

Original Article

Low-load resistance training with hypoxia mimics traditional strength training in team sport athletes

WORRAWUT THUWAKUM¹, MICHAEL J. HAMLIN², NUTTASET MANIMMANAKORN³,
NARUEMON LEELAYUWAT⁴, PREETIWAT WONNABUSSAPAWICH⁵,
DISSAPHON BOOBPACHAT⁶, APIWAN MANIMMANAKORN⁷

^{1,5,6} Exercise and Sport Sciences Program, Graduate School, Khon Kaen University, THAILAND

² Department of Tourism, Sport and Society, Lincoln University, NEW ZEALAND

³ Department of Rehabilitation Medicine, Faculty of Medicine, Khon Kaen University, THAILAND

^{4,7} Department of Physiology, Faculty of Medicine, Khon Kaen University, THAILAND

^{4,5,7} Exercise and Sport Sciences Development and Research Group, Khon Kaen University, THAILAND

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Abstract

The aim of this study was to investigate the effects of low-load resistance training under hypoxia compared to conventional resistance training. Forty male team sport athletes (20.2 ± 1.7 y, 172.5 ± 5.6 cm, 66.3 ± 9.6 kg) were divided into 4 resistance training groups; normobaric 30%1RM (CT₃₀), normobaric 80%1RM (CT₈₀), hypoxic 30%1RM (HT₃₀), and hypoxic 50%1RM (HT₅₀). Resistance training included 3 sets of 15 repetitions of knee extensions and 3 sets of knee flexion, 3 day a week for 5 weeks. The hypoxic condition was set at $FiO_2 = 14\%$. Isometric, isokinetic and isotonic maximal voluntary contractions (MVC) along with blood lactate were measured before and after the five week training program. Compared to CT₃₀, isometric MVC increased substantially in all other groups after training (CT₈₀ $21.0 \pm 14.7\%$, HT₃₀ $16.9 \pm 12.3\%$, HT₅₀ 16.7 ± 7.9), however there was no significant difference between groups. Compared to CT₃₀ 1RM increased post training in the CT₈₀ and HT₅₀ groups ($23.7 \pm 10.8\%$ and $24.4 \pm 3.8\%$ $p = 0.004$, $p = 0.045$ respectively) with little difference found between CT₈₀ and HT₅₀ groups ($0.6 \pm 8.4\%$). Low-load resistance training under hypoxic conditions (HT₅₀) mimics the strength benefits gained from traditional high load training.

Keywords: Muscle strength, Endurance, Simulated altitude, High load, Blood Lactate

Introduction

Sport scientists, athletes and coaches recognize that resistance training is integral for improving muscular strength, however the ideal training regime for strength improvement is debatable. Muscular strength is a key factor in many sports, not only for improvement in physical performance but also for the prevention of injury (Kraemer & Fleck, 2005). Muscle strength and size gain is dependent on the type of exercise and intensity of resistance as well as training volume of the strength training program. Workloads of at least 65% of one repetition maximum (1RM) are required to achieve a substantial increase in strength. However, training benefits can be obtained throughout the RM range. High-loads (> 80% of 1RM) are used when the goal is maximum strength gain, moderate (> 50% of 1RM) for hypertrophy and muscle power enhancement, and low-loads (> 20% of 1RM) for muscular endurance (American College of Sports, 2009; Hillman SK, Perrin DH., 2005). Even though high resistance loads are commonly used by athletes, such training may result in muscle, ligament and tendon injury (Raske & Norlin, 2002) and reduced central arterial compliance (Miyachi et al., 2004) which may increase systolic blood pressure. In addition, some people are not able to lift such heavy weights (injured athletes, bed-rest patients, etc.). Therefore, to promote safe resistance training (avoid muscle injury and reduce risk of dangerous blood pressure changes) and to assist those who cannot perform high-load resistance exercise, low-load resistance exercise has been utilized by sport scientists in combination with other strategies such as venous occlusion and simulated altitude or hypoxia. Using low-load resistance exercise alone, has little adaptive effect on muscular strength, however, combined with either venous occlusion or hypoxia, low-load resistance training results in increased stress on the musculature which forces the muscle fibres to adapt and strengthen (Wernbom, Augustsson, & Raastad, 2008). As such, low-load resistance exercise (20-50% of 1RM), in combination with venous occlusion, has been proposed as an alternative to high-load resistance exercise⁷.

