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7th International Congress
MOUNTAIN, SPORT & HEALTH
updating study and research from laboratory to field
9-10 November 2017 Rovereto (TN) Italy

Interaction between hypoxia exposure and exercise intensity

Aldo Savoldelli

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Why hypoxia?

At the origin:

- The ability to reach the top of Everest **with** (Hillary and Norgay, 1953) or **without** (Messner and Habeler, 1978) an **external oxygen supply** (West, 1983).
- 1990s new methods and devices for pursuing performance enhancement (at sea level) through altitude training (Levine BD, 1991).

More recently:

- Sprint training in Hypoxia (RSH) – for team sport athletes (Brocherie, 2017)
- Therapeutic Use of Exercising in Hypoxia (Millet GP, 2016; Hobbins, 2017)
- Hypoxia induced by voluntary hypoventilation (Woorons, 2017; Trincat, 2017)
- Resistance training in hypoxia (RTH) to improve muscular size and strength (Ramos-campo D.J., 2017)

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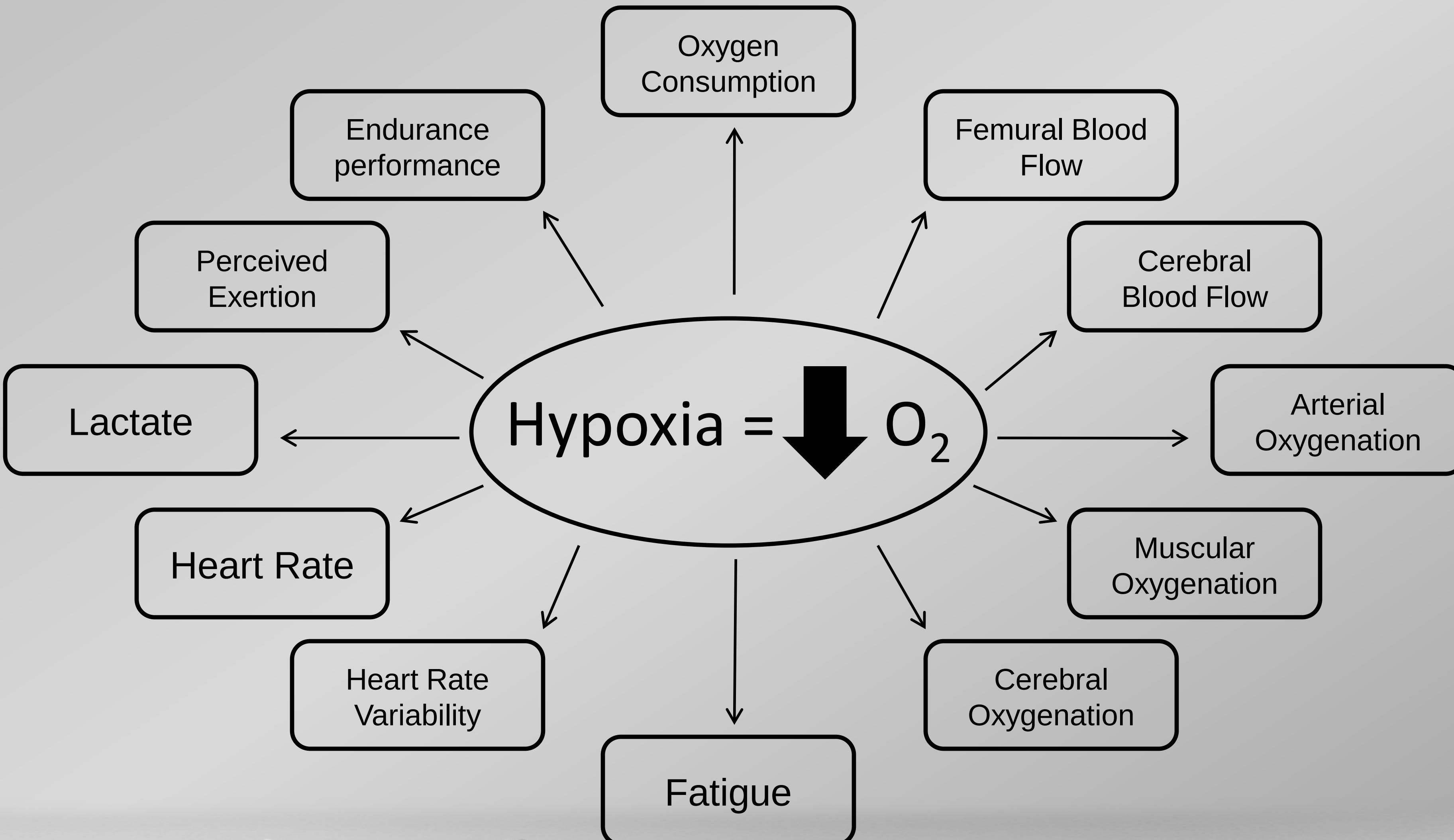
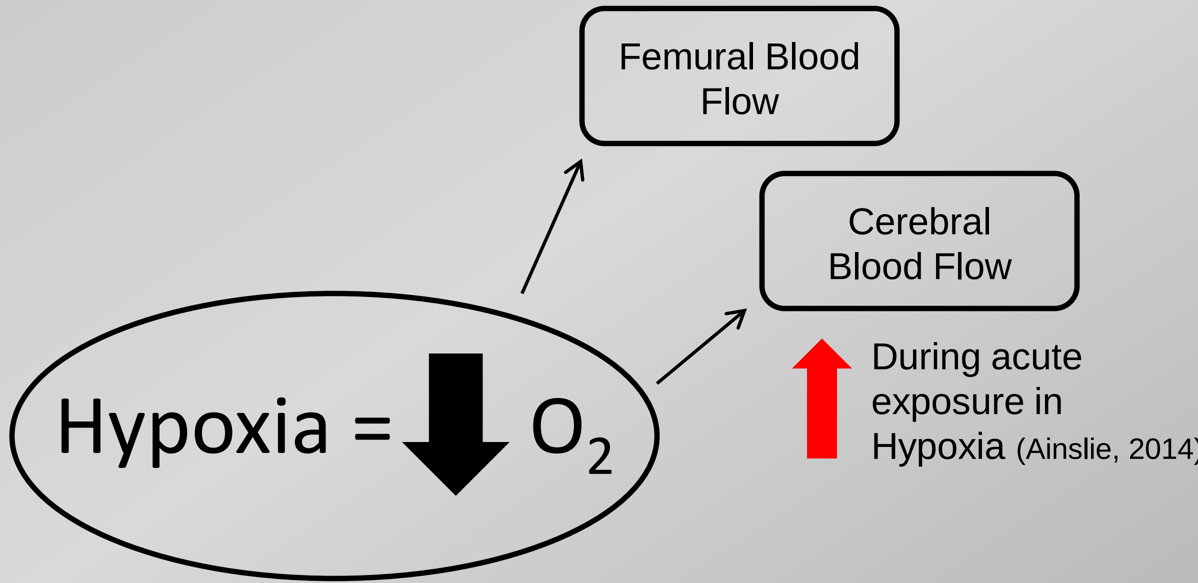
Overview




Femural Blood Flow
Cerebral Blood Flow
During acute exposure in Hypoxia (Ainslie, 2014)
Hypoxia = ↓ O2

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Hypoxia = ↓ O_2

→ Arterial Oxygenation

→ Muscular Oxygenation

→ Cerebral Oxygenation

↓ During early acute exposure in Hypoxia (Rupp, 2013)

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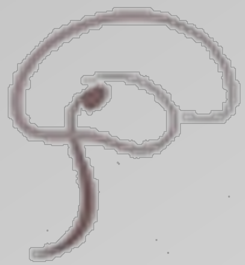
Hypoxia = ↓ O_2



Central

Fatigue

Peripheral

↑ During/After exercise in Hypoxia (Goodall, 2012)

✓ Any exercise-induced decline in maximal force or power produced by a muscle (Gandevia, 2001).

→ After exercise in Hypoxia (dependent on severity of hypoxia) (Amann, 2007)

Hypoxia = ↓ O_2

Heart Rate

Heart Rate Variability

↑ Sympathetic activity at rest during acute exosure at Hypoxia (Bucheit, 2004; Rupp, 2013)

