Summary

Private and central sports centers are run in Poland. They offer possibility of trainings on bicycle trainer or treadmill in controlled hypoxia. Professional sportsmen and ambitious amateurs more and more willingly use simulated hypoxia conditions to improve effects of their trainings and to make desired adaptive changes in their body. That changes are impossible to achieve in normoxia conditions or they require greater training loads. The emergence of that positive adaptive changes (that contributes to improving training skills) is multifactorial. The most important factors are: exercise intensity and intensity and time of hypoxia conditions. Mentioned factors determine the most physiological response to hypoxia. Applying too intense physical activity or too much hypoxia may have reverse effects and lead to deterioration of training capacity, whereas low intensity and short time of exposure to hypoxia may not cause any adaptive changes. Many researches of hypoxic trainings did not fill the gap associated with changes in anaerobe threshold (AT) and maximal lactate steady state (MLSS) under the influence of different intensity of hypoxia. AT and MLSS are commonly used for evaluation of physical capacity and to define individual training intensity to make the training process individualized. Training zones used in hypoxia but set in normoxia, would be ineffective without modifications because of specific body reactions, especially respiratory and circulatory systems' reactions. For this reason the primary research goal was analysis of influence of normobaric hypoxia in different intensity FiO_2=16% and FiO_2=14.5% on AT and MLSS in mountain bikers.

Sixteen male mountain bikers were qualified to the research. They were 25-40 years old. Each participant made cardiopulmonary exercise tests (CPET) and tests with constant intensity to set MLSS in normoxia and normobaric hypoxia in intensity FiO_2=16% and FiO_2=14.5% It corresponds to being at altitude of ~2000 and ~3000 m.a.s.l. Cardiorespiratory indicators, such as: oxygen uptake (VO_2), carbon dioxide elimination (VCO_2), heart rate (HR), minute ventilation (VE) and periodic measurement of lactate concentration in blood were taken during every done test both in normoxia and normobaric hypoxia. The data were used to designate metabolic threshold using ventilator threshold (VT1 and VT2), D-max and MLSS.

Obtained results showed that sudden exposition on normobaric hypoxia in FiO_2=14.5% intensity significantly ($p<0,05$) reduced threshold values of WR determined by method: VT1, VT2, D-max and MLSS by 22%, 18.2%, 13.4% and 18.5% compared with the results set in normoxia. Hypoxia in lower intensity - FiO_2=16% - caused significant ($p<0.05$) reduction of threshold values of WR determined by method VT1 and VT2 by 11% and 9.5% compared with the results set in normoxia, but it did not caused changes in WR determined by method D-max and MLSS. Normobaric hypoxia in FiO_2=16% and FiO_2=14.5% intensity caused significant ($p<0.05$) reduction of SpO_2. What's more, significant ($p<0.05$) positive correlation between SpO_2 and WR was demonstrated for every applied method that designated AT and WR on MLSS and WRmax. In conditions of FiO_2=14.5%, VO_2 on the threshold VT1 and on MLSS was significantly ($p<0.05$) reduced by 18% and 18.5% compared to normoxia. Changes in FiO_2=16% were not statistically significant. Threshold oxygen uptake on VT2 did not show changes in new conditions. VO_2 on D-max was not set. Threshold HR was designated by methods: VT1, VT2, D-max and MLSS. The data showed that HR did not change in hypoxic conditions and the method of determination was not important. Blood lactate concentration (La) on MLSS, taken after 20 and 30 minutes after the effort, was significantly ($p<0.05$) higher in FiO_2=16%, compared to normoxia. Higher intensity of hypoxia FiO_2=14.5% did not cause any further significant changes ($p=0.08$ and $p=0.09$). Normobaric hypoxia of FiO_2=16% and 14.5% intensity had no influence on changes in HRmax, VEmax, VTmax or BFmax. Mentioned conditions had significant ($p<0.05$) influence on reduction of VO_2max/kg and WRmax compared to results in normoxia. What's interesting is FiO2=14.5% affected on significant ($p<0.05$) reduction of slope VO_2/WR and OUES, but changes in %VO_2max, VO_2/WR, VT1 and VT2 were not noticed.

Hypoxia, resulting from the increase in height, causes reduction in ability to perform long-term exercises. The most important findings of that research was that in condition of normobaric hypoxia (intensity of FiO_2=16% and FiO_2=14.5%), we observe reduction of WR on VT1 of 11% and 22% compared to normoxia (that means average decrease by 11.5% on every 1000m) and on VT2 of 9.5% and 18.2% compared to normoxia (average decrease by 13.9% on every 1000m). Normobaric hypoxia FiO_2=14.5% caused 13.4% reduction of WR, determined by the method of D-max, and 18.5% reduction of MLSS compared to normoxia. Threshold, designated by La measurement, showed less sensitivity to concentration drop of O_2 in the air than ventilation thresholds. Moreover, the research results indicate that the effort in normobaric hypoxia of intensity of FiO_2=16% and FiO_2=14.5% did not influence HR changes on AT thresholds and on MLSS. This is probably due to reduction in load and training power requirements. The results complement previous scientific reports on VO_2max/kg reduction by 9% and 20% compared to normoxia (average decrease by 10.5% on every 1000m) and WRmax by 8.3% and 15.4% compared to normoxia (average decrease by 11.9% on every 1000m). It was also observed that in such environmental conditions bikers perform maximal effort without significant changes in values VE, VT, BF and HR. As O_2 concentration in the air decreases, reduction of OUES and increase of slopeVO_2/WR are visible, which indicates poorer oxygen consumption efficiency.