Similar to venous occlusion in combination with low-load resistance training, recent studies have reported an enhancement in muscle strength after low-load resistance exercise combined with hypoxia (Manimmanakorn, Hamlin, Ross, Taylor, & Manimmanakorn, 2013). It is thought that during venous occlusion the blood flow restriction probably causes substantial hypoxia (Downs et al., 2014; Loenneke & Pujol, 2009; Manini & Clark, 2009) which may play a key role in muscle adaptation (Manimmanakorn et al., 2013; Scott, Slattery, Sculley, & Dascombe, 2014), thus training under hypoxic conditions may be responsible for the increased force, endurance and size of the muscle rather than the reduced blood flow per se.

To date, there has been little research exploring what resistance loads when combined with hypoxia, produces the optimal strength adaptation and whether such training is superior to the standard practice of 80%1RM training under normoxia. Therefore, this study aimed to investigate the effectiveness of 5 weeks of two different low resistance training loads (30%, or 50%1RM) combined with hypoxia (compared to low-load 30%1RM and conventional resistance training of 80%1RM in normoxia) on muscle strength, endurance and physical performance.

Methods

Participants

Forty high performance team sports athletes (males aged 20.2 ± 1.7 years, soccer = 27, hockey = 3, basketball = 10) volunteered for this study. All athletes completed the Physical Activity Readiness Questionnaire and a skill-related physical fitness test. All athletes met the inclusion criteria as follows: they reported no exposure to an altitude of $> 1,000$ m within the last 3 months, no history of severe acute mountain sickness, no contraindicative health conditions, or medications (e.g., anabolic steroids, creatine, sympathoadrenal drugs) during the study and no resistance training within past 3 months. The athletes were excluded if they had any health problems such as; hypertension, cardiovascular disease, pulmonary disease, diabetes mellitus, orthopedic problems (e.g. bone, joint, muscle) and carcinoma. Athletes were informed about the experimental procedures as well as the purpose of the study and what was required of them. A written informed consent approved by the local human ethics committee was attained by each athlete prior to the start of the study.

Experimental design

This study is a randomized controlled trial comparing the muscle strength and endurance of extensor muscle groups, physical fitness performance and blood lactate changes after five weeks of training. The athletes were randomly divided into four experimental groups based on the level of intensity of resistance training: the low-load resistance exercise (30%1RM) while breathing room air (normobaric control training; CT₃₀, n = 10), the low-load resistance exercise (30%1RM) while breathing 14% oxygen concentration [hypoxic condition (hypoxic low-load resistance exercise; HT₃₀, n = 10)], low-load resistance exercise (50%1RM) while breathing 14% oxygen concentration [hypoxic condition (hypoxic low-load resistance exercise; HT₅₀, n = 10)], and high-load resistance exercise (80%1RM) while breathing room air (normobaric high-load resistance exercise; CT₈₀, n = 10). The hypoxic condition was generated using a hypoxicator machine (model: ATS-HP-Hyperoxic; Altitude Technology Solutions Co., Ltd. (ATS), Australia).

Exercise testing

After familiarization and 1-2 days prior to the training, all muscle contractile force estimations were conducted on the dominant leg with the athletes seated in an isokinetic machine (Primus RS, model: PR30, BTE technology USA). Athletes were placed on a chair with their back upright and with their dominate leg firmly secured (via Velcro straps) to the lever of the machine. The pivotal point of the lever was visually aligned with the rotation axis of the knee joint to maintain appropriate position during all testing. Maximal isometric voluntary contraction (MVC) measurements were completed by asking the athletes to produce the highest possible force for six seconds (MVC₆). The athletes were verbally encouraged to produce the highest force possible during the two trials (interspersed with one minute recovery); subsequently the highest force from both trials was used for analysis. After 3-5 min rest, a muscle isokinetic strength test was conducted at angular velocities of $60^{\circ} \cdot \text{sec}^{-1}$ and $180^{\circ} \cdot \text{sec}^{-1}$, between the range of 0 and 90 degrees. Five trials were taken at each angular velocity (2 min rest between each trial), and the highest value obtained was used for further analyses. After a further 3-5 min rest, dynamic muscle endurance was measured by calculating the number of repetitions the athletes could complete at a constant cadence (1 s concentric and 1 s for eccentric movement) with a 40%1RM load (Reps40 of 1RM). After a further 3 min rest, the muscle strength-endurance test was performed on the isokinetic machine. Athletes performed 3 sets of 10 repetitions (30s recovery between each set) at angular velocity of $60^{\circ} \cdot \text{sec}^{-1}$. Fatigue was calculated from this test by comparing the peak force developed in the first versus the last set. One to three days before and after the 5-week resistance training period all athletes completed a series of sport specific performance tests. The athletes were accustomed with these tests which were a regular part of their normal testing routines. Explosive power was estimated by the maximum effort countermovement jump test using standard procedures (Swift Yardstick Vertical Jump Tester made in Australia), while explosive speed was measured by a 10 and 20-m sprint. We used standard procedures for testing aerobic fitness via the maximal multistage 20-m shuttle run test (20-MST) (Leger & Lambert, 1982). Maximal oxygen consumption ($V\dot{O}_{2\text{max}}$) and maximal attained speed (MAS) were predicted based on 20-MST data (Flouris, Metsios, & Koutedakis, 2005). All tests were performed at the same time of day under similar temperature conditions on a nonslip surface in a covered stadium. A visual analogue scale was used for determining knee extensor muscle pain (Summers, 2001). The athletes were asked to record their daily subjective rating of pain (0 no pain, 10 severe pain) after they completed their resistance training.