Lactate ← Hypoxia = ↓ O_2

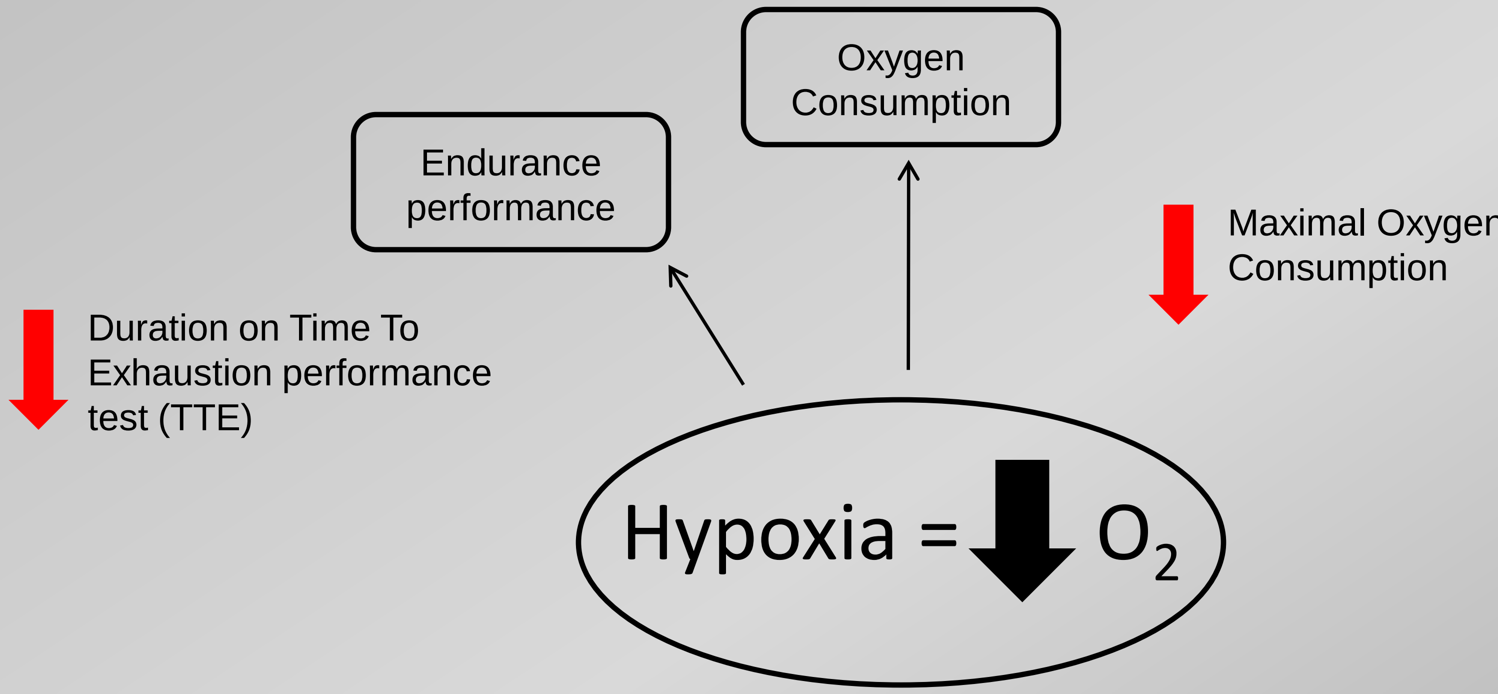
↑ During acute exercise in Hypoxia
(Richardson, 1998)

→ Lowered/unchanged during exercise after chronic exposure to Hypoxia in low natives
(Wagner & Lundby, 2007 / van Hall, 2009)
- “lactate paradox” ? (West, 2007; van Hall, 2007)

Perceived Exertion

Unchanged After maximal exercise in Hypoxia (Horstman, 1997)

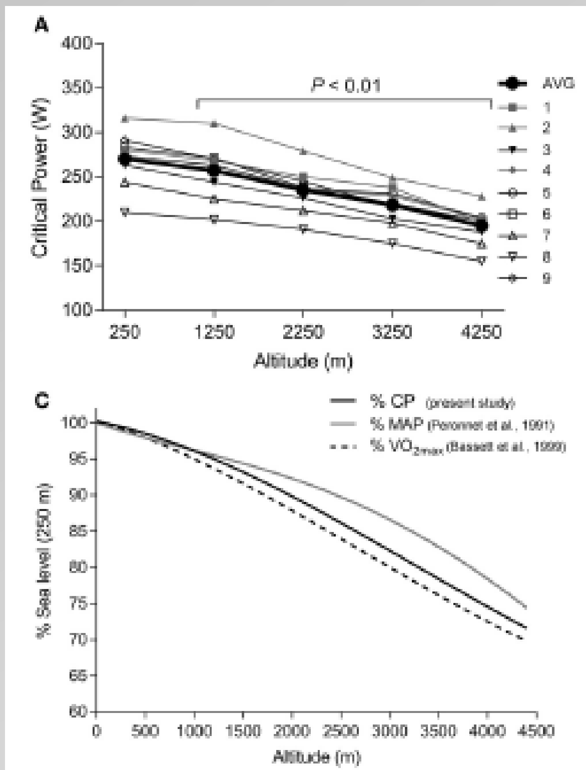
Hypoxia = ↓ O_2



(Wehrlin,2006)
(Calbet, 2003)
(Garvican-Lewis, 2015)

Competitions in Hypoxia

However, a lot of the endurance and ultra endurance performances take place in hypoxic environments
(Clark, 2007)



(modified from Townsend, 2017)

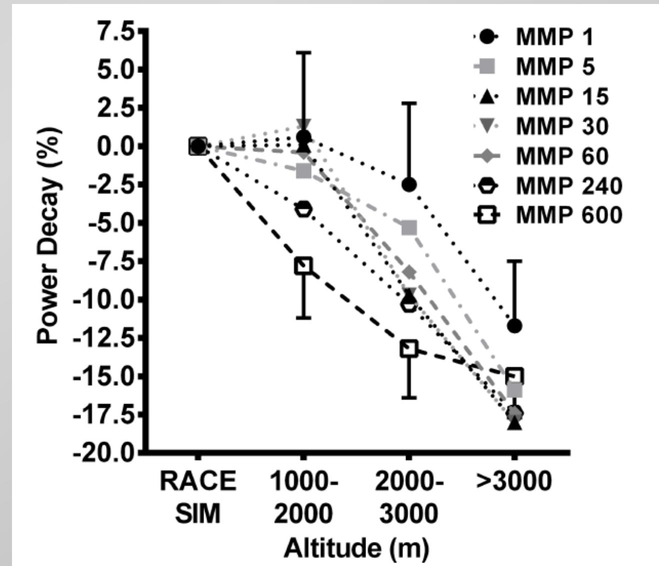
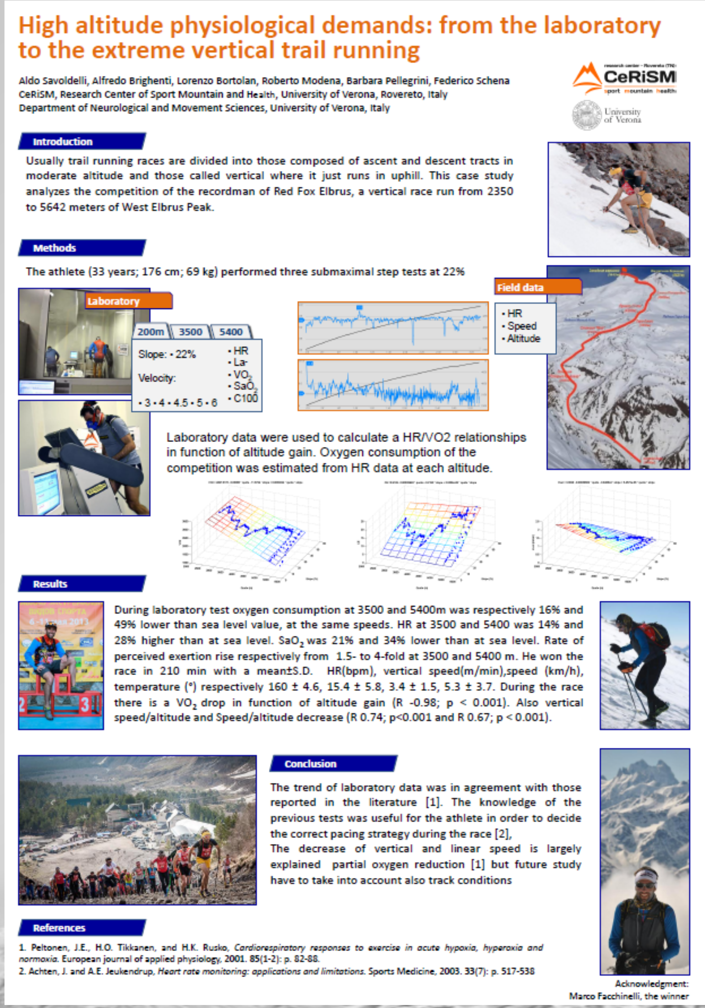


Fig 2. Rate of decline for specific mean maximal power as altitude increases. RACE SIM = Race simulation completed near sea-level in Australia.
doi:10.1371/journal.pone.0143028.g002

(Garvican-Lewis, 2015)

A question "from the field"



High altitude physiological demands: from the laboratory to the extreme vertical trail running

Aldo Savoldelli, Alfredo Brighenti, Lorenzo Bortolan, Roberto Modena, Barbara Pellegrini, Federico Schena
CeRiSM, Research Center of Sport Mountain and Health, University of Verona, Rovereto, Italy
Department of Neurological and Movement Sciences, University of Verona, Italy

Introduction

Usually trail running races are divided into those composed of ascent and descent tracts in moderate altitude and those called vertical where it just runs in uphill. This case study analyzes the competition of the recordman of Red Fox Elbrus, a vertical race run from 2350 to 5642 meters of West Elbrus Peak.

Methods

The athlete (33 years; 176 cm; 69 kg) performed three submaximal step tests at 22%

Laboratory data were used to calculate a HR/VO2 relationships in function of altitude gain. Oxygen consumption of the competition was estimated from HR data at each altitude.

Results

During laboratory test oxygen consumption at 3500 and 5400m was respectively 16% and 49% lower than sea level value, at the same speeds. HR at 3500 and 5400 was 14% and 28% higher than at sea level. SaO_2 was 21% and 34% lower than at sea level. Rate of perceived exertion rise respectively from 1.5- to 4-fold at 3500 and 5400 m. He won the race in 210 min with a mean±S.D. HR(bpm), vertical speed(m/min),speed (km/h), temperature (°) respectively 160 ± 4.6, 15.4 ± 5.8, 3.4 ± 1.5, 5.3 ± 3.7. During the race there is a VO_2 drop in function of altitude gain (R -0.98; $p < 0.001$). Also vertical speed/altitude and Speed/altitude decrease (R 0.74; $p<0.001$ and R 0.67; $p < 0.001$).

