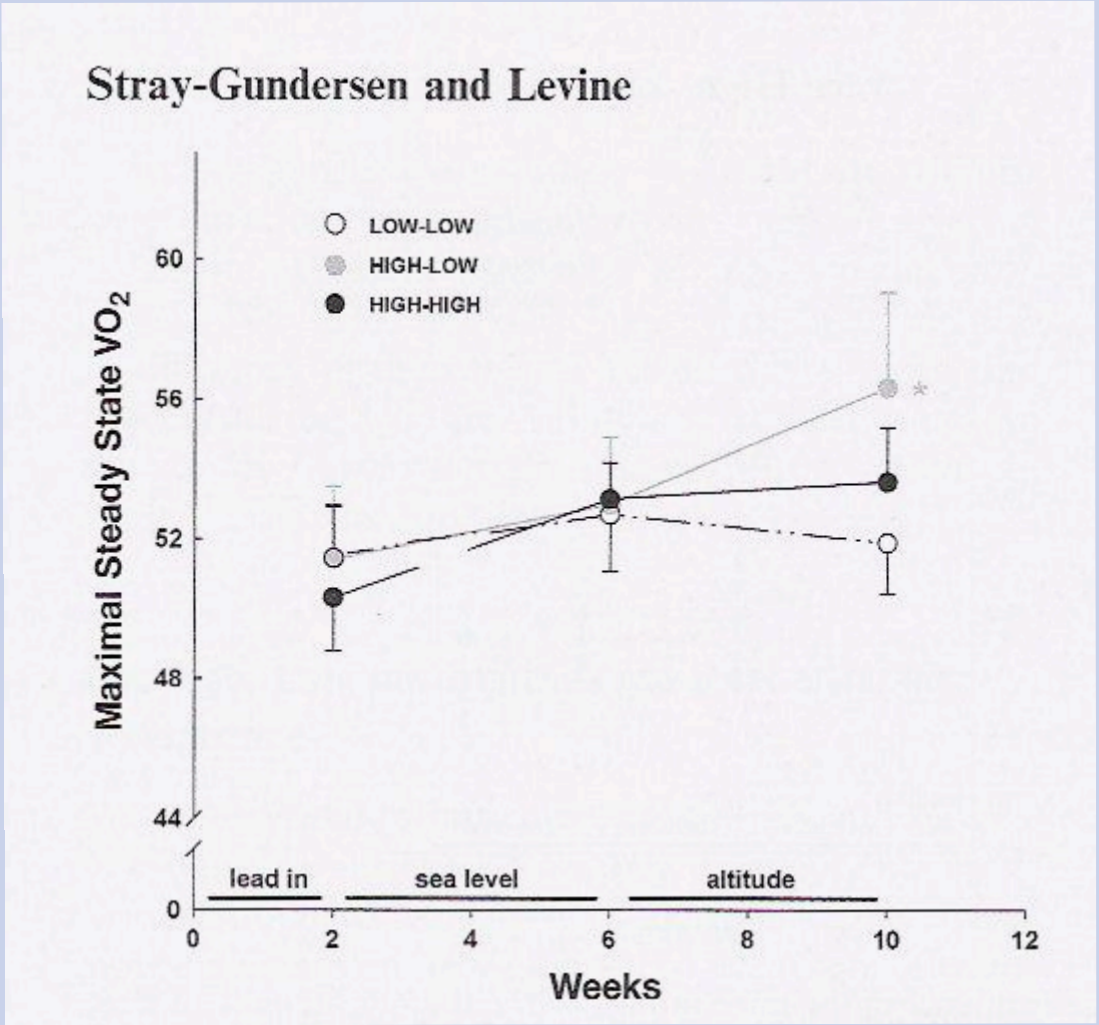
How to get a hypoxic environment ?



Real altitude: Levine et al. study (1994-1995)

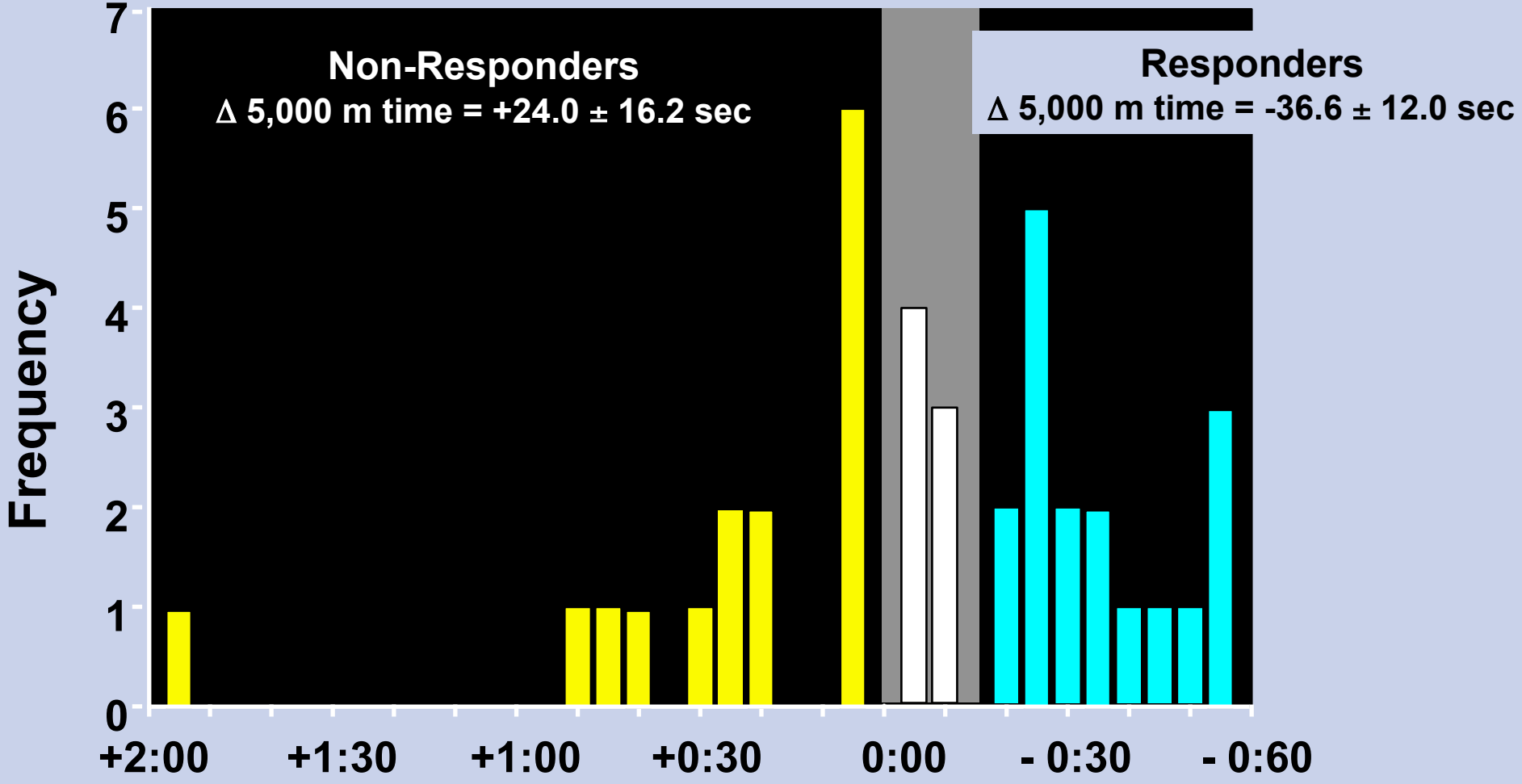
The “Live high - train low” model.





