Title: Running mechanics adjustments to perceptually-regulated interval runs in hypoxia and normoxia

Abstract

Objectives: We determined whether perceptually-regulated, high-intensity intermittent runs in hypoxia and normoxia induce similar running mechanics adjustments within and between intervals.

Design: Within-participants repeated measures.

Methods: Nineteen trained runners completed a high-intensity intermittent running protocol (4 × 4-min intervals at a perceived rating exertion of 16 on the 6–20 Borg scale, 3-min passive recoveries) in either hypoxic (FiO_2 = 0.15) or normoxic (FiO_2 = 0.21) conditions. Running mechanics were collected over 10 consecutive steps, at constant velocity (~15.0±2.0 $km.h^{-1}$), at the beginning and the end of each 4-min interval. Repeated measure ANOVA were used to assess within intervals (onset *vs*. end of each interval), between intervals (interval 1, 2, 3 *vs*. 4) and FiO_2 (0.15 *vs*. 0.21) main effects and any potential interaction.

Results: Participants progressively reduced running velocity from interval 1–4, and more so in hypoxia compared to normoxia for intervals 2, 3 and 4 (P<0.01). There were no between intervals (across all intervals P>0.298) and FiO_2 (across all intervals P>0.082) main effects or any significant between intervals × within intervals × FiO_2 interactions (all P>0.098) for any running mechanics variables. Irrespective of interval number or FiO_2, peak loading rate (+10.6±7.7%; P<0.001) and duration of push-off phase (+2.0±3.1%; P=0.001) increased from the onset to the end of 4-min intervals, whereas peak push-off force decreased (–4.0±4.0%; P<0.001).

Conclusions: When carrying out perceptually-regulated interval treadmill runs, runners adjust to progressively slower velocities in hypoxia compared to normoxia. However, only subtle constant-velocity modifications of their mechanical behaviour occurred within each set, independently of FiO_2 or interval number.

Keywords: Hypoxia; Instrumented treadmill; Running kinematics; Self-selected exercise.

Practical applications

- With the addition of hypoxia, runners should not fear substantial detrimental effects on their running technique during high-intensity intermittent exercise compared to normoxia.
- Only subtle alterations in running mechanics patterns develop within intervals, with adjustments occurring mainly within rather than between intervals.
- From the onset to the end of each of the four intervals, running mechanics adjustments are mainly visible during the push-off as opposed to the braking phase.

1. Introduction

High-intensity intermittent exercise (HIIE) is a popular intervention in athletic and clinical populations for improving exercise capacity. Although there is a large variation in HIIE regimens applied, broadly it consists of repeated bouts of high-intensity intervals of exercise interspersed with low-intensity or passive rest periods.[1] The physiological responses associated with HIIE have been studied for decades and continue to generate considerable research interest.[2] Comparatively, the kinetic and kinematic (biomechanical) perturbations as a consequence of this exercise modality (i.e., run-based studies) have received less attention. Biomechanical manifestation of fatigue during HIIE at constant running velocity have yielded inconsistent findings. When completing treadmill intervals (6 × 30-s repetitions at ~20 $km.h^{-1}$ with 30 s of rest), high-level team-sport players modified their running mechanics towards longer step lengths, decreased step frequencies and vertical stiffness.[3] Contrastingly, an intense interval training session consisting of 10 × 400-m repeats with recoveries ranging 60–180 s did not consistently or substantially alter the running kinematics of well-trained male runners.[4] Information about the pattern of running is important since an impaired tolerance to ground impact may detrimentally affect mechanical load control, and consequently increase injury risk.[5]

A number of studies have recently been conducted to identify the acute effects of HIIE with the addition of hypoxia.[6,7] When performing HIIE, current literature indicates that an acute hypoxic environment accentuates physiological stress compared to normoxia, thus limiting absolute exercise intensity due to reduced maximal oxygen uptake.[8] When an attempt is made to match relative intensity between groups, carrying out a fixed-intensity HIIE protocol (3 × 5 min with 90 s of rest) at a simulated altitude of ~2400 m (FiO_2 = 0.15) was not associated with an exacerbated physiological stress in young, highly-trained runners compared to near sea-level conditions (velocity associated with maximal oxygen uptake = 84% *vs*. 90%).[9] Much of the current literature examining acute effects of performing HIIE protocols include implementation of a fixed intensity of exercise.[3,4,9]