Training program Resistance training was performed on a knee extension and flexion training device (leg flexion-extension S-105, STRIVE Fit LLC, USA) and consisted of 3 sets for flexion followed by 3 sets for

extension, (i.e.6 sets of 15 reps), with 60 s of rest between sets, three training session per week for 5 weeks. All groups performed a warm-up consisting of 3 sets of 10 reps at 20%1RM, before each training session. Training intensity and volume for the normoxic groups ($F_{I}O_2 = 20.9\%$) were set at 30% of 1RM (CT_{30}) and 80% of 1RM (CT_{80}). The hypoxic groups (HT_{30} and HT_{50}) trained with $F_{I}O_2 = 14\%$ and either 30% of 1RM (HT_{30}), or 50%1RM (HT_{50}). The 1RM strength was re-assessed after 3 weeks of training to adjust the training load in all groups. The range of motion in each set of exercise was from 90 to 0° (0° at full extension).

Measures

1RM measurement

One day before and 3 days after the 5-week training period, we measured knee extension 1RM using a stationary weight machine (leg extension S-105, STRIVE Fit LLC, USA) under normoxic conditions. Athletes completed a standardized warm-up and stretch for several minutes prior to testing. To estimate 1RM we used the 10RM test and formula as proposed by Brzycki (1998) ($1RM = \text{weight lifted}/1.0278 - (0.0278 \times \text{reps})$) (Brzycki, 1998).

Heart rate and SpO_2 monitoring Heart rate and SpO_2 were monitored daily before training and at the end of each set (6 sets/day) of resistance exercises by a pulse oximeter (Beurer model: PO30 made USA) in all groups.

Blood lactate measurement A small blood sample (15-50 μ l) was taken from the athlete's fingertip before, immediately, 15 and 30 minutes after the isokinetic knee extension fatigue test and analysed for blood lactate (ACCU-CHEK Safe-T-Pro Plus).

Statistical analysis The values are expressed as means \pm SD. All data were tested for normal distribution with the Kolmogorov-Smirnov statistic. The results in SpO_2 , blood lactate, heart rate, %fatigue and subjective scores (RPE score, Pain score) were analysed using an independent t-test to compare the control groups with the hypoxic groups. To compare pre and post training within groups a paired t-test was conducted. The MVC_6 , number of Reps, 1RM, isokinetic force (velocity at 60°. sec^{-1} and 180°. sec^{-1}), and physical performance parameters were analysed using a specifically designed spreadsheet available for controlled trials to calculate magnitude-based inferences for effect sizes (Hopkins, 2006). Then to make suppositions about true (population) values of the effect, the uncertainty in the effect was expressed as 90% confidence limits (CL). The chances that the true effects were substantial were estimated by the spreadsheet (Hopkins, 2006). We generated the smallest worthwhile change value by multiplying the baseline between-subject standard deviation by Cohen's value of the smallest worthwhile effect of 0.2 (Cohen, 1998). Effects that were simultaneously both > 75% likely positive and < 5% negative were considered large and beneficial. An effect was regarded unclear if its confidence interval overlapped the thresholds for substantiveness; that is, if the effect could be considered positive and negative.

Results

There were no significant differences in athletes' characteristics between groups. Compared to normoxic groups (CT_{30} , CT_{80}) the hypoxic groups (HT_{30} , HT_{50}) blood oxygenation saturation showed substantially lower blood oxyhaemoglobin saturation levels (SpO_2) at the end of every set (set 1 to set 6) during training (Fig. 1). No difference in SpO_2 levels were found within either normoxic or hypoxic groups.

