EFFECT OF FATIGUE ON RUNNING KINETIC ASYMMETRY IN NON-CONTINUOUS & CONTINUOUS CONSTANT SPEED PROTOCOL

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Kinetic asymmetries in running gait are well studied but their interaction with muscular fatigue, perceived exertion and/or metabolic stress, need further attention to understand and report limb laterality with fatigue progression during a non-continuous & a continuous constant speed protocols. For study, 13 young male runner's data for kinetic asymmetry (KA), stride rate (SR), blood lactate (BLa), muscular fatigue (RPE_M) and overall body feel (RPE_o) were collected. Kendall's tau-b reported a non-significant correlation between KA, SR and RPE_M and a significant correlation between BLa, RPE_O and RPE_M whereas Tuckey post hoc test revealed non-significant effect of RPE_M on KA and SR in both tests. Findings suggest that, observed KA with fatigue progression may be subject to inter-individual coordinated motor control based on overall running physiological state of body as there has no direct correlation been found with BLa, RPE_M and RPE_O in this study.

KEY WORDS: kinetic asymmetry, muscular fatigue, limb laterality, tibia impact

INTRODUCTION: Understanding kinetic asymmetry during running is important for preventing loading stresses, side to side kinematic adjustments and in resolving unilateral pathology. Kinetic asymmetry has also been identified as a risk factor as it may expose bones to higher loading stresses (Mizrahi, Verbitsky et al. 2000) and also rejects the hypothetical assumption of considering lower limb symmetry in order to address gait interpretations (Sadeghi, Allard et al. 2000). This acceptance of asymmetry due to limb dominance is not only important for clinicians, coaches and investigators but also important for researchers to understand its effect on biomechanics and how it could be related with fatigue.

In biomechanical literature, research studies have reported kinematic and kinetic asymmetries in runners (Vagenas and Hoshizaki 1992, Chang and Chiu 2010, Brown, Zifchock et al. 2014, Frayne 2014, Rumpf, Cronin et al. 2014, Pappas, Paradisis et al. 2015) and have tested variety of parameters, like leg stiffness, contact time, step length, flight time, joint kinematics, ground reaction force, vertical loading rates and tibia impact acceleration. For kinetic asymmetry evaluation in this study, tibia impact acceleration is considered due to its significant correlation with vertical ground reaction force (Lee, Chou et al. 2009), localized muscle fatigue (Flynn, Holmes et al. 2004) and overall body fatigue development (Mizrahi, Verbitsky et al. 2000, Derrick, Dereu et al. 2002).

In our knowledge, no study has evaluated kinetics asymmetry (KA) and its interaction with muscular rated perceived exertion (RPE_M), blood lactate (BLa) and overall rated perceived exertion (RPE_O) at individual's critical speed during a non-continuous and continuous fatigue protocol. As fatigue alters running mechanics in as little as 15 minute of running (Derrick, Dereu et al. 2002), it is hypothesized that, if dominant and non-dominant behaves differently during non-continuous and continuous speed protocol, they will fatigue at different rate and KA will be different. It is also hypothesized that KA may be linked with metabolic stress (BLa) and/or RPE_o and may give us a better explanation about the understanding of existence running asymmetry against fatigue and may also be used as a predictor of fatigue in future.

METHODS: A "Health History Questionnaire" and "Activity Questionnaire" was administrated to shortlist injury free active runners and written informed consent was signed by 13 participants (23.2±1.16 years, 173.4±5.95 cm, and 63.5±7.52 kg). Total 3 test sessions, with recovery period of 5-7 days in-between, were conducted: (1) $VO_{2submax}$ progressive treadmill run to exhaustion, (2) non-continuous constant speed test, and (3) continuous constant speed test. All these tests were performed under controlled laboratory conditions and

participants were instructed to wear same running attire and shoes and to have 4 hours of fasting before start of each test.

Test session 1 was executed to determine individual's onset of blood lactate accumulation (OBLA) speed (Ghosh 2004), known as critical speed, from $VO_{2submax}$ test (Gupta, Balasekaran et al. 2012). For session 2, treadmill (WOODWAY) was programmed to run at the individual's critical speed (3.73±0.44 m/sec) with 4 min active and 1 min resting intervals till volitional exhaustion. Whereas in session 3, treadmill (WOODWAY) was programmed to run at individual's critical speed continuously till volitional exhaustion. The test, in session 2 & 3, was terminated when individual's BORG's RPE scale (Borg 1998) reached 10.