Conclusion

The trend of laboratory data was in agreement with those reported in the literature [1]. The knowledge of the previous tests was useful for the athlete in order to decide the correct pacing strategy during the race [2],
The decrease of vertical and linear speed is largely explained partial oxygen reduction [1] but future study have to take into account also track conditions

References

1. Peltonen, J.E., H.O. Tikkanen, and H.K. Rusko, *Cardiorespiratory responses to exercise in acute hypoxia, hyperoxia and normoxia*. European journal of applied physiology, 2001. 85(1-2): p. 82-88.
2. Achten, J. and A.E. Jeukendrup, *Heart rate monitoring: applications and limitations*. Sports Medicine, 2003. 33(7): p. 517-538

Acknowledgment:
Marco Facchinelli, the winner

A more detailed knowledge on hypoxia should help athletes and coaches who need to plan their challenge in altitude in order to improve also their pacing strategy, especially in Hypoxic Competitions (Clark, 2007).

Missing in Literature

It is **well known** which are the **demands at different altitudes.**

But, the determinants of a performance where the **severity of hypoxia change** during the exercise are still unclear.

⬇

Progressive Hypoxia

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Aim of the study

to better understand the effects and the determinants of:

3 different intensities at the same 1 hour Progressive Hypoxic exposure

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Methods

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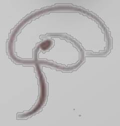
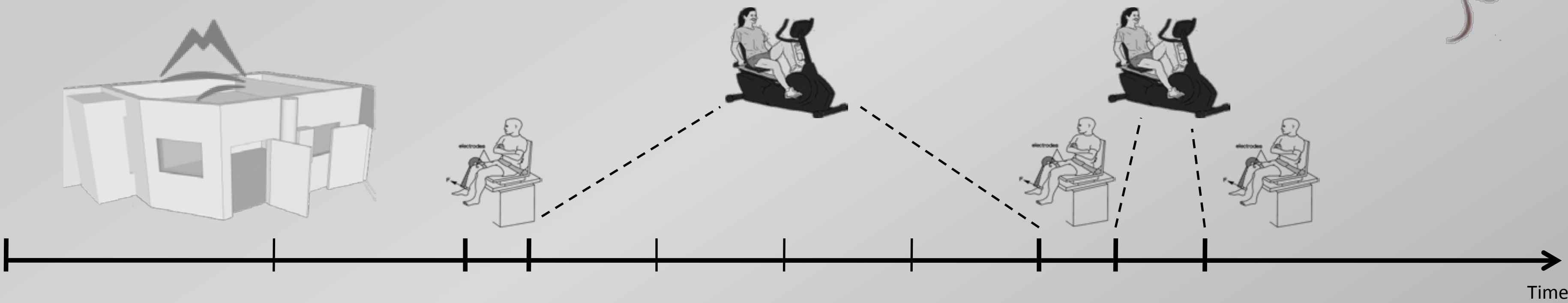
Two main focus in the data collection:

1) Determine the effects at: → Peripheral level; → Central level





2) Determine the effects: → Post and related to interventions; → Acute (during) interventions

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Materials

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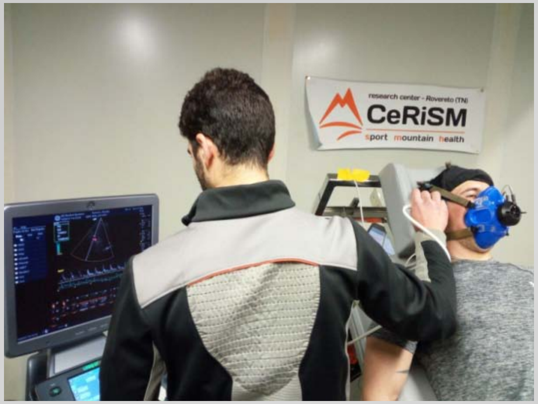
Ergometer, **Ergoline 1200**

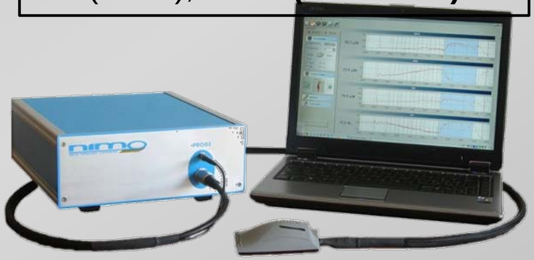
Doppler, **General-Electric S8**



Heart Rate monitor, **Polar 800 CX**

Strength ergometer, **Custom built at CeRiSM (Bortolan L.)**

Near Infra Red Spetroscopy (NIRS), **Nimo (2 channel)**



Oxygen consumption, **Cosmed CPET**

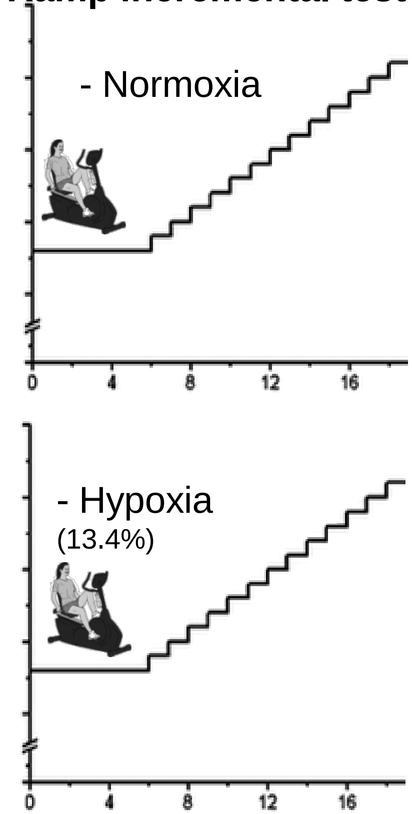
Perceived exertion, **CR100 Borg & Borg 1998**

Starting evaluations (Baseline)

13 endurance trained athletes (**11 eligible**)
Blind to the intervention conditions

Age (years):	32.1 ± 6.8
Weight (kg):	69.3 ± 6.5
%fat mass:	11.3 ± 3.2
Trainings/wk	3 to 5

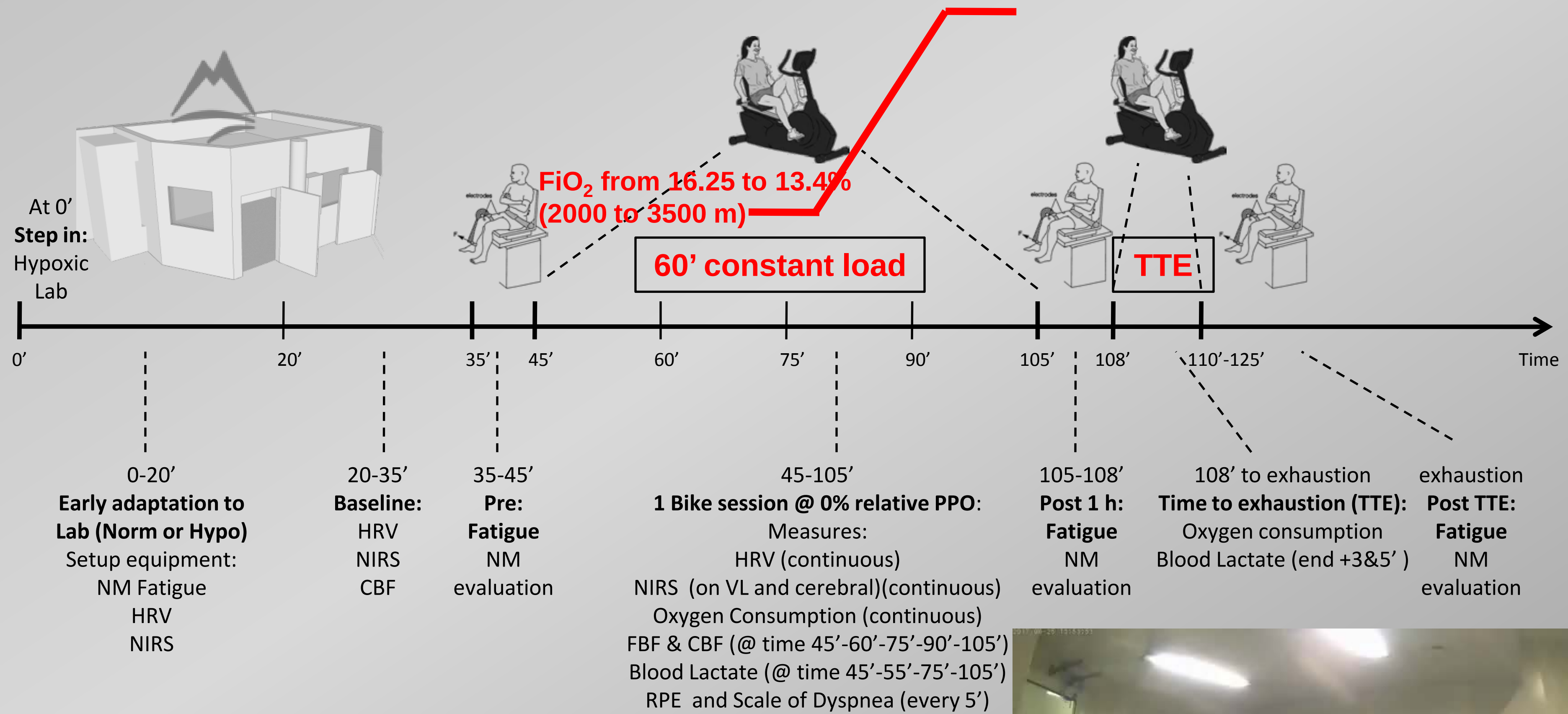
Ramp incremental test

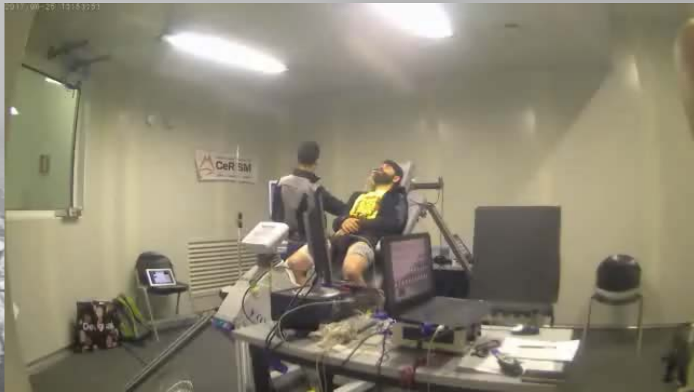


TTE at 80% of PPO - N
(247 ± 34 watt)
Normoxia (**FiO_2** 20.93%)