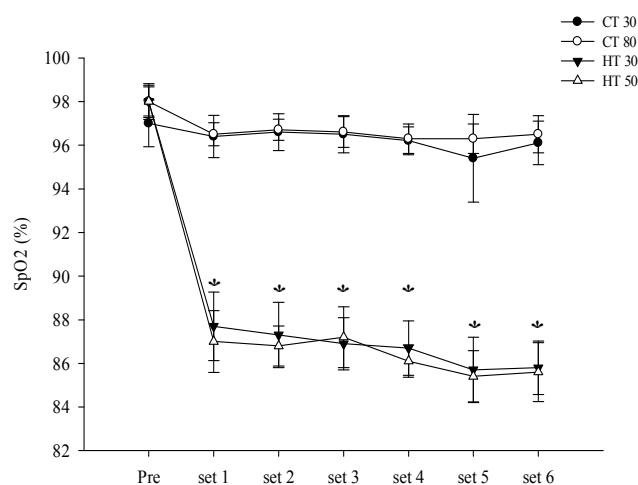


Figure 1. Changes in SpO_2 (mean \pm SD) before and at end of each set during training sessions in the hypoxia groups and control group *Significant difference between CT_{30} and HT_{30} or HT_{50}), #significant difference between CT_{80} and HT_{30} or HT_{50}).

Compared to baseline the MVC₆ of the knee extensors changed by $-0.9 \pm 7.4\%$, $21.0 \pm 14.7\%$, $16.9 \pm 12.2\%$, and $16.7 \pm 7.9\%$ in the CT₃₀, CT₈₀, HT₃₀, and HT₅₀ groups respectively, while 1RM increased over the same time by $4.9 \pm 4.8\%$, $23.7 \pm 10.8\%$, $5.0 \pm 2.5\%$, and $24.4 \pm 3.8\%$ in CT₃₀, CT₈₀, HT₃₀, and HT₅₀, and groups respectively. Relative to CT₃₀, HT₃₀ and HT₅₀ increased in explosive force measured via the vertical jump test ($5.3 \pm 3.7\%$ and $4.4 \pm 6.5\%$), speed 10 m ($-9.9 \pm 5.1\%$ and $-10.0 \pm 6.3\%$) after training (Table 1a-d). Compared to HT₃₀, individuals in the HT₅₀ group showed similar changes in performance indicators after the resistance training intervention (except for 1RM).

Table 1. The percent changes in muscular and physical performance indices after 5-weeks resistance training.

	Change in performance %		Chance that the true difference in substantial		
			Difference 90%CL	%	Qualitative
a) CT₃₀ vs HT₃₀					
	CT ₃₀	HT ₃₀			
Muscular Performance					
MVC ₆	-0.9 ± 7.4	16.9 ± 12.2	19.5 ± 12.7	98	very likely
Number of reps	-15.2 ± 25.6	10.8 ± 16.0	34.3 ± 20.9	99	very likely
1RM	4.9 ± 4.8	5.0 ± 2.5	-0.1 ± 3.8	33	unclear
Isokinetic contraction					
CON 60°.sec ⁻¹	33.8 ± 13.7	36.4 ± 18.6	-7.4 ± 24.1	24	unclear
CON 180°.sec ⁻¹	22.5 ± 17.9	37.7 ± 10.0	16.4 ± 15.9	94	likely
ECC 60°.sec ⁻¹	11.1 ± 22.2	18.0 ± 22.6	7.1 ± 24.9	68	unclear
ECC 180°.sec ⁻¹	23.0 ± 9.0	23.7 ± 15.1	0.2 ± 11.4	45	unclear
Physical Performance					
Vertical jump	-0.9 ± 4.5	5.3 ± 3.7	4.0 ± 4.8	86	likely
Speed 10 m	-3.4 ± 5.3	-9.9 ± 7.3	-3.1 ± 6.0	74	unclear
Speed 20 m	-5.0 ± 3.8	-7.1 ± 4.8	-2.0 ± 4.8	65	unclear
MAS	2.7 ± 4.1	4.3 ± 3.4	1.6 ± 3.9	60	unclear
V̇O _{2max}	3.6 ± 7.2	5.6 ± 7.1	2.1 ± 6.4	62	unclear
b) CT₃₀ vs HT₅₀					
	CT ₃₀	HT ₅₀			
Muscular Performance					
MVC ₆	-0.9 ± 7.4	16.7 ± 7.9	13.2 ± 7.7	99	almost certainly
Number of reps	-2.0 ± 27.9	16.0 ± 9.0	24.9 ± 22.1	96	very likely
1RM	4.9 ± 4.8	24.4 ± 3.8	12.2 ± 7.4	99	almost certainly
Isokinetic contraction					
CON 60°.sec ⁻¹	33.8 ± 13.7	37.9 ± 16.4	4.9 ± 22.0	29	unclear
CON 180°.sec ⁻¹	22.5 ± 17.9	27.4 ± 18.4	3.0 ± 18.9	58	unclear
ECC 60°.sec ⁻¹	11.1 ± 22.2	26.5 ± 16.0	5.9 ± 22.1	66	unclear
ECC 180°.sec ⁻¹	23.0 ± 9.0	25.5 ± 24.4	4.3 ± 14.0	67	unclear
Physical Performance					
Vertical jump	-0.9 ± 4.5	4.4 ± 6.5	5.5 ± 5.8	91	likely
Speed 10 m	-3.4 ± 5.3	-10.0 ± 6.3	-6.4 ± 6.0	94	likely
Speed 20 m	-5.0 ± 3.8	-7.4 ± 7.0	-2.4 ± 5.9	68	unclear
MAS	2.7 ± 4.1	1.5 ± 3.1	-0.6 ± 3.7	22	unclear
V̇O _{2max}	5.8 ± 5.7	3.5 ± 5.8	-1.4 ± 5.7	22	unclear