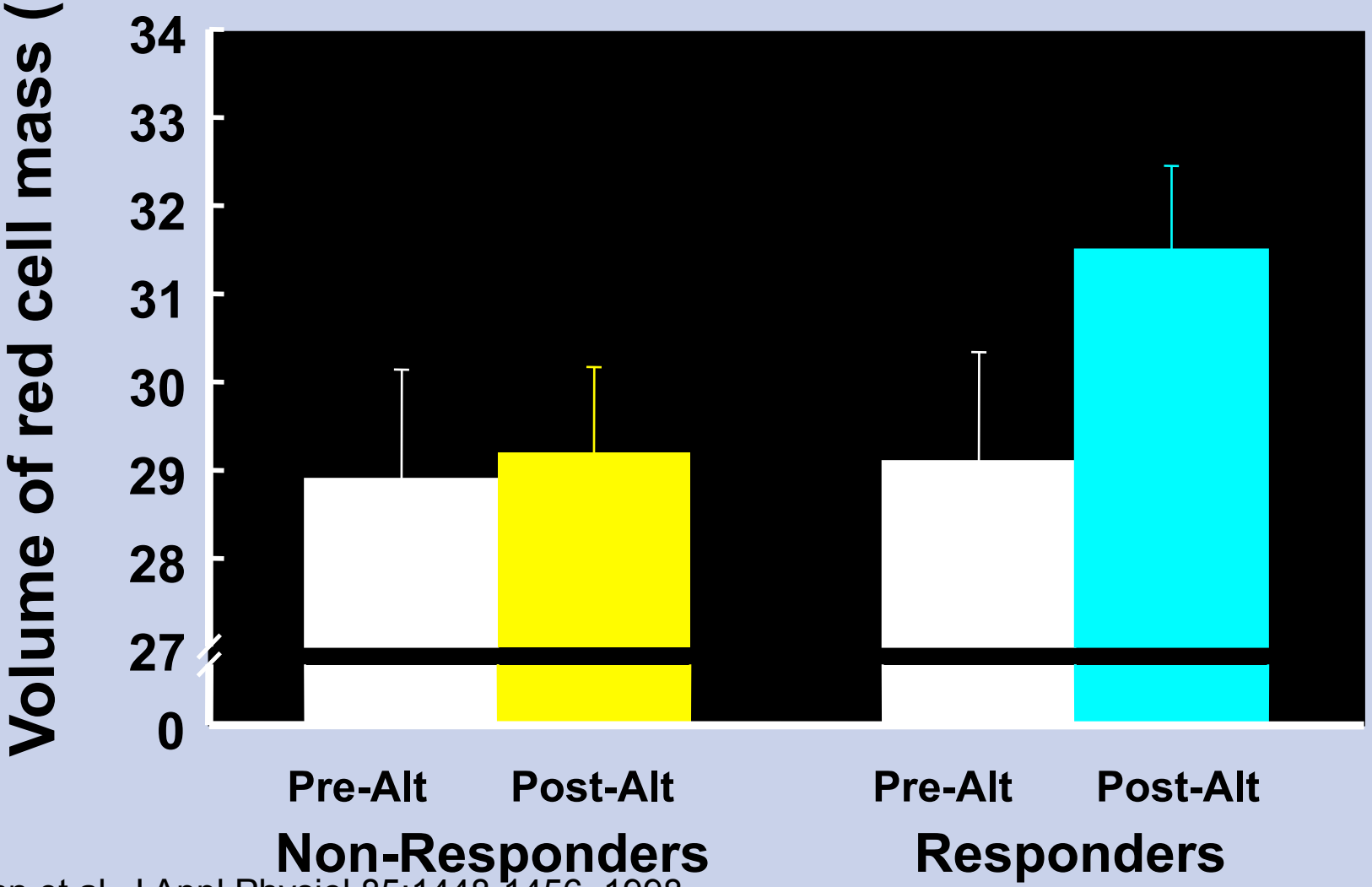
Levine et al, J Appl Physiol 82:102-112, 1997

Performance variation in response to 4 weeks of altitude training



Chapman et al, J Appl Physiol 85:1448-1456, 1998

Volume of red cell mass increases only in “responders”



