BAREFOOT RUNNING TRAINING: IMPLICATIONS FOR JOINT STIFFNESS AND MUSCULAR CO-ACTIVATION

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To determine the influence of barefoot training on neuromuscular co-activation of lower limb muscles on variables associated with injury risk during running. Trained shod runners (n=23) participated in a barefoot running program. Joint stiffness, agonist: antagonist co-activation and the co-activation index were calculated. A progressive barefoot training program induces longer co-activation of the shank complex and lower ankle stiffness in the barefoot condition. Footwear is implicated in changing injury risk factors whether soft tissue or bony related injuries. This paper suggests barefoot training as an avenue for rehabilitating ankle injuries as it promotes an even distribution of joint stiffness and lower ankle stiffness when compared to shod running.

KEY WORDS: electromyography, injury risk, exercise, biomechanics, shoes, footwear.

INTRODUCTION: Barefoot running has become popular based on the assumptions that it reduces the risk of injury and improves performance(Lieberman, 2012). However, the support for this is largely based on theoretical principles and lack of scientific studies supporting its benefits is scarce(Tam, Astephen Wilson, Noakes, & Tucker, 2014). Recently, researchers have found that acute barefoot running was associated with a longer co-activation index than shod running(Moore, Jones, & Dixon, 2014). This finding raises the question of how neuromuscular control and co-activation may influence joint stiffness and the risk of injury. Joint stiffness has been associated with injuries involving the knee and ankle during running where increased joint stiffness may be related to bony injuries whilst a decreased joint stiffness with soft tissue injuries(Butler, Crowell, & Davis, 2003). However, empirical evidence is sparse investigating the effect of footwear (or lack thereof) on co-activation and the associated kinematic and kinetic components that potentially may influence risk of injury. The relationship between the co-activation and joint stiffness is still poorly understood, it is hypothesized that an imbalance in muscle activation creates a net torque that exposes the joint to increased injury risk. The aim of this study was to examine the co-activation and joint stiffness changes associated with an eight-week progressive barefoot running program.

METHODS: Twenty-three trained runners (age: 29 ± 6 years; height: 175 ± 10 cm; mass: 72.6 ± 11.4 kg), participated in this study. All participants were habitual shod runners and had no prior barefoot training. Inclusion criteria for participants were defined as being able to complete a 10 km run in <50 minutes and running at least four times a week.
The intervention consisted of an 8-week progressive barefoot running training program. The running program introduced barefoot running training sessions in the participant's current shod running program which were introduced at the end of the normal shod training session, while the duration and intensity slowly increased over the training program to minimize injury risk. The program was also phased in to maintain typical volumes to avoid deconditioning and conditioning, so that the changes found could be attributed to barefoot running alone and avoiding control group recruitment. Participants were not briefed whether they should adopt any type of footstrike pattern and were instructed to run in a manner that maximized their subjective comfort.
Pre- and post- training intervention, all runners were tested while running barefoot and in their current training shoe. Running trials and gait analyses were conducted on a 60 m indoor

synthetic running track. At each visit participants completed 6 running trials in the barefoot and shod condition at 12 km·h^{-1}. Three-dimensional marker trajectories were captured using an eight-camera VICON MX motion analysis system (Oxford Metrics Ltd, UK), sampling at 250 Hz. Ground reaction force (GRF) data were collected using a force platform (AMTI, USA), sampling at 2000Hz, synchronized with the motion capture system. Kinematic and kinetic variables were resolved using the standard PlugInGait model.

Surface electromyography was measured in Bicep Femoris (BF); Rectus Femoris (RF); Lateral Gastrocnemius (LG); Medial Gastrocnemius (MG) and Tibialis Anterior (TA) according to SENIAM guidelines(Hermens, Freriks, Disselhorst-Klug, & Rau, 2000). After completing running trials at the designated speeds, the runners then completed three maximal sprints down the runway for subsequent normalization purposes.

Marker trajectory and kinetic data were filtered using a low-pass fourth-order Butterworth filter with a cut-off frequency of 8 and 60 Hz, respectively. Three-dimensional lower extremity joint angles and net resultant moments were calculated using a Newton-Euler inverse dynamics approach. Joint angles were described using the joint coordinate system. Three-dimensional muscle moments were expressed as Nm·kg^{-1}. Sagittal plane knee and ankle stiffness were calculated for load acceptance phase according to Hamill et al. (2014). The raw digital EMG signal of both sub-maximal and sprint trials processed according to Albertus-Kajee et al. (2011). Average EMG amplitude was calculated for pre-activation and stance phase, which was normalised to each participant's respective average contraction. Further, agonist:antagonist muscle pairs of RF: BF, MG:TA and LG: TA were computed for each phase(Kellis & Kouvelioti, 2009). Co-activation index (CI) were calculated following Franz et al. (2012). Two-factor ANOVA (condition x time) was used to assess differences between EMG and joint stiffness variables. Tukey's post-hoc analysis was used to identify specific differences when interaction effects were found. Relationships between co-activation (both CI and antagonist:agonist) and joint stiffness were further assessed with Pearson correlations. Differences were deemed statistically significant at $p<0.05$.