TTE at 80% of PPO - H
(211 ± 22 watt)
Hypoxia
(**FiO_2** 13.4%; ~ 3500 m asl)

The Protocol

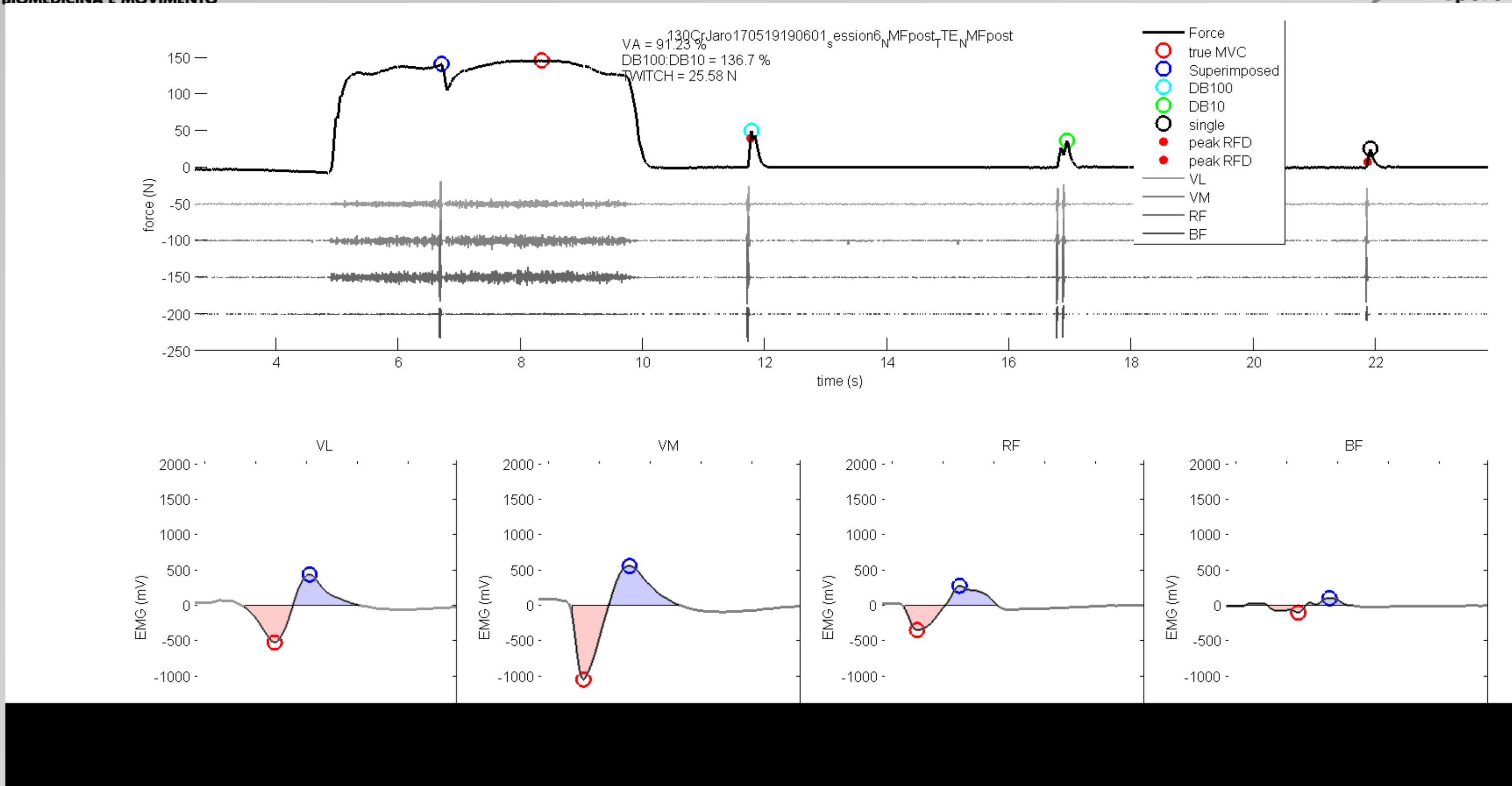


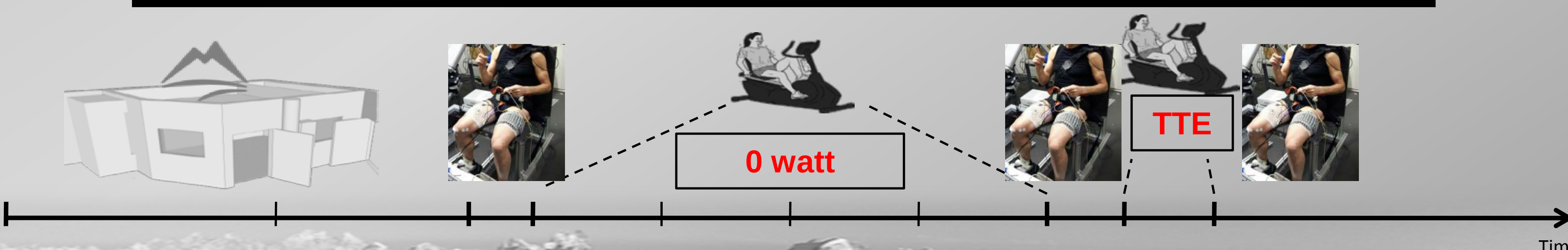


FiO_2 (%)= -0,047*time (min) + 16,16

Altitude (m) = 25*time (min) + 2000

The Protocol – NM fatigue





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Three sessions

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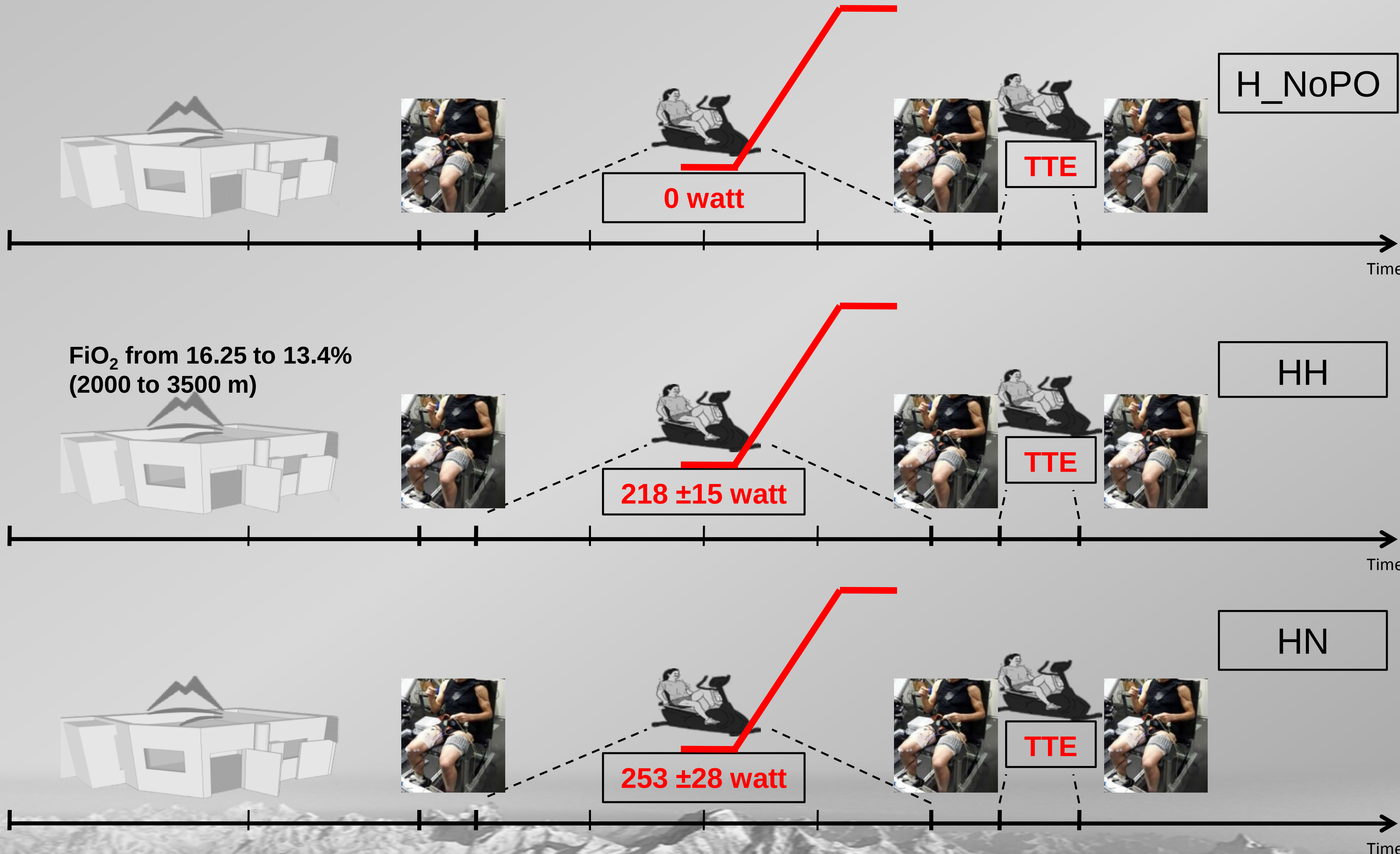
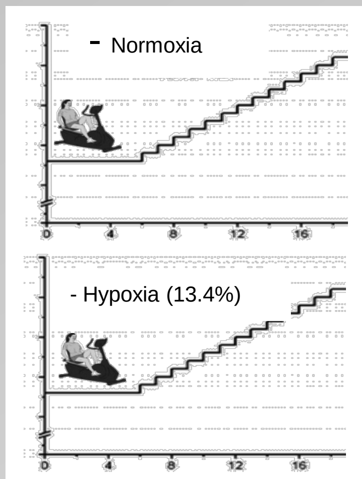