Chapman et al, J Appl Physiol 85:1448-1456, 1998

Genes and altitude I

Subjects with Acute Mountain Sickness or HAPE

- **Renin-angiotensin** system (A(1166)C and G(1517)T single-nucleotide polymorphisms in AT(1)R gene) *associated to HAPE in* Japanese, but not ACE-I/D polymorphism (Hotta, 2004)
- **ACE and Angio II receptor** (ACE (ACE(A-240T), dbSNP rs4291; and ACE(A2350G), dbSNP rs4343), the intronic Alu insertion in ACE (ACE I/D), and the SNP ATR(A1166C), (dbSNP rs17231380) in AGTR1d) and AMS in Nepalese : *no association* (Koehle, 2006)
- **ACE, tyrosine hydroxylase, serotonin transporter [5-HTT], endothelial NO synthase [eNOS]** genes : *no association* to HAPE (Mortimer, 2004)

Genes and altitude II

Subjects with AMS or HAPE

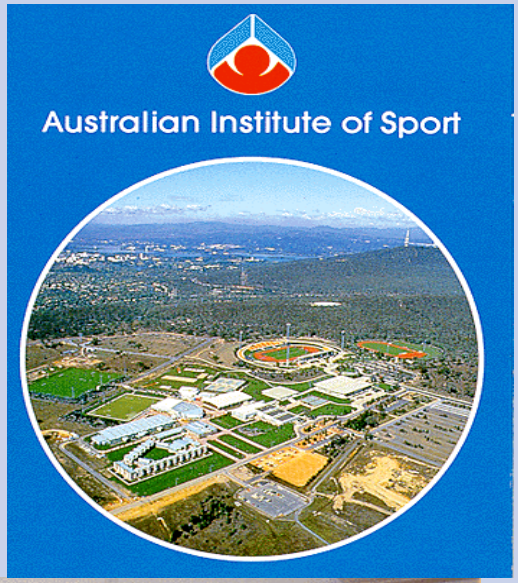
- **ACE** polymorphisms I/I, D/D, I/D : *no association* with AMS (Dehnert, 2003), polymorphism I/I more frequent in Kirghyz residents with PH (Aldashev, 2002), polymorphism D/D less frequent in COPD patients with RV hypertrophy (van Suylen, 1999)
- **eNOS**: (Glu298Asp variant and 27-base pair (bp) variable numbers of tandem repeats) *associates* to HAPE (Droma, 2003), wild alleles of the Glu298Asp and eNOS4b/a polymorphisms *more frequent* in Sherpas than in Nepalese (Droma, 2006)
- **Proteins A1 and A2 of the surfactant** (SP-A1 (C1101T, T3192C, and T3234C) and SP-A2 (A3265C)) *associated* to susceptibility to HAPE (Saxena, 2005)
- **hsp70-2 and hsp-hom** (+190 G/C, +1267 A/G, 2437 G/C) : *association* between hsp70-2 B/B, hsp70-hom A/B and B/B and AMS/HAPE in Chinese (Zhou, 2005)

Genes and altitude III

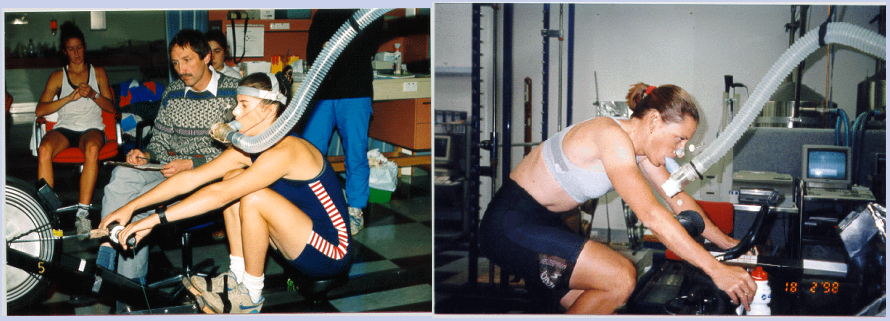
Athletes trained at altitude or sea level

- **ACE and EPO** in endurance athletes at moderate altitude : *no association* (Gonzalez, 2006)
- **ACE/DD** *associated* to LV hypertrophy in endurance athletes (Hernandez, 2003)
- **EPO** (marker D7S477), 3434 C --> T polymorphism in the 3' HRE sequence, and variability of erythropoietic response to altitude : *no association* (Jedlickova, 2003)

Normobaric hypoxia: the AIS Altitude House



Australian Institute of Sport, Canberra.



The partial pressure of oxygen in the house can be manipulated to allow altitude simulation.

Training in hypoxia in the endurance trained athlete

Effects on performance
Individual response factors
Potential risks for health

International Olympic Committee
Ministère des Sports, France
Groupe français de recherche sur l 'entraînement en hypoxie

COMITE INTERNATIONAL OLYMPIQUE

Main objectives

Evaluate the physiological changes induced by various modalities of training in hypoxia and their impact on performance.

- *Hypothesis: these methods improve performance at sea-level*

Evaluate the individual response to training

- *Hypothesis: there are biological, physiological or psychological markers of the variability of individual response to training in hypoxia*

Evaluate the potential risks for health

- *Hypothesis: these methods are safe at short, medium and long term, provided a medical control of training procedures*

