

1 Type of the Paper (Conference Paper)

2 Differences in physiological variables of U23 cyclists 3 between normoxia and hypoxia

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Received: date; Accepted: date; Published: date

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9 **Keywords:** altitude, testing, training, performance, cycling

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11 1. Introduction

12 For elite cyclists, the effect of altitude on
13 physiological parameters and thus on
14 endurance performance involves a complex
15 interplay of training, physiological
16 adaptation, and recovery¹⁻³. Since the
17 summer Olympic games 1968 in Mexico City
18 extensive research⁴ was conducted to study
19 the effects of altitude training on the human
20 body, as the physiological response to the
21 altitude stimulus can have a legal
22 performance enhancing effect for altitude
23 and sea level endurance performance^{5,6}.

24 2. Materials and Methods

25 The participants of the study were twelve
26 U23 cyclists (N=12) from a UCI continental
27 team. (Mean \pm SD: age 20.4 \pm 1.20 years; height
28 182.2 \pm 4.7 cm; body mass 68.4 \pm 6.6 kg; Pmax
29 6.6 \pm 0.4 W.kg⁻¹; VO_{2max} 72.6 \pm 5.1 ml.kg⁻¹.min⁻¹). The subjects were asked to avoid any
30 exhaustive activities and refrain from
31 caffeine and alcohol for the last 24 hours
32 before the graded incremental exercise test
33 (GXT). Participants were informed
34 adequately of the purpose and procedures of
35 the investigation. A written consent was
36 additionally obtained as set out in the
37 Declaration of Helsinki.

39 *Experimental Design* - The
40 experimental design included two GXT
41 within two days. The first GXT was

42 conducted in normoxia at 574m above sea-
43 level and the second GXT in a custom build
44 altitude chamber (GAIRRIT, Gerrit Glomser
45 GmbH, Kitzbühel, Austria) corresponding to
46 a simulated altitude of 1800m above sea-
47 level. Both GXTs were performed on the
48 participants' individual road bike mounted
49 on an electromagnetically braked ergometer
50 (Cyclus2, RBM elektronik-automation
51 GmbH, Germany) starting at an initial load of
52 100 watts with an increment of 20W every
53 minute until volitional exhaustion. Peak
54 power output (Pmax) in uncompleted stages
55 was calculated according to Kuipers et al.⁷.

56 *Measurements* - Open circuit spiro
57 ergometry with a breath-by-breath technique
58 (Cortex Metalyzer 3B, Cortex Biophysik
59 GmbH, Germany) was continuously
60 measuring respiratory flow, volume, and the
61 volume fractions of oxygen (O₂) and carbon
62 dioxide (CO₂) from expired air. The volume
63 and flow were calibrated with a 3l syringe,
64 gas analyzer calibration was performed
65 before each measurement as recommended
66 by the manufacturer (4.9 Vol% CO₂, 15.9
67 Vol% O₂, 79.2 Vol% N₂). Continuous
68 recordings of heart rate (HR) (Polar H9, Polar
69 Electro Austria GmbH, Austria) and oxygen
70 saturation (SpO₂) (Nonin® Pulse Oximeter,
71 Nonin Medical Inc, US) were measured at a
72 1Hz sampling rate. Measured variables
73 involved oxygen uptake (VO₂), carbon
74 dioxide release (VCO₂), minute ventilation
75 (VE), breathing frequency (BF) and tidal
76 volume (TV). The GXTs were performed in a

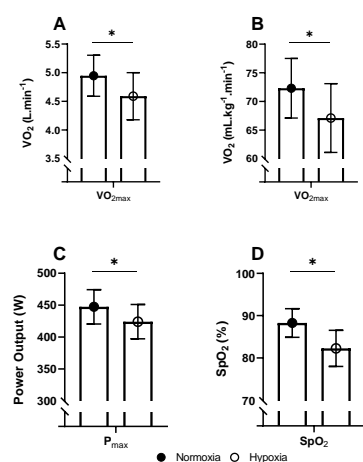


77 controlled environment (temperature
78 approx. 20°C, humidity approx.. 48%).

79 *Statistical Analysis* – All data are
80 represented as mean ± standard deviation
81 (SD) and checked for normality using
82 Shapiro Wilk ($p > .05$). Paired sample t-test
83 analyzed differences in physiological
84 variables between normoxia and hypoxia
85 conditions. The magnitude of the effect was
86 interpreted according to Cohen's d^8 for small
87 (.2 to .5), moderate (.5 to .8) and large (>.8)
88 effects. Statistical analysis was conducted
89 using a free available software package
90 (JASP, JASP Team, the Netherlands) and
91 graphs and figures were created with Prism8
92 (Graphpad software, US).

93 3. Results

94 Absolute VO_{2max} was significantly lower
95 ($d=1.21$, $p=.002$) in hypoxia (4.59 ± 0.36 L.min⁻¹)
96 than normoxia (4.95 ± 0.36 L.min⁻¹) – see
97 figure 1 A and B. Absolute P_{max} was
98 significantly lower ($d= 1.18$, $p=.003$) in
99 hypoxia (424 ± 27 W) than normoxia (447 ± 27
100 W) – see figure 1 C. Due to no significant
101 changes in body mass ($p > .05$) relative values
102 for VO_{2max} and P_{max} were also significantly
103 different between conditions. Peak SpO_2 was
104 significantly lower ($d=1.19$, $p=.003$) in
105 hypoxia (82.0 ± 4.3 %) than normoxia (88.0 ± 3.4
106 %) – see figure 1 D.



107

108 Figure 1: differences in absolute and relative
109 VO_{2max} - maximum oxygen uptake, P_{max} -

110 peak power and SpO_2 between normoxia and
111 hypoxia; *significantly different

112 No significant differences were found in the
113 other physiological variables including VE,
114 VT, BF and HR_{max} between normoxia and
115 hypoxia ($p > .05$).

116 4. Discussion

117 The present study investigated physiological
118 responses in normoxia at 574 m above sea-
119 level and in hypoxia at simulated 1.800 m
120 above sea-level to better understand the
121 immediate impact of altitude on physiology
122 determinants during cycling exercise.

123 The findings of the present study regarding
124 lower VO_{2max} values in hypoxia than
125 normoxia are in accordance with with
126 previous studies^{1,5,9}.

127 Lower P_{max} and decreased SpO_2 values in
128 hypoxia were also found by Gore et al.
129 studying elite cyclists.

130 Non-significant changes in VE were also
131 reported from Benoit et al.¹⁰ between hypoxia
132 and normoxia.

133 Although statistically analysis revealed no
134 differences in VE, VT and BF between
135 normoxia and hypoxia, inter-individual
136 differences might involve valuable
137 information about the altitude response of
138 the individual athlete. Combining
139 information from HR and ventilatory
140 responses across the whole intensity
141 spectrum^{6,11} might be beneficial to evaluate
142 immediate altitude effects on the human
143 body.

144 **Funding:** This research received no external
145 funding.

146 **Acknowledgments:** The authors would like to
147 thank all subjects for their willingness to
148 participate in this study.

149 **Conflicts of Interest:** The authors declare no
150 conflict of interest.

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