The utilization of pre-determined, fixed physiological intensities lacks ecological validity, as a training intensity is ultimately self-regulated by the individual carrying out the exercise.[10] Therefore, attention has recently shifted to perceptually-regulated exercise in the form of HIIE, where intensity/workload is freely adjustable also representing a feasible option to accommodate the strenuous nature of this exercise model.[11] When completing 5 × 2000-m runs at ~80% of the velocity associated with maximal oxygen uptake (Time = ~390–395 s; RPE range = 13–17) interspersed with 120 s of passive recovery, runners showed no noticeable alterations in their kinematics and foot strike patterns (Latorre-Roman et al. 2017).[12] To date, however, no study examined how the addition of an hypoxic stimulation during the course of a self-paced HIIE influences stride mechanics adjustments.

Our aim was to test the hypothesis that, compared to normoxia, perceptually-regulated HIIE in hypoxia induces similar mechanical adjustments during running within and between intervals.

2. Methods

Nineteen trained runners (3 females, 16 males; age: 33.4±9.1 years; stature: 176±88 cm; body mass: 76.3±10.9 kg) provided written informed consent to participate. The study was approved by *Anti-Doping Laboratory Ethics Committee* in Qatar (Agreement SCH-ADL-170), and conducted according to the *Declaration of Helsinki*.

The experimental design as well as the main psycho-physiological results have been reported previously.[13] Participants visited the laboratory on three occasions, each separated by ≥48 h. The first session included study familiarisation, during which preferred running velocity, corresponding to the velocity participants considered as an RPE of 16 (between "hard" and "very hard") or closest to a heart rate of 160 bpm, was determined for each participant in normoxia (see Hobbins et al.[13] for a detailed description). The second and third visits included completing a HIIE protocol in either hypoxia (FiO_2 = 0.15, equivalent to ~2700 m above sea level) or normoxia (FiO_2 = 0.21) in a randomized, counterbalanced order. After a standardized warm up (5 min at 10 km.h^{-1}), a facemask connected to a portable hypoxic generator (Altitrainer, SMTec SA, Nyon, Switzerland) was attached. Participants rested for 1 min (quiet standing) before a 1-min run (RPE = 16), followed after 3 min of passive rest by the HIIE protocol. Total hypoxic exposure corresponded to exactly 28 min.

Participants ran on an instrumented treadmill (ADAL3D-WR, Medical Development–HEF Tecmachine, France) in an indoor facility maintained at ~24°C and 45% of relative humidity. They completed four, 4-min intervals, interspersed with 3-min recoveries (quiet standing). This HIIE format with long intervals and performed on a treadmill is popular in trained runners for concurrently adapting cardiopulmonary function, causing moderate acute changes in neuromuscular performance and minimizing traumatic or overuse injury risk level.[14] Participants commenced all rest-to-exercise transitions (or *vice-versa*) by holding the sidebars of the treadmill, while stepping directly on the moving treadmill belt during work intervals or on the sides of the treadmill during the recovery periods, respectively.

The first 30 s and last 30 s of each 4-min interval were performed at constant velocity corresponding to an RPE of 16 (group average: ~15.0±2.0 km.h^{-1}), which was determined in normoxia. This running velocity was imposed externally via controlling treadmill belt velocity at the onset and end of each interval. The same velocity was imposed during the assessment phases in both normoxic and hypoxic trials, with participants blinded to actual treadmill velocities throughout the protocol. During the main part of each interval (i.e., for 3 min after excluding the first and last 30 s), participants were then free to decide if or how treadmill velocity needed to be adjusted (manually by one experimenter) to ensure maintenance of a RPE of 16 as checked and regulated every 30 s. Participants hand-signalled in response to the current velocity (finger up to increase, finger down to decrease, and circle using index finger and thumb to maintain); and signalled again to inform how much of an increase/decrease in velocity is required [1, 2 or 3 fingers up (faster) or down (slower) for 0.5, 1.0 or 1.5 km.h^{-1} changes, respectively]. The hand-signalling procedure was was trialled during familiarisation. Mild verbal encouragement to keep running at an RPE of 16 was used throughout HIIE.