For data collection in session 2 & 3, two Delsys TrignoTM IM sensors (±16g, 148 Hz) were mounted onto the tibialis anterior muscle, in the direction of tibia bone on both legs, to measure axial tibia impact acceleration during landing phase of running gait cycle. Tibia Impact acceleration was selected for KA evaluation due to its close association and significant identified correlation with ground reaction force (GRF) and peak plantar pressure (PPP) during running (Lee, Chou et al. 2009) and vertical jumping (Elvin, Elvin et al. 2007). whereas Borg RPE scale was used to measure RPE_M and RPE_O. During session 2, data was captured for 20 secs in last 30 secs before completion of each active stage and blood sample were collected during 1-minute resting intervals. Whereas in session 3, data was recorded after every 2 min, for 20 secs, till volitional exhaustion and blood samples were collected at start and end of the test, to determine the metabolic stress due to fatigue.

After data collection, IM data were filtered using 2nd order Butterworth filter with cutoff frequencies of 10-50Hz to remove noise, motion artifacts, dc content and tibia angular acceleration. Peak impacts were captured through peak detection algorithm followed by peak averaging algorithm for the recorded duration to determine the averaged right leg (I_{RT}) and left leg (I_{LT}) impacts. Kinetic asymmetry (KA) was calculated using [$KA= ((I_{RT}-I_{LT})/ (I_{RT}+I_{LT}))*100$] and stride rate (SR) was calculated using [SR = 3*(peak's count of both leg's recorded impacts)]. For statistical analysis, Kendall's tau-b correlation was computed between KA, SR, RPE_M, RPE_O and BLa, one-factor ANOVA was performed to determine significant difference in measured variables between participants and Tukey post hoc test was used to detect significant difference in variables of interest against RPE_M using $\alpha=0.05$.

RESULTS: The average duration for the participants to sustain with their critical speed was 37 minutes for non-continuous and 24 minutes for continuous test and participants experienced relatively higher metabolic stress in continuous test. The natural level of kinetic asymmetry at the start of test and kinetic adjustments along with RPE_M progression are shown in figure 1 for all participants during non-continuous and continuous speed fatiguing protocol.

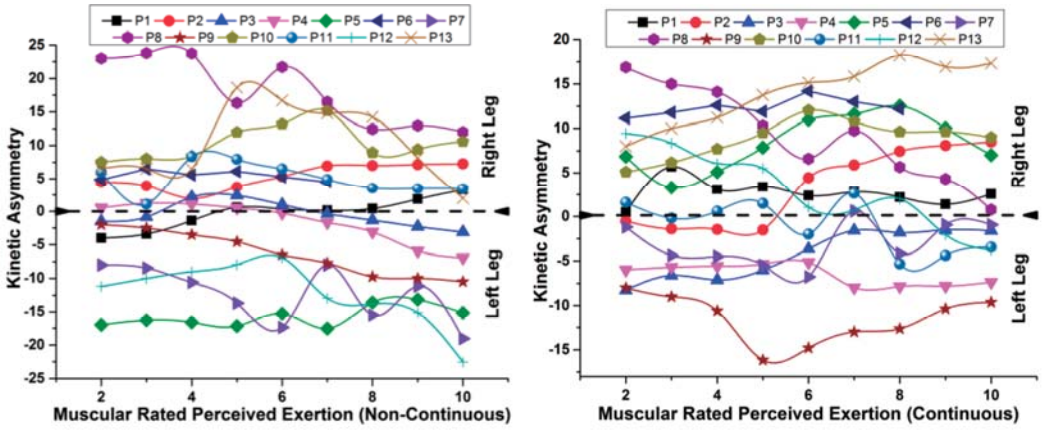

Figure 1: Kinetic asymmetry during landing phase of running gait cycle for selected participants during non-continuous (on left) and Continuous (on right) speed against RPE_M.

Mean±SD for variables of interest are shown in table 1, for both tests. No significant correlation has been found between KA, SR & RPE_M whereas BLa and RPE_O had a significant correlation with RPE_M in both tests. One-factor ANOVA with ($p<0.05$) reported a significant difference in the means of KA, SR and BLa response between participants whereas no statistically significant difference has been found in KA and SR at different levels of RPE_M.