Table 1 (Cont.)

	Change in performance %		Chance that the true difference in substantial		
			Difference 90%CL	%	Qualitative
c) CT₈₀ vs HT₅₀					
	CT ₈₀	HT ₅₀			
Muscular Performance					
MVC ₆	21.0 ± 14.7	16.7 ± 7.9	-4.3 ± 13.3	22	unclear
Number of reps	17.2 ± 12.7	16.0 ± 9.0	-4.5 ± 11.5	19	unclear
1RM	23.7 ± 10.8	24.4 ± 3.8	0.6 ± 8.4	46	unclear
Isokinetic contraction					
CON 60°.sec ⁻¹	39.5 ± 10.0	37.9 ± 16.4	-3.8 ± 13.8	25	unclear
CON 180°.sec ⁻¹	40.9 ± 10.6	36.9 ± 15.9	-9.1 ± 19.6	16	unclear
ECC 60°.sec ⁻¹	28.2 ± 9.4	26.5 ± 16.0	-1.7 ± 14.4	36	unclear
ECC 180°.sec ⁻¹	21.6 ± 12.3	25.5 ± 24.4	4.4 ± 20.1	63	unclear
Physical Performance					
Vertical jump	0.8 ± 2.3	4.4 ± 6.5	-0.1 ± 4.3	32	unclear
Speed 10 m	-6.4 ± 2.5	-10.0 ± 6.3	0.5 ± 5.8	31	unclear
Speed 20 m	-4.1 ± 4.8	-7.4 ± 7.0	-3.3 ± 4.7	59	unclear

MAS	1.6 ± 4.9	1.5 ± 3.1	-0.1 ± 4.2	31	unclear
V̇O _{2max}	3.6 ± 8.7	3.5 ± 5.8	-5.9 ± 4.7	39	unclear
d)					
<i>HT₃₀ vs HT₅₀</i>					
Muscular Performance					
MVC ₆	16.9 ± 12.2	16.7 ± 7.9	-1.5 ± 11.6	34	unclear
Number of reps	10.8 ± 16.0	16.0 ± 9.0	6.0 ± 19.4	68	unclear
1RM	5.0 ± 2.5	24.4 ± 3.8	15.1 ± 3.0	100	almost certainly
Isokinetic contraction					
CON 60° .sec ⁻¹	36.4 ± 18.6	37.9 ± 16.4	1.4 ± 17.7	40	unclear
CON 180° .sec ⁻¹	37.7 ± 10.0	36.9 ± 15.9	-1.2 ± 16.2	51	unclear
ECC 60° .sec ⁻¹	18.0 ± 22.6	26.5 ± 16.0	-4.6 ± 18.0	27	unclear
ECC 180° .sec ⁻¹	23.7 ± 15.1	25.5 ± 24.4	0.1 ± 27.9	47	unclear
Physical Performance					
Vertical jump	5.3 ± 3.7	4.4 ± 6.5	-1.7 ± 5.7	19	unclear
Speed 10 m	-9.9 ± 5.1	-10.0 ± 6.3	1.4 ± 6.3	24	unclear
Speed 20 m	-7.1 ± 3.6	-7.4 ± 7.0	-0.7 ± 6.0	46	unclear
MAS	4.3 ± 3.2	1.5 ± 3.1	-2.5 ± 3.5	5	unclear
V̇O _{2max}	5.6 ± 5.8	3.5 ± 5.8	-4.2 ± 5.9	6	unclear