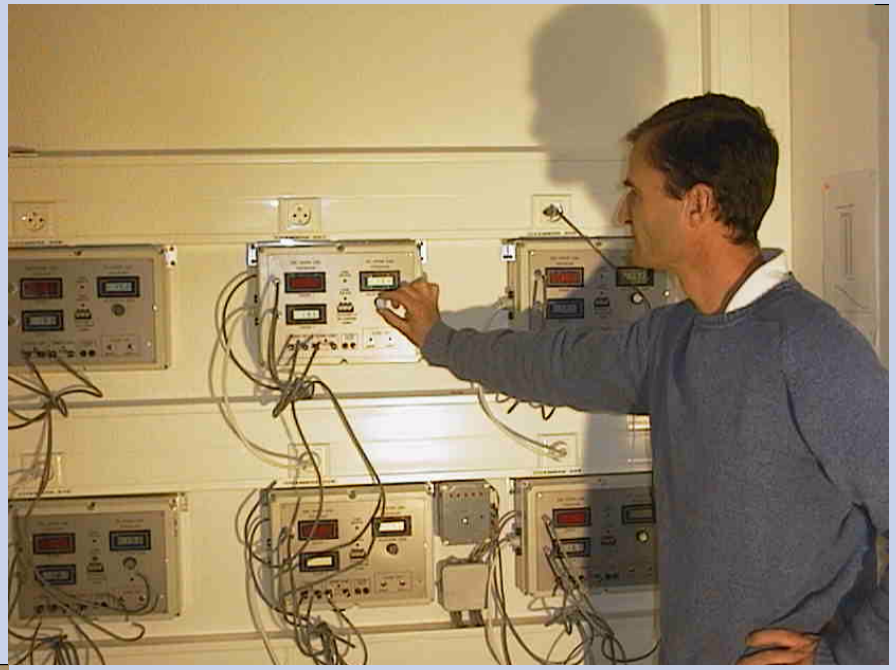
« Live/sleep high - train low »

Effects of intermittent exposure to hypoxia coupled to training at low altitude on performance in elite endurance athletes

(nordic ski, swimming, track and field, using hypoxic rooms)

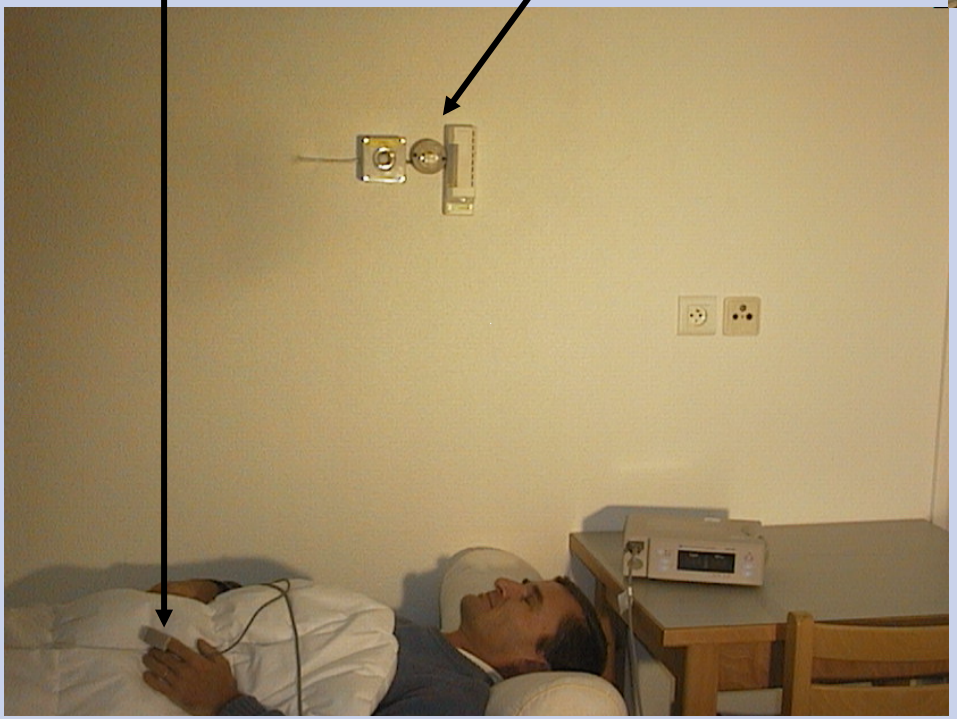
Centre National de Ski Nordique
Prémanon, Jura, France