Data were continuously sampled at 1,000 Hz, and after appropriate filtering (Butterworth-type 30 Hz low-pass filter), instantaneous data of vertical, net horizontal and total (resultant) GRF were averaged for each support phase (vertical force above 30 N). These data were determined by measurement of the main step kinematic variables: contact time (s), aerial time (s), step frequency (Hz) and step length (m). Peak braking and peak propulsive forces (BW), duration of braking and propulsive phases (s) along with braking and push-off impulses (N.s) were determined. Finally, vertical mean loading rate (LR) was calculated as the mean value of the time-derivate of vertical force signal within the first 50 ms of the support phase, and expressed in BW.s^{-1}.[15]

A linear spring-mass model of running was used to investigate the main mechanical integrative parameters characterizing the lower limb behaviour during running. Vertical stiffness ($kN.m^{-1}$) was calculated as the ratio of peak vertical force (N) to the maximal vertical downward displacement of centre of mass (m), which was determined by double integration of vertical acceleration of centre of mass over time during ground contact. Leg stiffness ($kN.m^{-1}$) was calculated as the ratio of peak vertical forces to the maximum leg spring compression [maximal vertical downward displacement + L_0 - $\sqrt{L_0^2}$ – (0.5 × running velocity × contact time)^2, m], both considered to occur at mid-stance. Initial leg length (L_0, great trochanter to ground distance in a standing position) was determined from participant's stature as $L_0 = 0.53 \times$ stature.[16]

Running mechanics were collected over 10 consecutive steps, at constant velocity corresponding to an RPE of 16 (group average: 14.8±1.9 $km.h^{-1}$), at the onset and the end of each 4-min interval (i.e., after running for ~15 s and ~3 min 45 s, respectively) and average values were calculated for further analysis. Heart rate was monitored telemetrically with a Polar transmitter-receiver (Polar S810, Kempele, Finland), while arterial oxygen saturation was assessed via finger pulse oximetry (Palmsat 2500, NONIN Medical Inc., Plymouth, MI, USA) at the same time intervals. Instantaneous running velocity, heart rate watch and oximeter receiver were outside of the participants' view.

Values are presented as mean±SD. Three-way repeated-measures analysis of variance (ANOVAs) [Between intervals (interval 1, 2, 3 *vs*. 4) × Within intervals (onset *vs*. end) × FiO_2 (0.15 *vs*. 0.21)] were used to compare investigated variables. A Bonferroni *post-hoc* multiple comparison was performed if a significant main effect or interaction was observed. For each ANOVA, partial eta-squared (η^2) was calculated as measures of effect size. Values of 0.01, 0.06 and above 0.14 were considered as small, medium and large, respectively. All statistical calculations were performed using SPSS statistical software V.24.0 (IBM Corp., Armonk, USA). The significance level was set at $P<0.05$.

3. Results

Compared to interval 1, participants adjusted to a progressively slower running velocity during intervals 2, 3 and 4 (−2.8±2.6%, −5.2±3.3% and −7.0±3.6%, respectively; $P<0.01$). Running speed was also slower for intervals 2, 3 and 4 (−4.6±2.1%, −6.4±3.2% and −7.9±3.6%, respectively; P<0.01) in hypoxia than in normoxia (Figure 1). Arterial oxygen saturation was lower in hypoxia compared to normoxia (86.0±4.2% *vs*. 94.8±2.2%; P<0.01), whereas heart rate did not differ.

There were no between intervals (all P>0.298; $0.04<\eta^2<0.65$) and FiO_2 (all P>0.082; $0.01<\eta^2<0.16$) main effects or any significant between intervals × within intervals × FiO_2 interactions (all P>0.098; $0.01<\eta^2<0.11$) for any running mechanics variables (Figures 2 and 3). Irrespective of interval number or FiO_2, peak loading rate (95.4±3.1 *vs*. 103.7±2.8 $BW.s^{-1}$; +10.6±7.7%; P<0.001) and duration of push-off phase (0.110±0.003 *vs*. 0.112±0.003 s; +2.0±3.1%; P=0.001) increased from the beginning to the end of 4-min intervals, whereas peak push-off force decreased (0.430±0.018 *vs*. 0.413±0.018 BW; –4.0±4.0%; P<0.001) (Figure 3). Neither braking (pooled values: -21.9±4.5 vs. -21.4±4.0 N.s; P=0.842; $\eta^2=0.002$) nor push-off (pooled values: 22.5±4.5 vs. 21.9±4.1 N.s; P=0.598; $\eta^2=0.016$) impulses differed between hypoxia and normoxia, with also no significant within and between intervals main effects (all P>0.122; $\eta^2>0.032$).