± 90% confidence limits; MVC₆, the peak maximum isometric voluntary contraction in 6s; Number of reps, the number of repetitions able to be performed at 40% 1RM; 1RM, one repetition maximum; CON, concentric contraction; ECC, eccentric; MAS, maximal attained speed during the 20-m shuttle run test; V̇O_{2max}, estimated maximal oxygen consumption from the 20-m shuttle run test.

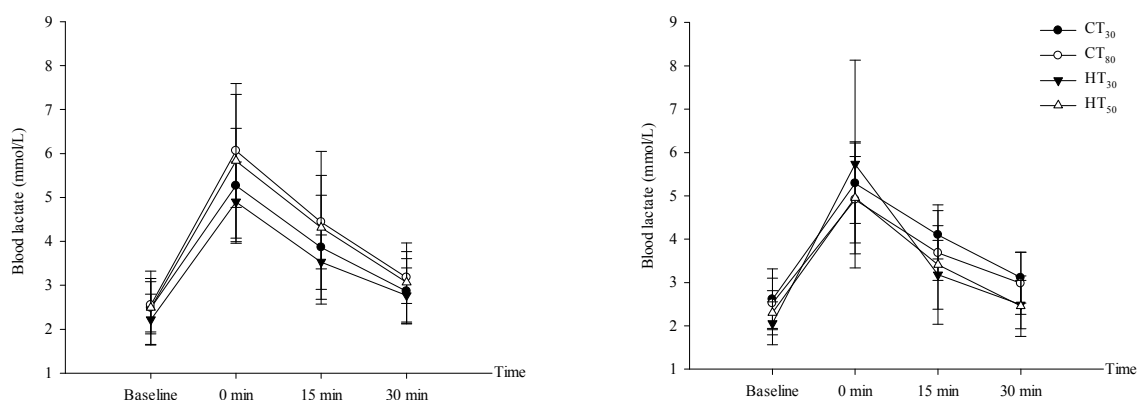


Figure 2. Changes in blood lactate concentration before, immediately, 15 min and 30 min after the first day (a) and last day (b) of training session. *Significant $p < 0.05$ (CT₃₀ vs HT₃₀), #significant $p < 0.05$ (CT₃₀ vs HT₅₀) The blood lactate concentration increased immediately after the initial exercise in all groups however, no significant differences between groups ($p < 0.05$) were found (Fig. 2a). After 5 weeks of training however, compared to CT₃₀, HT₃₀ and HT₅₀ groups showed substantially lower post-exercise (15 and 30 min post) blood lactate concentration (Fig. 2b).

The amount of fatigue during isokinetic knee extension was significantly decreased in HT₃₀ (-10.2 ± 5.5%) and HT₅₀ (-7.4 ± 2.2%) when compared to their baseline. In addition, the difference between pre and post-tests in fatigue rate improved by 7.2%, 6.6%, 12.5%, and 13.2% in the CT₃₀, CT₈₀, HT₃₀, and HT₅₀ respectively. Significant differences were only found when comparing CT₃₀ and HT₅₀ (Table 2).

Table 2. The knee extension fatigue rates (%) in concentric and eccentric contraction before and after 5-week resistance training.

		CT ₃₀	CT ₈₀	HT ₃₀	HT ₅₀
Fatigue rate (%) concentric	pre	20.8 ± 8.1	18.7 ± 7.8	22.8 ± 9.7	20.6 ± 12.1
	post	13.6 ± 3.9	12.0 ± 9.7	10.2 ± 5.5*	7.3 ± 2.2*
	differences	-7.1 ± 8.9	-6.6 ± 6.3	-12.5 ± 8.8	-13.2 ± 12.1 [#]
Fatigue rate (%) eccentric	pre	20.0 ± 15.7	20.4 ± 10.9	18.1 ± 10.4	20.8 ± 16.3
	post	16.2 ± 9.9	14.1 ± 8.9	16.7 ± 8.2	10.0 ± 6.2
	differences	-3.7 ± 19.6	-6.4 ± 10.2	-1.3 ± 15.0	-10.8 ± 19.0

Values are mean ± SD. *Significant $p < 0.05$ (CT₃₀ vs HT₅₀), #Significant $p < 0.05$ (pre vs post).