HYPOXIC ROOM



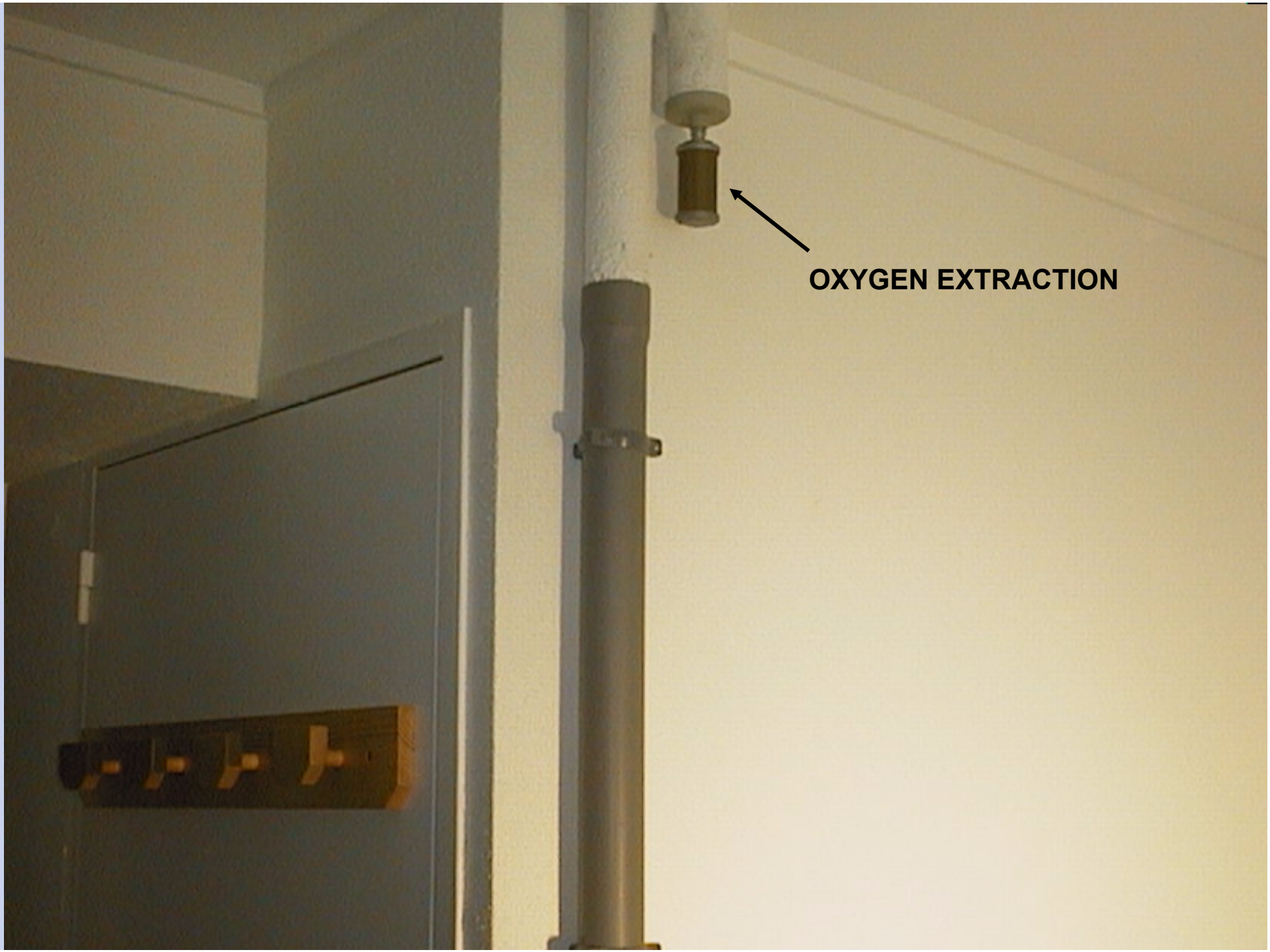
Control of arterial oxygen saturation

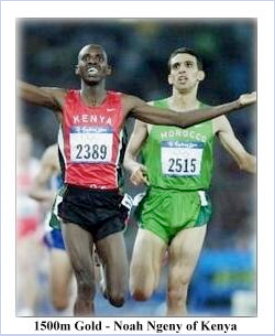
Control of ambient O_2 and CO_2



CONTROL CENTER with a physician during the night

Prémanon, Jura, France


OXYGEN EXTRACTION



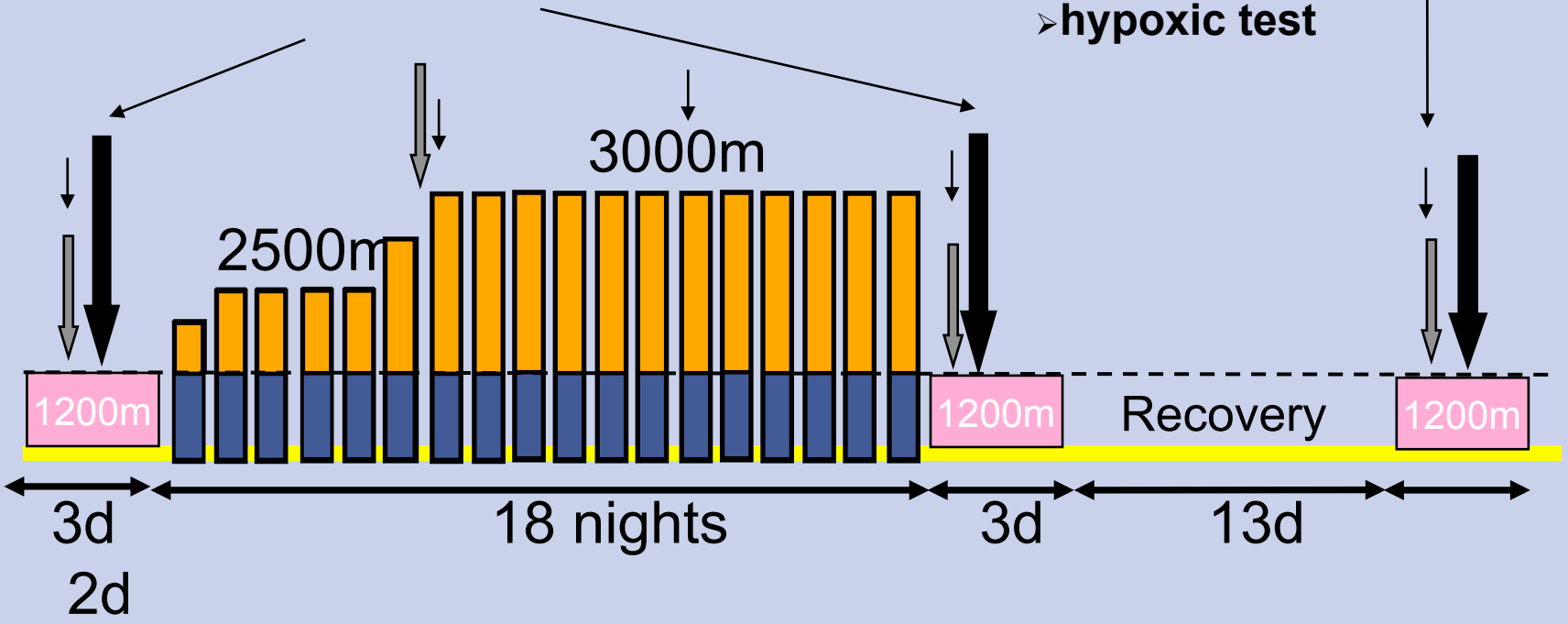
1500m Gold - Noah Ngeny of Kenya

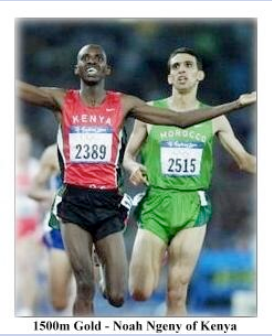
Track and field

Hypoxia
Control
Tests
Blood sampl. TDF

- VO_2max (treadmill)
- field test 10 min at 90% MAS.
- Blood sampl., PV
- TFM, TDF
- hypoxic test