Results – starting (Baseline) evaluations

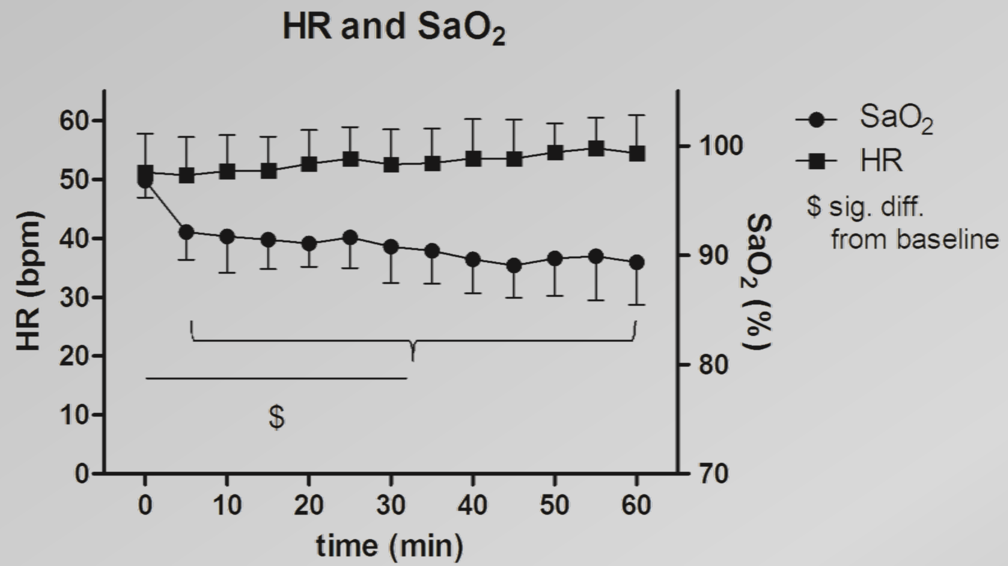


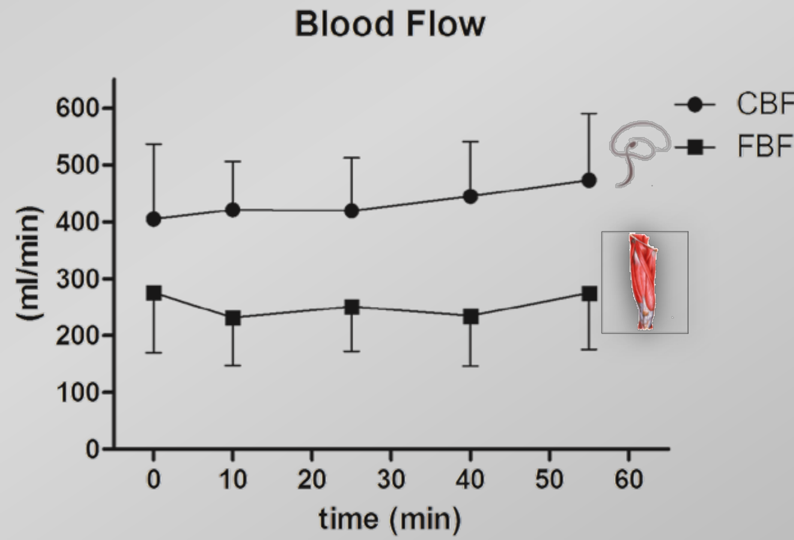
Ramp incremental test	Normoxia	Hypoxia	Difference (%)	*P value*
VO_{2max} ($mlO_2 \cdot min^{-1}$)	3996 ± 518	3172 ± 157	-20.6	*<0,001*
VO_{2max}/kg ($mlO_2 \cdot min^{-1} \cdot kg^{-1}$)	57.6 ± 6.3	46.5 ± 3.6		
Peak Power Output (watt)	316 ± 36	271 ± 19	-14.2	*<0,001*
VE ($L \cdot min^{-1}$)	156.6 ± 21.4	160.7 ± 12.4	2.6	*0.006*
Rate of Perceived Exertion (CR100)	96.4 ± 10.3	97.7 ± 5.6	1.4	*n.s.*
Blood $Lactate_{max}$ ($mM \cdot L^{-1}$)	12.5 ± 2.0	13.3 ± 1.7	7.1	*n.s.*
Heart $Rate_{max}$ (bpm)	180 ± 12	173 ± 12	-3.6	*n.s.*
Time to Exhaustion				
PowerOutput (watt)	253 ± 28	217 ± 16	-14.2	*<0.001*
Duration (sec)	643 ± 210	418 ± 86	-35.0	*0.003*
Blood $Lactate_{max}$ ($mM \cdot L^{-1}$)	10.1 ± 1.5	11.2 ± 1.9	11.9	*0.019*
RPE overall (CR100)	91.1 ± 4.6	95.7 ± 8.3	5.1	*n.s.*
RPE leg discomfort (CR100)	92.5 ± 6.4	96.5 ± 8.4	4.4	*n.s.*
RPE dyspnea (CR100)	85.8 ± 7.6	93.7 ± 12.6	9.2	*n.s.*

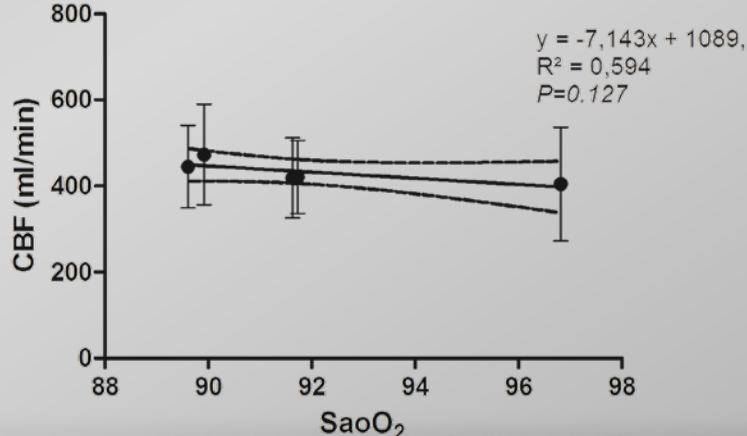
TTE at 80% of PPO - N
Normoxia
(**FiO_2** 20.93%)

TTE at 80% of PPO - H
Hypoxia
(**FiO_2** 13.4%, ~ 3500 m asl)