The mean ± SD of daily pain score at the end of exercise for 15 training sessions in CT₈₀ was significantly higher (5.5 ± 1.0, $p < 0.05$) compared with HT₃₀ (3.5 ± 1.0) and HT₅₀ (4.5 ± 1.0).

Discussion

This research investigated the optimal load for resistance training combined with hypoxia (30%1RM and 50%1RM) in an attempt to improve muscular strength and increase physical performance in team sport athletes. This study has three major findings. Firstly, for the most part, muscular performance (MVC_6 , $CON60^\circ \cdot sec^{-1}$, $CON180^\circ \cdot sec^{-1}$, number of reps and 1RM) improved in the hypoxia groups (HT_{30} , HT_{50}) compared to the normoxia control group (CT_{30}) and it seems that 50%1RM may be slightly better than 30%1RM when compared with CT_{30} for resistance training under hypoxic conditions. Secondly, low-load resistance (50%1RM) training under hypoxia has similar performance benefits to the more traditional high-load resistance training protocol and finally, compared to CT_{30} resistance to muscular fatigue was only improved in the HT_{50} group. Moreover resistance training while breathing hypoxic air was at least as effective as traditional resistance training using 80%1RM (CT_{80} group). These results represent new and unique findings as there has been no study that has investigated the effects of resistance exercise with different loads combined with hypoxia and no study that has compared low-loads (30%1RM and 50%1RM) with traditional high resistance training (80%1RM).

We found a positive effect of low-load resistance training under hypoxic conditions on muscular strength. Peak force or MVC_6 significantly increased by 16.9 % and 16.7% in the HT_{30} and HT_{50} groups respectively, but dropped by -0.9% in the control group (CT_{30}) after 5 weeks of knee extension training (Table 1). Moreover, this peak force increase was similar to the force changes that occur with traditional high-load resistance training (CT_{80} = 21% improvement). These results suggest that both hypoxic workloads were sufficient to induce muscular strength gains after short-term resistance training in hypoxia corroborating previous research on female athletes (Manimmanakorn, Hamlin, Ross, Taylor, & Manimmanakorn, 2013). In the current study, the low-load resistance training without hypoxic gas showed no improvement in muscle strength while the addition of a hypoxic stress to the working muscles resulted in performance improvement. These results have demonstrated that 30%1RM and 50%1RM loads in addition to breathing 14% O_2 put more stress or created more work load for the muscles than that in control training group. Obviously the only thing different between these two hypoxic groups is the amount of workload as the hypoxic stress was the same. So a substantial improvement in 1RM strength (almost certainly) between groups is due to the additional stress caused by the different workloads not the different hypoxic loads. With more stress on the muscle perhaps the more anaerobic it becomes and the greater the hypoxic stress. This would be intriguing to measure in future studies.

The mechanisms involved in higher force production with hypoxic resistance training include an enhancement in muscle cross-sectional area. Previous research found that resistance training under hypoxic conditions induced greater muscle hypertrophy (hypoxic group increased 16.6% compared to 5.8% in the normoxic group) of the triceps muscle following an 8-week elbow extension training protocol (3 sets, 3 days a week at an intensity of 10RM at 12.7% F_iO_2) compared with normoxic training (Kurobe et al., 2015). Similar results were reported by Manimmanakorn et al. (2013) after 5 weeks of low-load resistance knee extensor training (20%1RM) in female athletes. It is possible that hypoxia may have a direct effect on contractile protein accretion and thereby contribute to the hypertrophy stimulus, although, this has not been well examined (Manimmanakorn, Hamlin, Ross, Taylor, & Manimmanakorn, 2013). It is suggested that increases in myofibrillar volume, sarcoplasmic reticulum, cytoplasmic density, T-tubule density, and sodium potassium ATPase activity occur following resistance training. Consequently, these changes result in muscle size gain, and induce increased muscle function, and enable greater expression of strength (Baechle, Earle, 2008). The addition of hypoxia may exacerbate these cellular adaptations. But not all studies report hypertrophy accompanying resistance training with hypoxia. Friedmann et al. (2003) reported that 4 weeks of low-load (30%1RM) knee extension exercise under normobaric hypoxia (F_iO_2 = 12%) did not induce muscle cross-sectional area (Friedmann et al., 2003). Differences in training protocols, duration of training and recovery between sets and hypoxic stress conditions probably contribute to the differences reported in these studies.