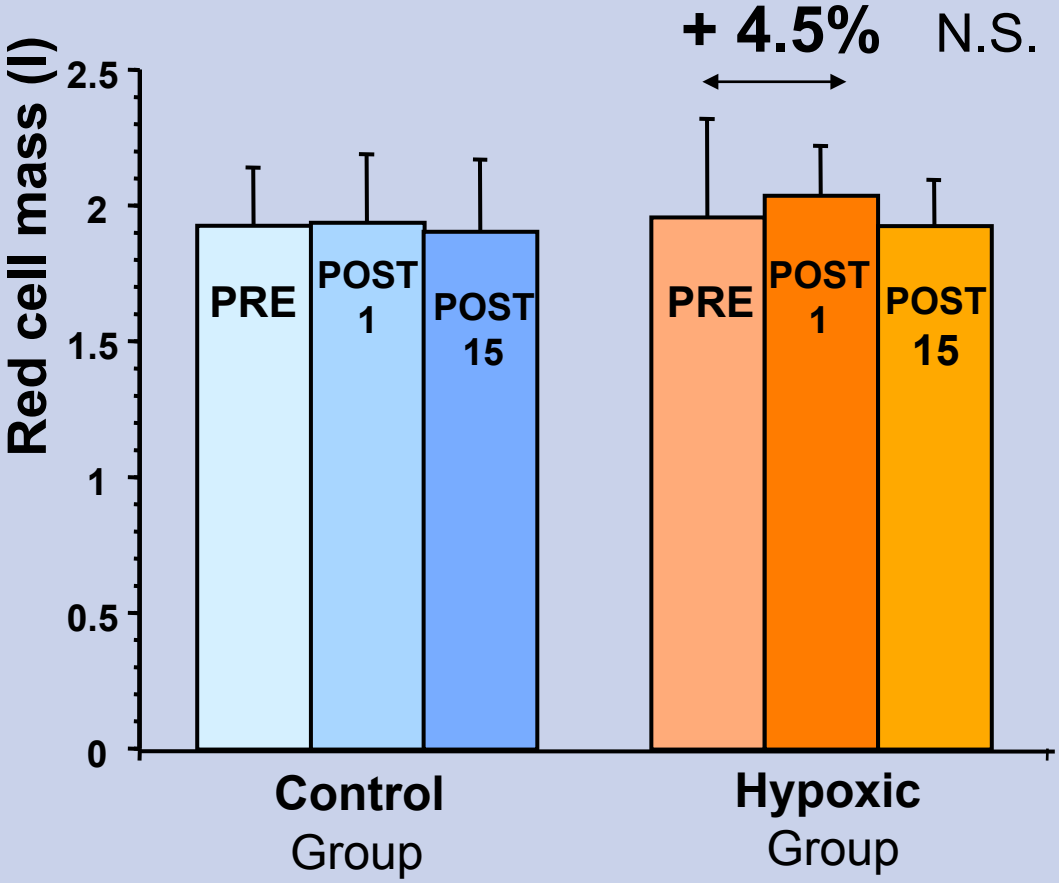
- VO_2max (treadmill)
- field test 10 min at 90% MAS
- Blood sampl., PV
- TFM, TDF
- hypoxic test

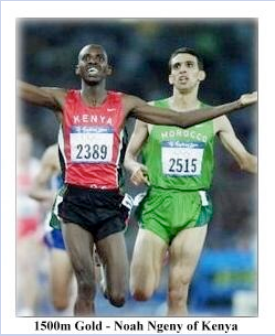




1500m Gold - Noah Ngeny of Kenya

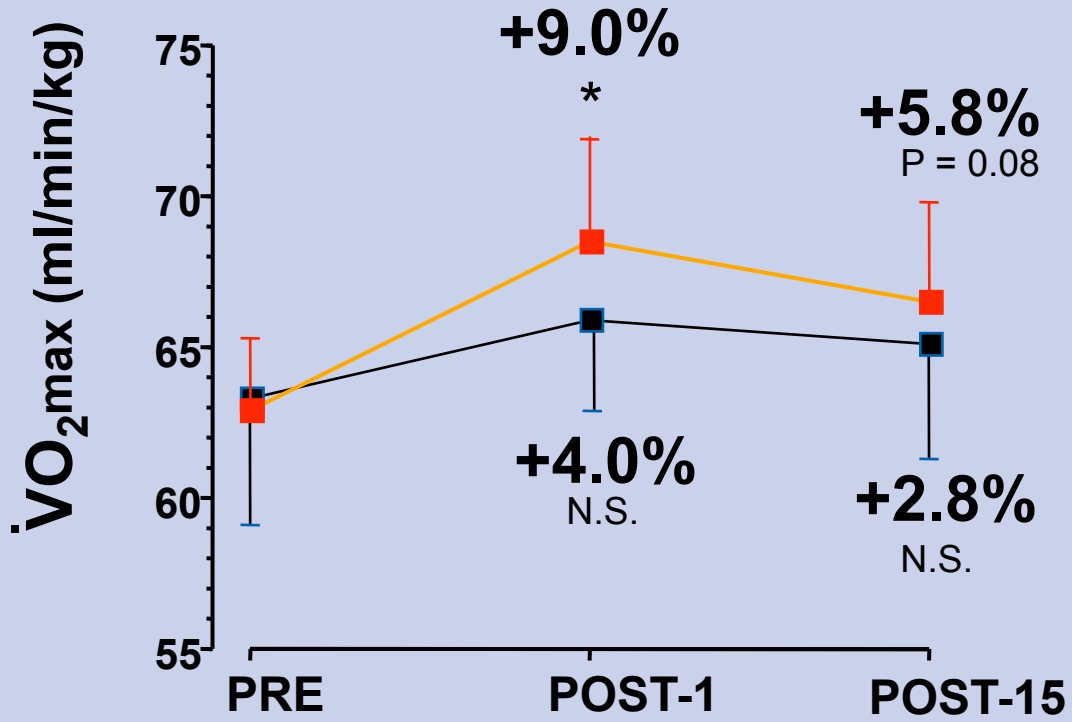
Red cell mass





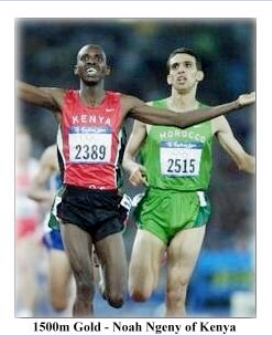
1500m Gold - Noah Ngeny of Kenya

Performance ($\dot{V}O_2max$)