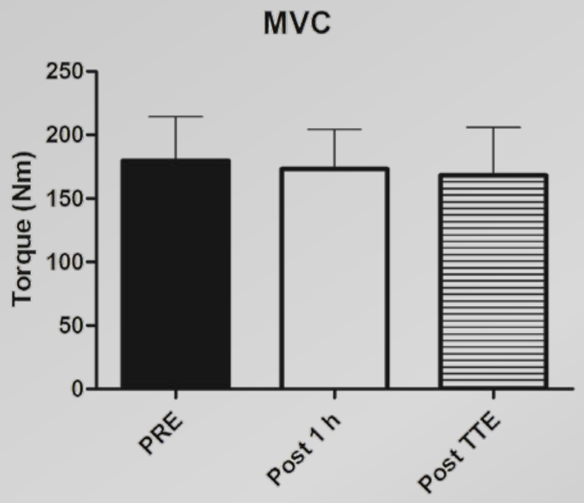
Results (during intervention) H_NoPO

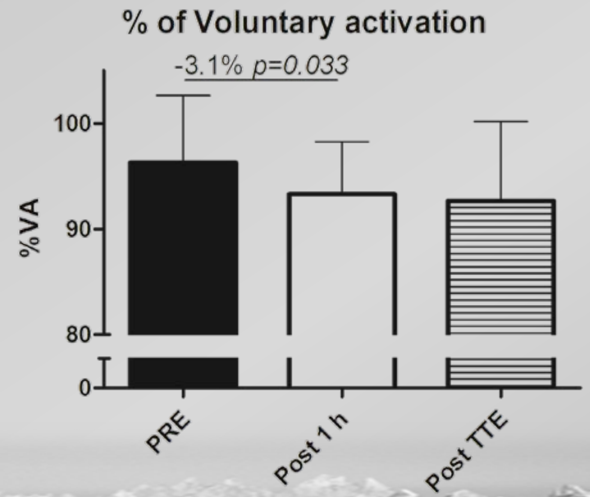


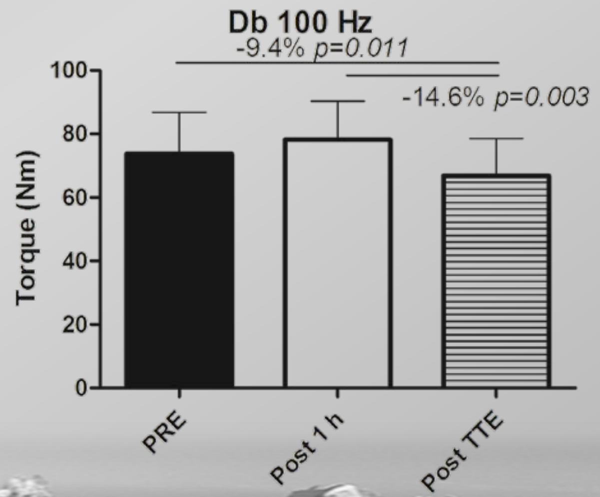


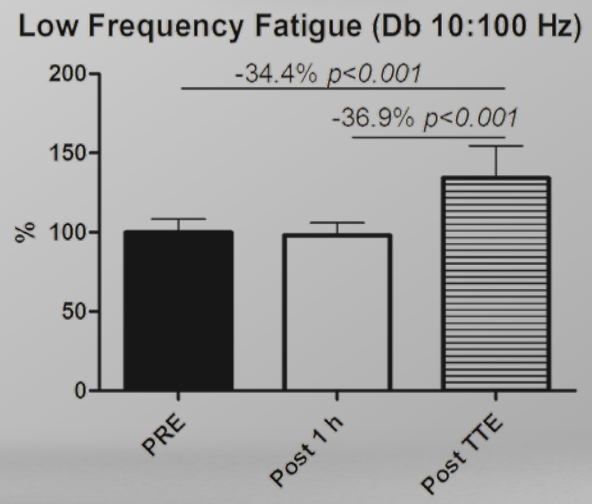


Results (pre to post changes) – H_NoPO

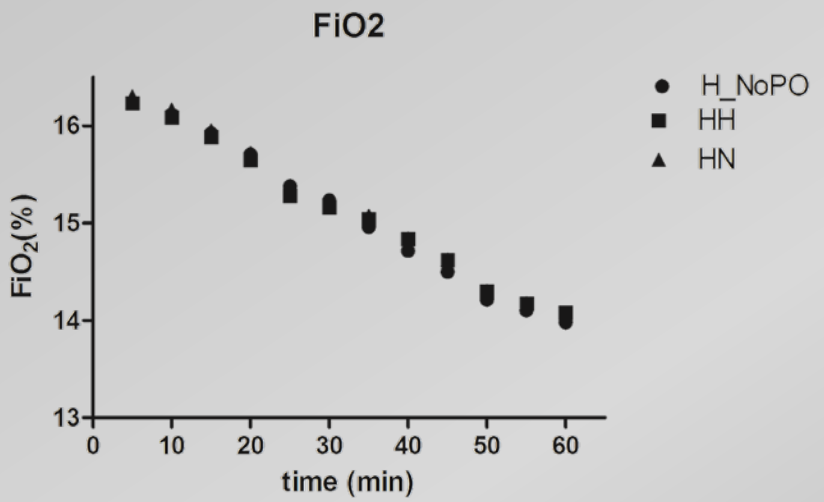


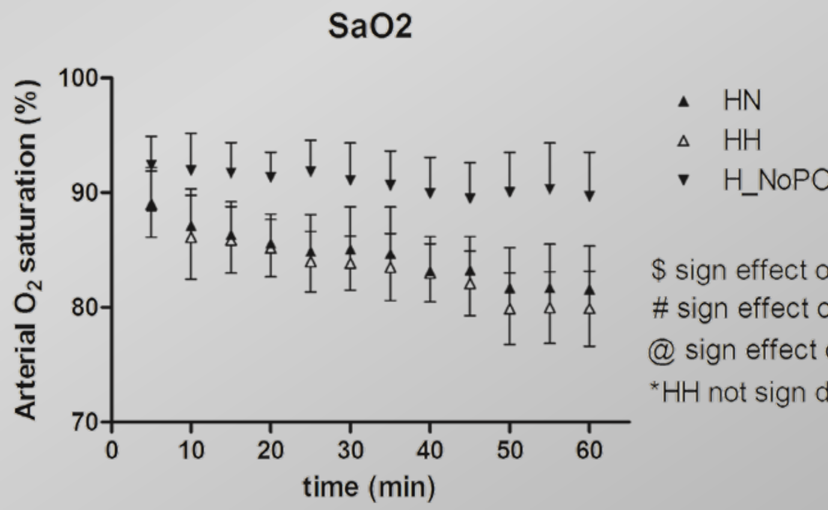






Results (during intervention)





$ sign effect of time
# sign effect of condition
@ sign effect of condition*time
*HH not sign diff from HN

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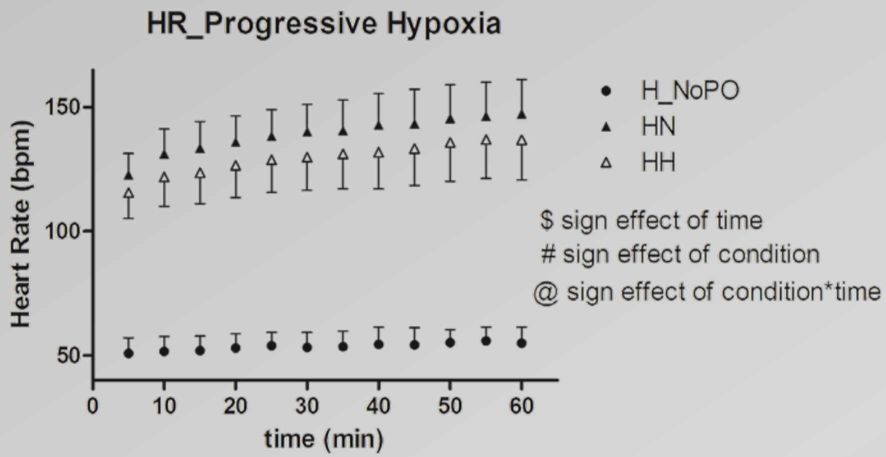
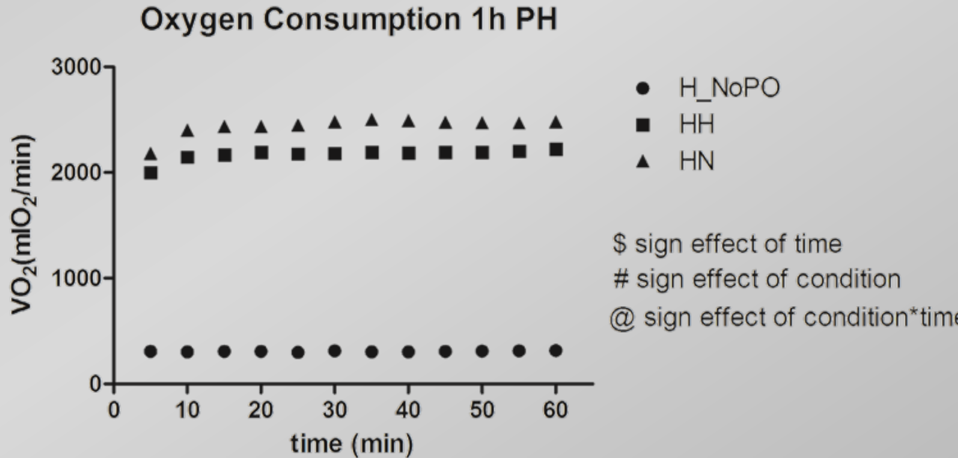
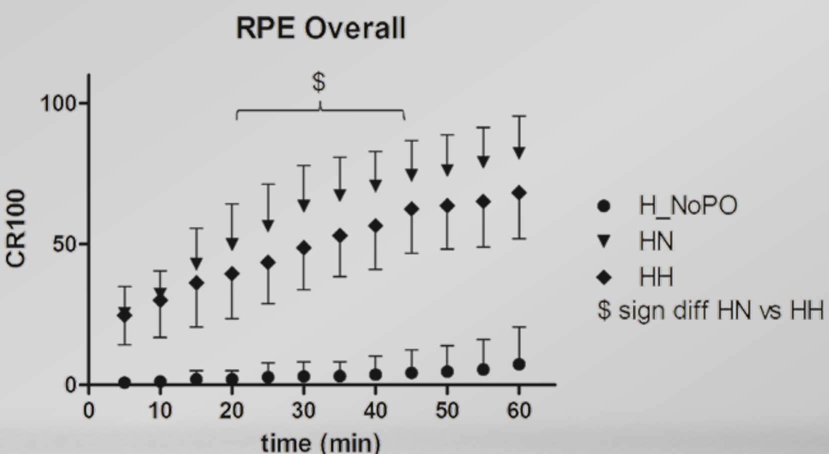
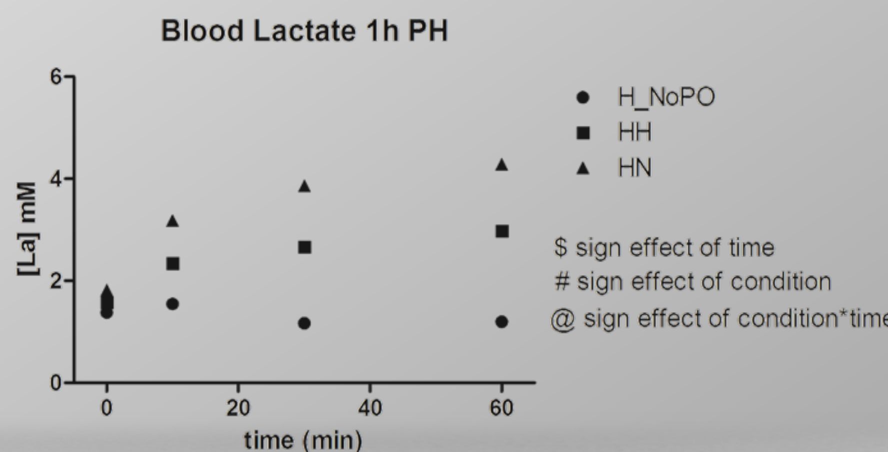
Results (during intervention)