We speculate that the hypertrophy of the muscle undergoing hypoxic training may be caused by the reduced oxygen delivery. Reduced oxygen to the muscle can cause a complex of downstream biological events resulting in metabolic and hormonal alternations (Scott et al., 2014). Evidence for this anaerobic situation during hypoxic training was found in the blood lactate levels immediately after the first training session which were substantially higher in the hypoxic (HT_{30} , HT_{50}) compared to the normoxic control group (CT_{30}). High levels of blood lactate have a stimulatory effect on serum growth hormone release (Stokes, 2003), which may account for higher levels of growth hormone found after hypoxic training (Kon et al., 2010; Kurobe et al., 2015; Nishimura et al., 2010), and the subsequent muscle hypertrophy (Manini & Clark, 2009).

A new finding of the present study was that low-load (50%1RM) resistance training under hypoxic conditions (F_iO_2 = 14%) illustrated the potential to replace the conventional high-load 80%1RM resistance training. Our result demonstrated that the addition of a hypoxic environment (HT_{50}) significantly increased most indices of muscular performance. In addition, no significant differences were found between CT_{80} and HT_{50} in all muscular performance parameters such as MVC_6 ($21.0 \pm 14.7\%$, $16.7 \pm 7.9\%$), number of reps ($17.2 \pm 12.7\%$, $16.0 \pm 9.0\%$) and 1RM ($23.7 \pm 10.8\%$, $24.4 \pm 3.8\%$), (Table 1c). These results suggest that the low-load

resistance training under hypoxic conditions resulted in sufficient stress to induce muscular strength gains similar to the high-load resistance training.

In addition to maximal voluntary force improvements, strength training under hypoxia also resulted in improvement of fatigue measures (Table 2). We also found that post-training blood lactate concentration rapidly decreased at 15 and 30 min after muscle strength testing (Fig. 2b). This may indicate enhanced blood lactate wash out from the circulation post training. Similar findings have been reported after 18-21 days of real altitude acclimatization (Bender et al., 1989; Brooks et al., 1991) or 17 days of intermittent hypoxic training with cycle ergometry (Casas et al., 2000). The lowering of the blood lactate concentration after strength testing in this study may have influenced the fatigue scores witnessed. This post-exercise reduction in blood lactate concentration may be associated with the haemodynamic response associated with hypoxia. The reduction of oxygenation level during exercise with hypoxia can cause greater reactive hyperaemic response following exercise (Scott et al., 2014). Moreover, exercise in hypoxia is a known stimuli for compensatory vasodilatation which aims to match an increased oxygen supply to the increased demand at the muscle level (Casey et al., 2010). Both hyperaemia and vasodilation after hypoxic resistance training may play a key role in attenuating the blood lactate concentration. On the other hand, the increased anaerobic nature of hypoxic training may evoke adaptation and subsequent improvement in the muscles ability to continue to function under a low oxygen environment. Kon (2014) recently reported increased plasma VEGF concentration and capillary-to-fiber ratio following training under hypoxic conditions (Kon et al., 2014). Improvements in muscular endurance were associated with increases in skeletal muscle oxidative fiber types, increased activity of metabolic enzymes, improvement in muscle buffering capacity, and enhanced capillarization (Friedmann et al., 2003). All of these changes may result in lowered post-exercise blood lactate concentration and possibly an improvement in strength-endurance as was found in the current study.

Finally, this study found that hypoxic resistance training was similar to traditional high-load training in its effect on sport-specific performance (e.g. explosive power and 10-m speed). Moreover, daily pain score during training session also show substantial higher in traditional resistance training compared with hypoxic groups. These results suggest that low-load intensity resistance training under hypoxic conditions is able to evoke muscular adaptations and less muscular pain which are likely to be beneficial for team-sport athletes.

Conclusion

Compared to a normobaric control group, low-load resistance exercise (either at 30%1RM or 50%1RM) under hypoxic conditions improved muscular performance (MVC₆, 1RM, number of reps and % fatigue rate). In addition, while both HT groups substantially increased physical performance (vertical jump and speed 10 m), only the group that trained at 50%1RM matched all strength benefits gained (MVC₆, 1RM, and number of reps) by traditional high load (80%1RM) resistance training.

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