- Control (n=6)
- Hypoxic (n=6)

* P<0.05 vs PRE



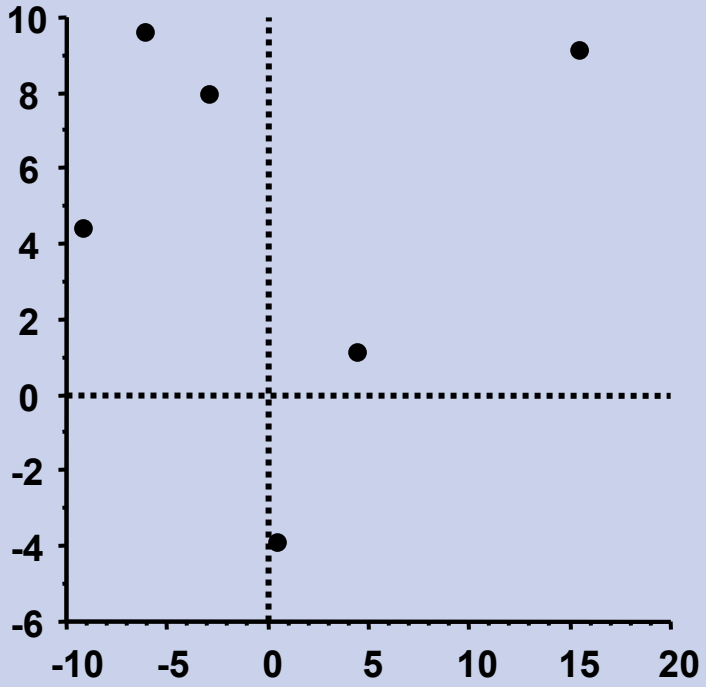
1500m Gold - Noah Ngeny of Kenya

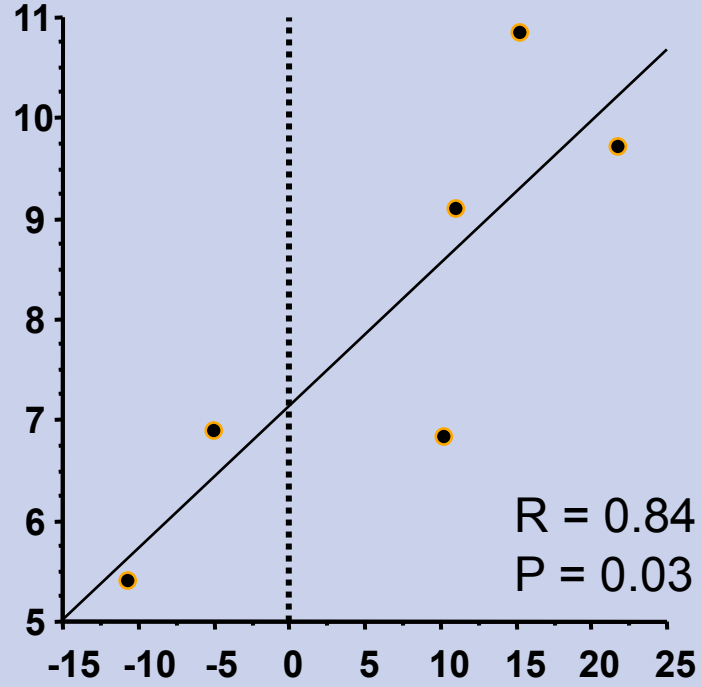
Variations of performance vs hemoglobin

Control group

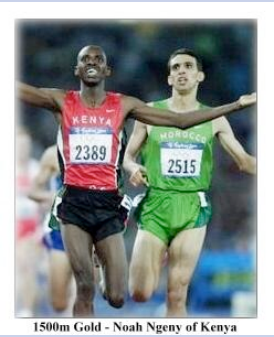
Hypoxic group

$\Delta \dot{V}O_2max$ (%) (POST1 – PRE)