*** Figures 1, 2 and 3 about here ***

4. Discussion

During perceptually-regulated (RPE = 16) HIIE, participants ran progressively slower across intervals with larger decreases in hypoxia compared to normoxia, which was also accompanied by lower arterial oxygen saturation values but similar heart rates. Similar findings were obtained in obese adults where preferred walking velocity (RPE = 10) in hypoxia ($FiO_2 = 0.15$) was 7% slower than in normoxia, while heart rate did not differ between conditions.[17] The combination of slower running velocities and acute hypoxic exposure is an intervention that is cardio-metabolically similar to exercising near sea level, yet it is associated with a reduced external workload. Practically, HIIE of this nature could be useful for athletes during intensified periods of training when large exercise volumes can cause excessive mechanical loading in their lower extremities or those suffering from musculoskeletal disorders. It is well described that the mechanical behaviour is substantially influenced by running velocity variations.[18] This implies that, during self-paced runs, it is difficult (if not impossible) to distinguish the effects of fatigue from auto-regulatory mechanisms associated with a pacing strategy on stride mechanics parameters. To overcome this limitation, despite being free to vary during the proposed HIIE, the running velocity was externally imposed for 30 s both at the onset and end of each 4-min interval for the purpose of assessing acute changes in stride mechanics.

A novel finding was that completing HIIE with the addition of hypoxia had no measurable effect on any biomechanical variable. As with this study, changes of time-based gait parameters (e.g., slower cadence, longer stride time, and larger temporal gait variability) from the beginning to the end of a 40-min treadmill walk were similar at 2600 m simulated altitude and near sea-level in healthy, older individuals.[19] On the other hand, compared to normoxia, severe (~3600 m) but not moderate (~1800 m) hypoxia accentuates the fatigue-related inability to effectively apply forward-oriented ground reaction force and to maintain vertical stiffness and stride frequency during repeated treadmill sprints.[20] Discrepant findings could be due to the level of fatigue attained by participants that in turn depends on the manipulation of variables used to prescribe HIIE and the “hypoxic dose”. We cannot exclude that other HIIE formats may have differently influenced the decision to either slow down or speed up every 30 s and thereby the resulting mechanical adjustments. Analogously, despite higher thermal, cardiovascular and perceptual strain and impaired proprioception under heat stress, fatigue-induced changes in spatio-temporal parameters and joint angles (at a given sub-maximal velocity) resulting from a 30-min self-paced treadmill run did not differ between temperate and hot conditions.[21] Collectively, perceptually-regulated HIIE in normoxia and moderate hypoxia are two very similar protocols in terms of running mechanics adjustments.

In general, running mechanics pattern remained largely unaffected between early and late stages (i.e., from first to last interval) of our proposed HIIE, a finding also reported elsewhere.[4,22,23] For instance, intense interval training session consisting of either 10 runs of 400 m (rest = 60–180 s) or 40 runs of 100 m (rest = 25–30 s) did not modify the running kinematics of well-trained male runners.[4,23] However, having individuals constrained to run at a set velocity during fatigued running lacks ecological validity.[10] Our spring-mass model results are partially in accordance with previous observation of modified mechanical behaviour towards lower vertical stiffness but constant leg stiffness during interval-training treadmill runs.[3] A possible explanation would be that our participants may have used different ‘control’ strategies during later compared to initial stages of the intervals since fatigue incurred by the runs was probably mild due to the possibility of frequently adjusting running velocity. In the absence of surface EMG (e.g., muscle activation patterns) and high-speed camera (e.g., joint angles at touch down/take-off) recordings, however, it is difficult to discuss whether compensatory neuromuscular system strategies were adopted. More generally, up to nine variables are considered when prescribing HIIE; *i.e.*, work interval intensity and duration, relief interval intensity and duration, exercise modality, number of repetitions, number of series, as well as the between-series recovery duration and intensity.[1] Manipulation of these variables likely affect acute physiological responses and load imposed to the neuromuscular system.