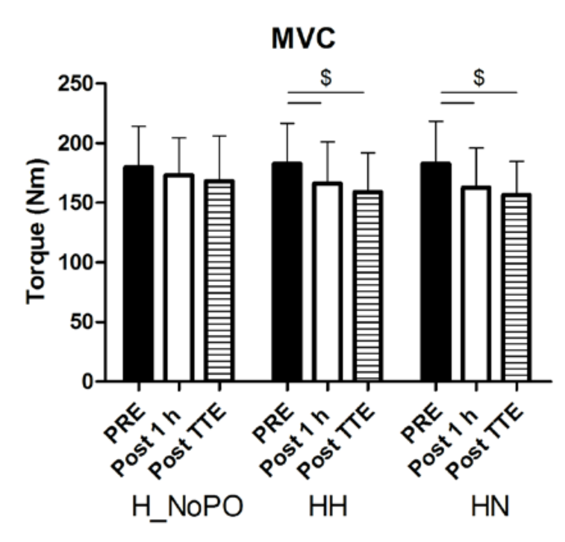


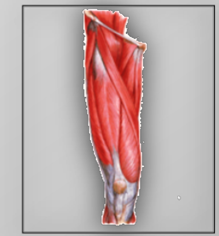


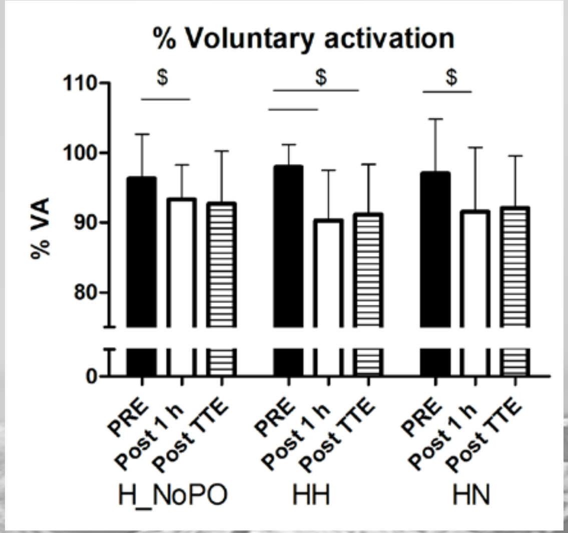


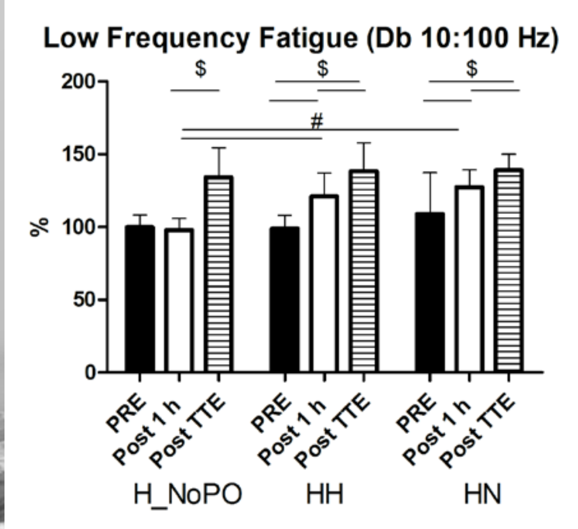


Results (pre to post changes) - Fatigue

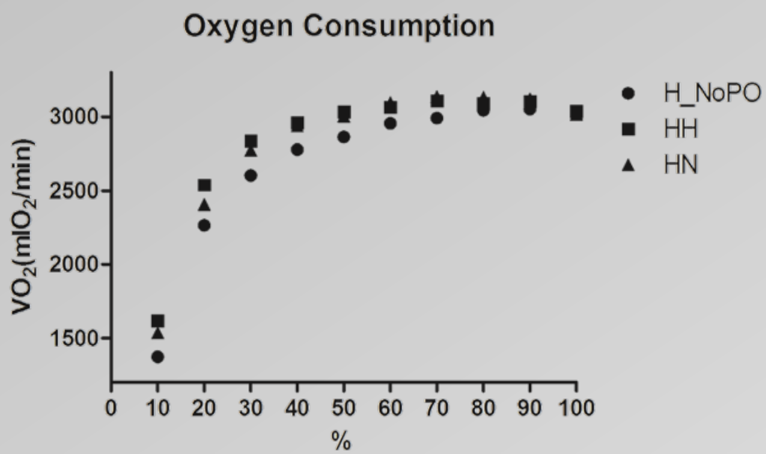


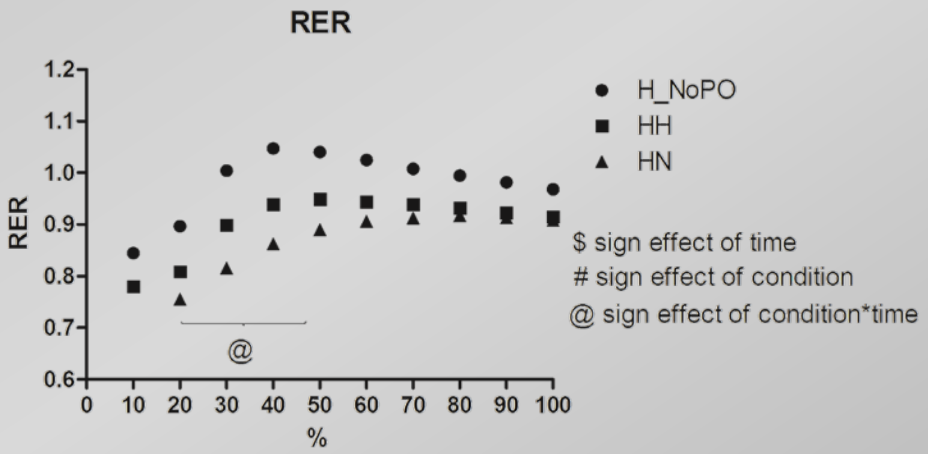


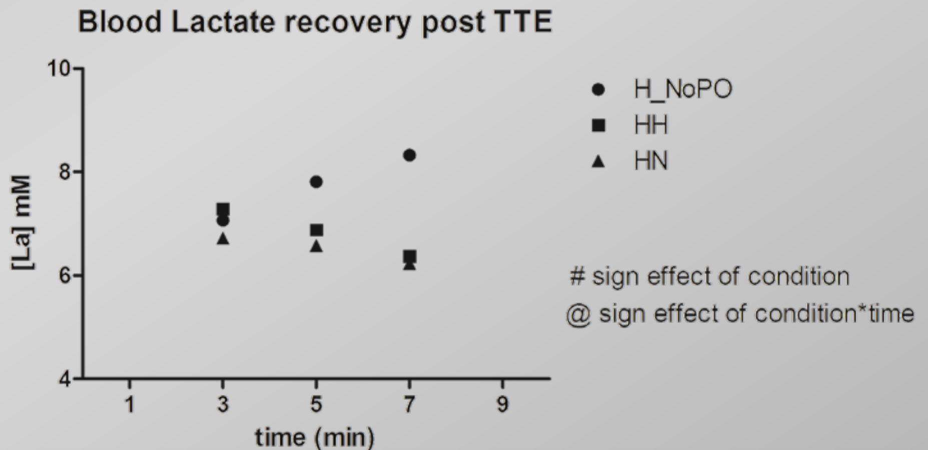




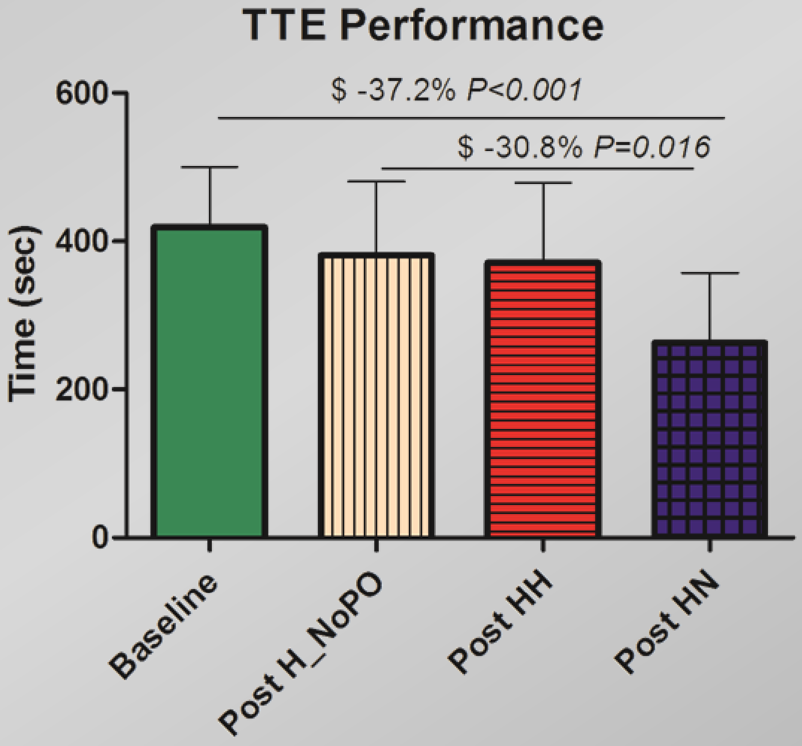
Results during TTE Performance







Results (pre to post changes) - Performance



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Discussion - fatigue

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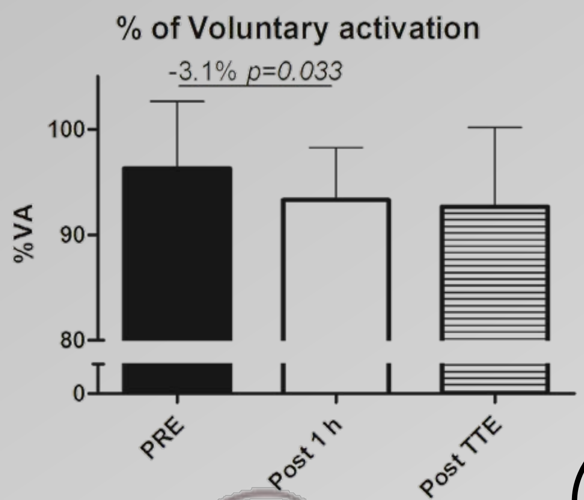
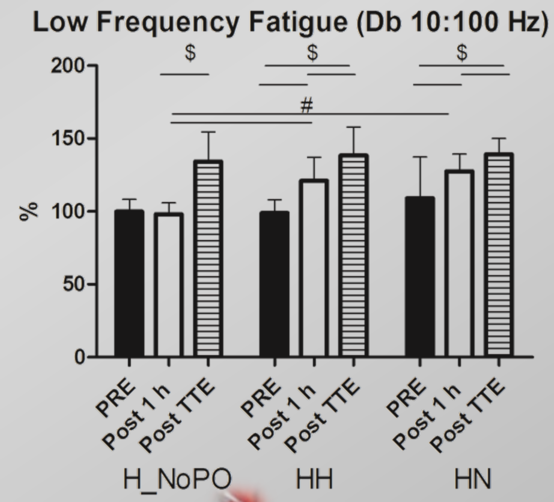
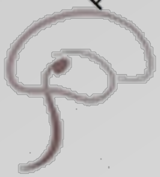




Hypoxia = ↓ O_2



Central

↑ During/After exercise in Hypoxia (Goodall, 2012)

Fatigue

Peripheral

→ After exercise in Hypoxia (dependent on severity of hypoxia) (Amann, 2007)

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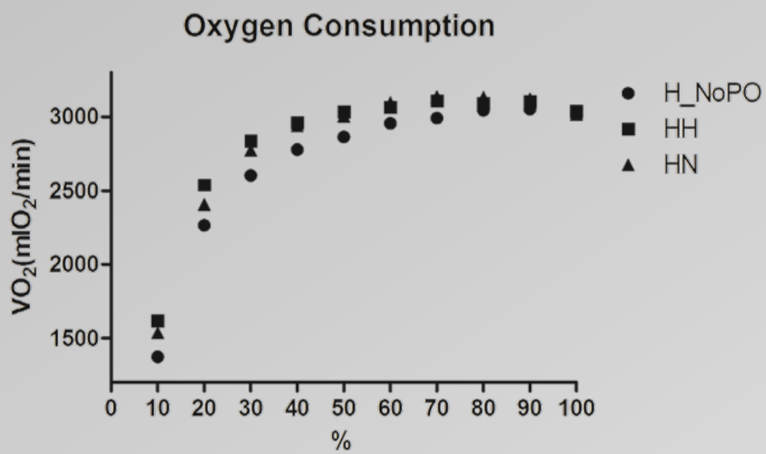
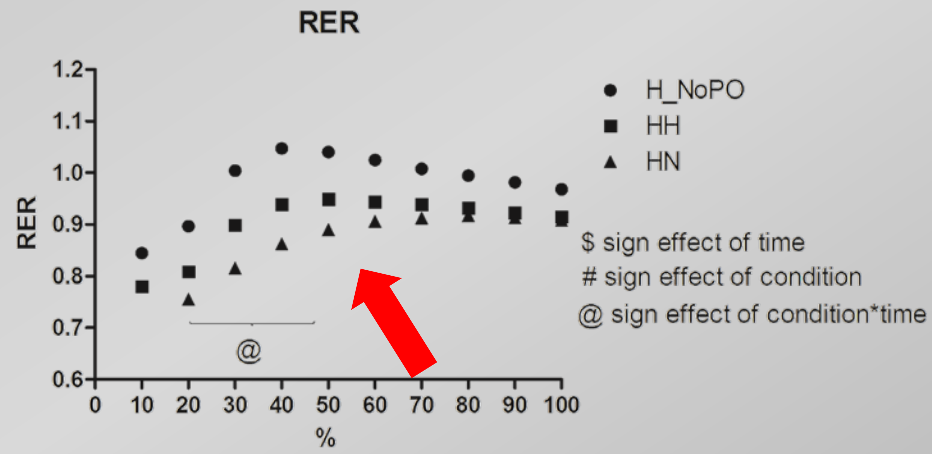
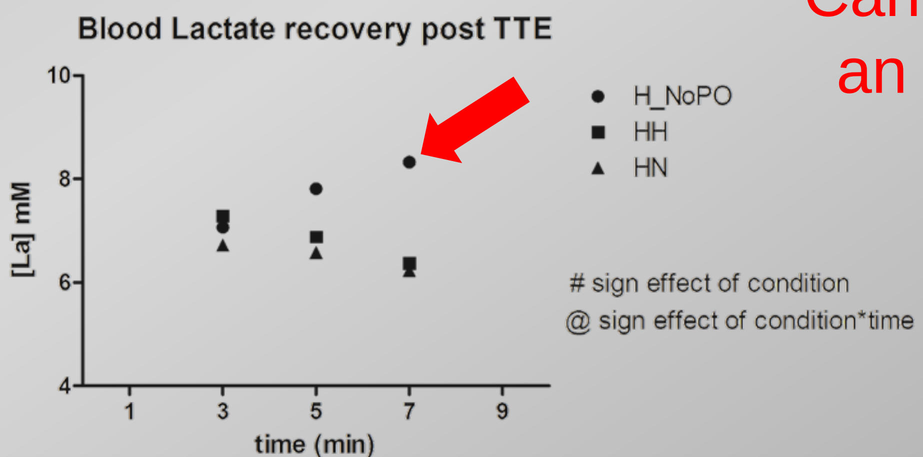
Discussion - TTE Performance

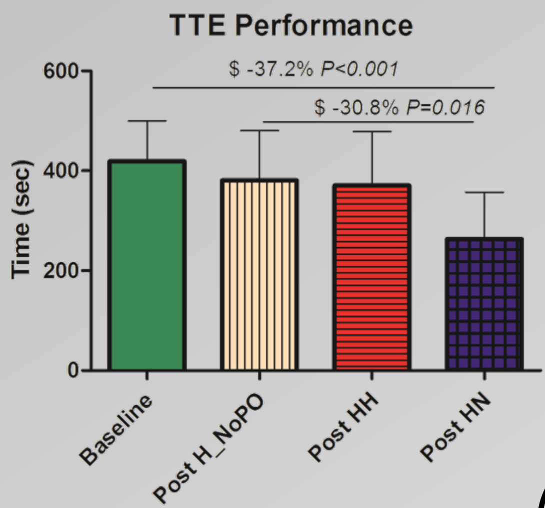






Can be considered an effect of early lactate?

Discussion - performance



Is only a matter of Altitude?

Hypoxia = ↓ O_2

Endurance performance

Oxygen Consumption

-14.3% each 1000 m+ ↓

-6% each 1000 m+ ↓

(Wehrlin,2006)
(Calbet, 2003)
(Garvican-Lewis, 2015)

Take home message

This is a first overview of what happen during an acute exposure to PH.
We need more work, but:

• When an athlete need to compete in altitude, is better to test his performance in altitude (chose a wrong performance intensity can influence negatively the race)

• Before a competition in hypoxia athletes need a proper warm up (probably with an intensity close to 50% of PPO)

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Future Perspectives

- Complete the data analysis, to obtain a better overview of the effects of PH
- Try to figure out the best timing to reach the start of a competition in hypoxia
- Predict performance in the TTE starting from data that can be collected on field during an effort in progressive Hypoxia
- Set up a protocol of testing procedures which allows to prescribe properly exercise intensity for competition in Hypoxia (and the pacing strategy)

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Grazie!

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