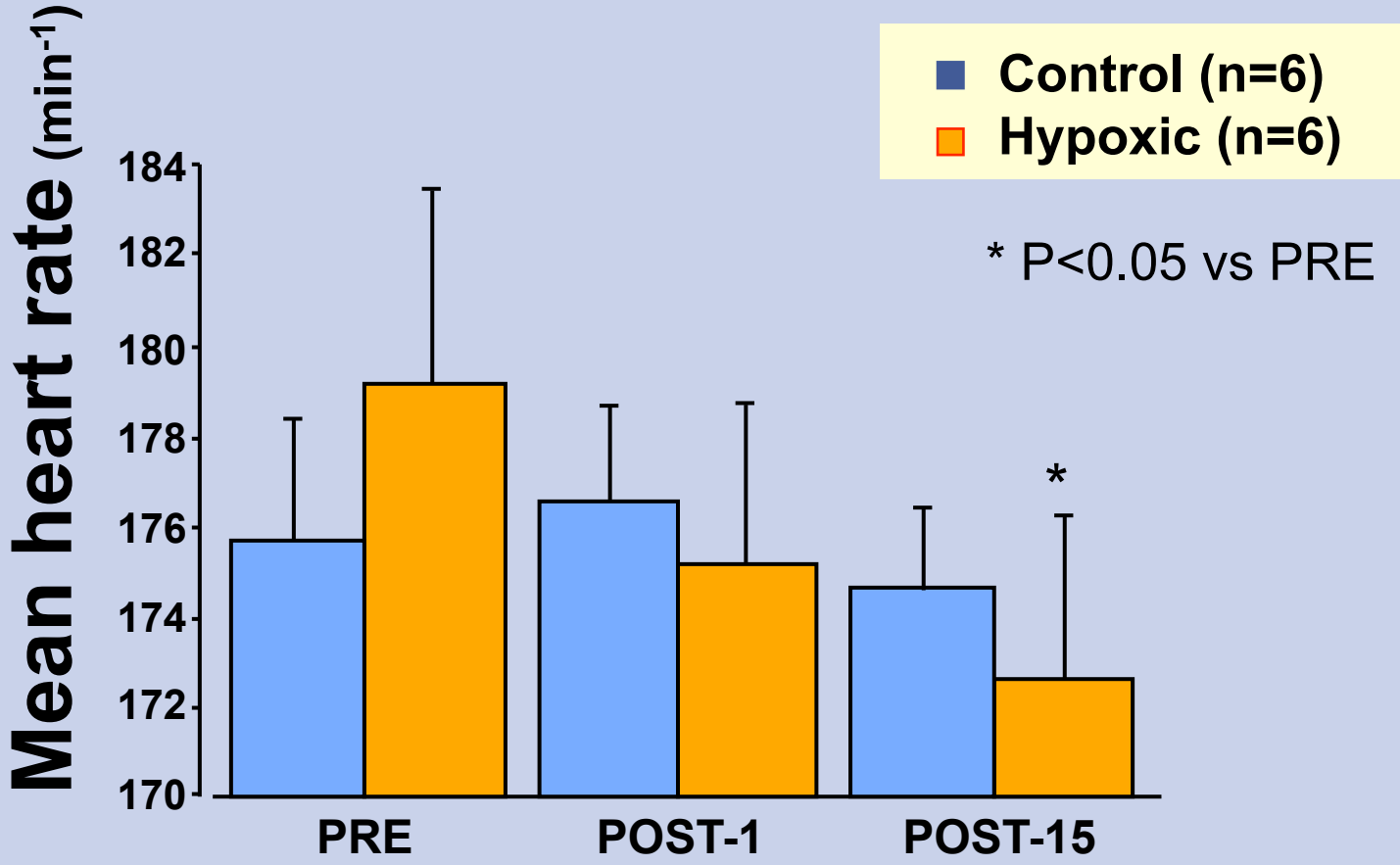
ΔnHb (%) (POST1 – PRE)



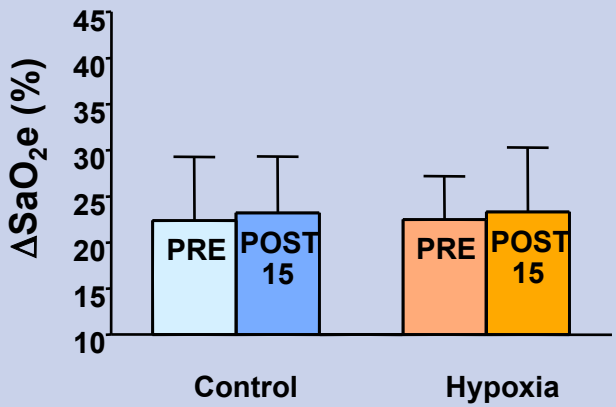
1500m Gold - Noah Ngeny of Kenya

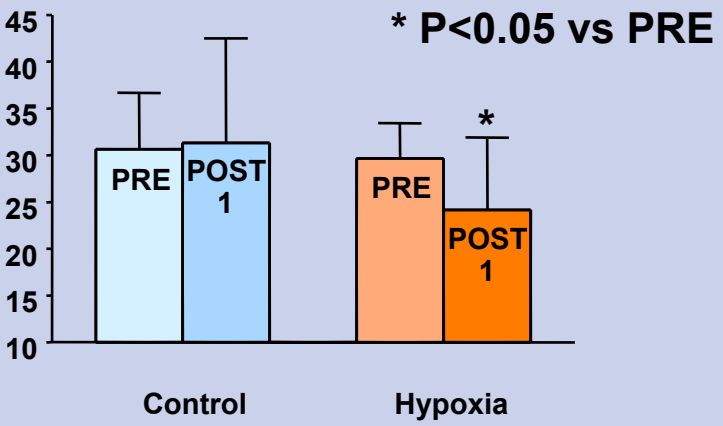
Field test: 10 min at 19.5 km/h

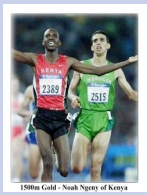
(≈ 90% of maximal aerobic speed)



Hypoxic exercise- induced desaturation (ΔSaO_2e)

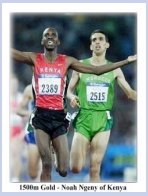






Lesser desaturation at exercise (ΔSaO_2e) at the end of the training session = sign of ventilatory acclimatization at exercice in hypoxia.

Nocturnal oxygen saturation (SaO_2)



1500m Gold - Noah Ngeny of Kenya

Sleep in hypoxic chambers induces episodes of desaturation, without apparent consequences on athlete's health

Tolerance and acclimatization

« Live high – train low » (3000/1200)

- does not induce symptoms of Acute Mountain Sickness.
- may induce sleep perturbations and fatigue (if ≥ 3500m and training load not reduced)
- may induce sleep apneas in some subjects, without apparent clinical consequences during the day.

Tolerance and acclimatization

« Live high – train low » (3000/1200)

- is not dangerous for the health of the athlete
- induces a ventilatory acclimatization (lower desaturation at exercise in hypoxia) that fades away 15 days after the training session

Recommendations for « live /sleep high - train low »

Altitude: 2500 - 3000
Duration: 3 weeks
Daily hypoxic exposure: 12-14 hours
Reduce training load during the first 3 days
Control nocturnal O_2 saturation
Control training post hypoxic exposure

Reference: *Scand. Journal Med. & Science in Sports Vol 18, suppl 1, August 2008*

Thanks to:

Julien Brugniaux
Jérémy Cornolo
Laurent Schmitt
Paul Robach
Nicolas Bourdillon
Pascal Mollard
Xavier Woorons
Murielle Letournel
Christine Lamberto
Laboratoire “Réponses cellulaires et fonctionnelles à l’hypoxie” Université Paris 13
AP-HP Hôpital Avicenne, Bobigny, France