Previous HIIE biomechanical analyses have been restricted to exploring mechanical behavior alterations across intervals, yet with no focus on changes during each exercise bout. Inspection of running mechanics changes from the onset to the end of each 4-min interval during the present HIIE indicates that running pattern is not largely altered. That said, a unique and important finding was that vertical mean loading rate increased by ~10%, irrespective of exercise bout or FiO_2. This increase (already visible during the initial interval) is substantially larger than the ~1% increase from the first to the last interval of a HIIE composed of six 30-s runs with 30 s of rest.[3] Using HIIE with long intervals, experienced runners may produce gait patterns within each interval that would result in more stressful ground impacts. Other explanations may lie with the inherent characteristics of running shoes (i.e., not standardized across participants) and the foot strike pattern of the tested runners (i.e., not assessed), which may slightly alter loading rate values.[24] This result is clinically relevant given that higher vertical loading rates have been associated, among other medical conditions, with patellofemoral pain and/or history of *plantar fasciitis*.[25,26]

One strength of our study is that running mechanics were derived from direct ground reaction forces recordings in both vertical and antero-posterior directions, as opposed to the majority of available HIIE studies using high-speed cameras to measure sagittal-plane kinematics.[22,23] From the onset to the end of 4-min intervals we observed a lengthening of the push-off phase duration (+2%) accompanied by lower peak push-off forces (–4%). For the first time with HIIE, our unique antero-posterior force data indicate that fatigue-induced modifications within each interval primarily occur during the push-off as opposed to the braking phase. More specifically, with acute fatigue, participants modify their foot strike pattern by pushing less ‘forcefully’ forward in the horizontal direction.

Our conclusions must remain specific to the athlete cohort and experimental conditions of this study. Perhaps our experienced runners may have taken advantage of the moving belt on the treadmill, for instance by increasing stride length to cope with fatigue development as opposed to stride frequency, compared to running on the solid ground.[27,28] That said, the absence of significant changes for contact and aerial times during the course of the proposed HIIE seems to contrast with our previous observations obtained during six 30-s treadmill runs with 30 s of recovery.[3] This would indicate that our tested athletes did not strategically alter their gait as fatigue developed so that more time was spent airborne to allow the treadmill to pass under them. We[29] and others[30] also found that the stride mechanical pattern of elite athletes is not detrimentally affected by completion of an altitude training camp, while these observations were not derived from a HIIE with long intervals as performed in this study. Finally, the lack of any systematic or progressive changes in running mechanics might in part be attributable to a large inter-individual and intra-individual variability.

5. Conclusion

This study is the first to describe alterations in running mechanics patterns within and between intervals during perceptually-regulated HIIE (4 × 4-min intervals at RPE 16; rest = 3 min) in hypoxia and normoxia. The most important finding was that the addition of hypoxia does not alter running kinetics/kinematics or spring-mass characteristics substantially, when participants were tested for similar treadmill velocities. To limit the negative effect of high running velocities on mechanical constraints, future studies should determine if exercising in an O_2-deprived environment at slower velocities represents an effective strategy to reduce the load across the lower extremity joints, while providing adequate (i.e., similar to faster running velocities near sea level) physiological stimulus for physiological adaptations.

6. References

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7. Figure legends

Figure 1 – Running velocity (A), heart rate (B) and arterial oxygen saturation (C) during the high-intensity intermittent running protocol in hypoxia (black circles) and normoxia (white circles). Note that shaded areas correspond to periods of mechanical assessment conducted at constant velocity. Values are mean±SD (n=19).

 # denotes a statistically significant difference between conditions for a given interval ($P<0.05$); ^{1}, ^{2} and ^{3} denote a statistically significant difference vs. interval 1, 2 and 3, respectively ($P<0.05$).

Figure 2 – Stride kinematics and spring-mass characteristics measured at the onset and the end of each four, 4-min interval in hypoxia (black bars) and normoxia (white bars). Contact time (A), flight time (B), step frequency (C), step length (D), vertical stiffness (E) and leg stiffness (F).

Values are mean±SD (n=19).

Figure 3 – Stride kinetics measured at the onset and the end of each four, 4-min interval in hypoxia (black bars) and normoxia (white bars). Peak braking force (A), peak propulsive force (B), braking phase duration (C), propulsive phase duration (D) and vertical mean loading rate (E).

Values are mean±SD (n=19).

** denotes a statistically significant difference between onset and end ($P<0.05$).*