Table 1

Variables presented as means ±SD, Kendall's tau-b correlation with RPE_M, and p-values of ANOVA b/w participants and p-value of Tuckey post hoc test against factor RPE_M.

	RPE_M (Non-Continuous Speed Protocol)					Kendal Tau_b Corr.	ANOVA b/w Participants (p value)	Tuckey post hoc (p-value)
	2	4	6	8	10			
KA	0.29 ± 9.81	0.98 ± 10.5	1.33 ±11.4	-.87 ±10.22	-2.32 ±10.88	-0.027	0.0001**	0.995
SR(min^{-1})	173.1 ±9.82	172.2 ± 9.1	171.9 ±9.11	172.15 ±8.44	171.7 ±8.13	-0.014	0.0001**	1.000
BLa (m.mol/l)	3.10 ±0.46	3.93 ±1.2	4.63 ±1.22	5.36 ±1.26	5.97 ±1.21	0.534**	0.0001**	0.0001**
RPE_O	2.38 ±0.51	4.46 ±0.52	6.53 ±0.77	8.6 ±0.77	9.7 ±0.59	0.903**	0.988	0.0001**
	RPE_M (Continuous Speed Protocol)							
KA	2.85 ±7.81	2.25 ±7.87	2.64 ±9.05	2.84 ±9.1	2.15 ±7.7	0.007	0.0001**	1
SR(min^{-1})	172.1 5±9.2	171.9 ±7.78	171.9 ±9.19	171.2 ±7.79	170.5 ±7.33	-0.074	0.0001**	1
BLa (m.mol/l)	--	--	--	--	7.29 ±2.14	--	--	--
RPE_O	2	4	6.23 ±0.59	8.53 ±0.66	10	0.956**	1	0.0001**

** Significant at the 0.05 level (2-tailed)

DISCUSSION: As leg dominance seems to be the function of type of activity (Velotta, Weyer et al. 2011), one objective was to report the existence of kinetic asymmetry during landing phase of running gait cycle. The observed findings have witnessed the existence of KA within almost all individuals and its behavior is also subject to change with fatigue progression, shown in figure 1. Though the sample mean±SD for KA did not show any significant correlation and/or statistically significant difference against RPE_M and BL_a, their might be no causal effect of RPE_M and BLa on KA. SR also had no significant correlation with KA, hence it is justifiable to say that findings in this study do not explain the probable reason for the observed changes in KA with fatigue progression. As kinetic asymmetry also accounts for neurophysiology and motor control (Sadeghi, Allard et al. 2000), the lack of collaboration between neuroscientists and biomechanists may also be the probable reason that laterality and it's interaction with fatigue in running mechanics is not well understood so far. This will leave an open question to the investigators/researchers regarding the cause of these kinetic asymmetries that is influencing motor control pattern for the lower extremity.

Existence of kinetic asymmetry has also been linked with higher tibia stress (Clansey, Hanlon et al. 2012), though there has been no strong evidence in studies (Bredeweg, Buist et al. 2013) to link kinetic asymmetry as a probable reason for injury development and/or to differentiate between injured and non-injured runners. Furthermore, as no significant relation has been found between KA and progressive fatigue measures in this study, probably KA cannot be used as a predictor of fatigue and/or be linked with higher tibia stress measure.

This study was not without limitations as it was conducted on motorized treadmill under laboratory controlled conditions and the absence of effect of fatigue may be due to selection of variables, utilized for comparison. Future studies will be more focused on validating the quality of movement science to quantify kinetic asymmetry and to have better understanding in relation with neuromuscular activation patterns for the lower extremity.

CONCLUSION: The natural level of kinetic asymmetry at baseline were different for selected participants and inter-individual differences were large with fatigue progression in both non-continuous and continuous speed protocols. However, these relationships did not remain significant once they were averaged for the selected group of participants and no significant effect of fatigue has been observed on kinetic asymmetry and stride rate. However, muscular fatigue had a strong correlation with metabolic stress and overall body feel. Considering the limitations and presented results, kinetic asymmetry cannot be used as a predictor of fatigue for the group of participants, selected in this study.

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