RESULTS: Agonist-antagonist and co-activation index differences were found between the footwear conditions and over time (Table 1). Joint stiffness differences were found between joints in the shod condition and footwear differences were also found in the ankle (Table 1).

Table 1
Neuromuscular and joint stiffness variables over training and in different footwear conditions

	BASELINE		POST TRAINING	
Agonist-antagonist ratio	**Barefoot**	**Shod**	**Barefoot**	**Shod**
LG:TA Pre-activation	0.41 ± 0.48*	0.24 ± 0.24	0.36 ± 0.42	0.20 ± 0.21
Weight Transfer	2.91 ± 2.15*	3.76 ± 2.76	2.75 ± 1.33	2.94 ± 1.75
Weight Acceptance	2.67 ± 1.22	2.38 ± 1.20	3.02 ± 1.53*	2.27 ± 1.21
MG:TA Pre-activation	0.30 ± 0.32	0.21 ± 0.13	0.57 ± 0.64*#	0.30 ± 0.32
Weight Transfer	3.04 ± 2.66	3.68 ± 2.84	3.13 ± 1.83	3.17 ± 1.84
Weight Acceptance	2.50 ± 1.25	2.32 ± 1.44	2.82 ± 1.43	2.29 ± 1.21
Co-activation Index (CI)				
LGTA (% stance)	66.11 ± 12.80*	61.64 ± 13.91	66.21 ± 11.78	62.73 ± 12.32
MGTA (% stance)	64.95 ± 10.94	61.80 ± 12.25	65.13 ± 11.92	62.76 ± 14.10
Joint stiffness				
Ankle Stiffness (Nm/^{o})	7.39 ± 2.29*	9.91 ± 3.61^	6.82 ± 1.83*	10.03 ± 3.32^
Knee Stiffness (Nm/^{o})	6.47 ± 2.01	6.07 ± 1.54	6.59 ± 1.77	6.13 ± 3.32

*footwear difference ($p<0.05$); # training difference ($p<0.05$); ^joint difference ($p<0.05$)

Negative correlations were found between the LGTA CI and ankle joint stiffness ($p<0.05$)(Figure 1).

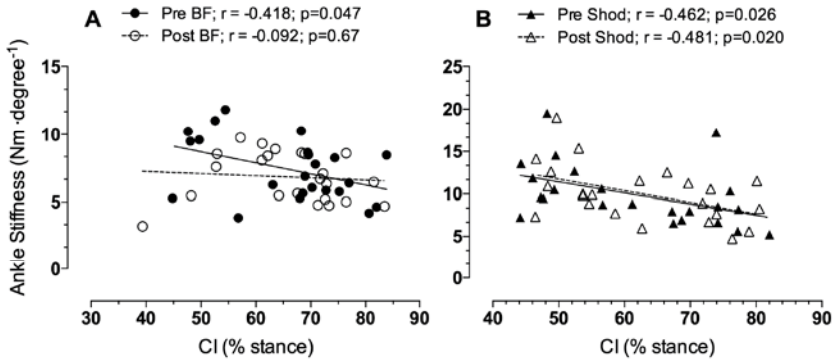

Figure 1: Ankle stiffness and co-activation index (CI) between footwear over the study.

DISCUSSION: The first finding was that ankle stiffness was greater in the shod condition regardless of the intervention. This finding is likely related to the increase in the sagittal ankle range of motion when running barefoot. Additionally, an uneven distribution of joint stiffness across the ankle and knee was also found when shod. An even joint stiffness distribution is favourable as it allows for a balanced force distribution across the joints. In the barefoot condition similar joint stiffness was observed across the ankle and knee. The increased ankle joint stiffness when shod is either an increase in joint moment or a decreased ROM. Our data suggest the latter, as shod ankle ROM was lower compared with the barefoot ankle ROM. It appears that shod running is associated with a decreased sagittal ROM, and a greater ankle joint stiffness, whereas when barefoot, a greater ROM in the ankle joint occurs than when shod. Thus, it was proposed that even though footwear affects foot strike pattern, footwear independently affects ankle angle, regardless of the strike type. Thus footwear selection influences joint stiffness distribution greater than how you run. It has been suggested that footwear does not affect sagittal knee angles(Fredericks et al., 2015). Our findings disagree, as shod knee flexion ROM was greater than when barefoot over the entire training study. Higher shod ankle stiffness maybe related to heel cushioning from the shoe this may decrease the somatosensory feedback and a subsequent decreased attenuation in ground reaction forces.

Longer barefoot LGTA co-activation was observed during pre-activation and may be a result of improved synchronization of the LG with the TA from the absence of cushioning and lateral stability from shoes. This finding also emphasizes increased co-operation between the two muscles over the entire stance phase with barefoot training and could directly relate to the foot strike pattern of the habitually shod runners, whom had a tendency to adjust their footstrike pattern to a midfoot-forefoot strike due to the barefoot training and continued this trend post-training. Additionally, barefoot foot strike angle was lower than shod, suggesting greater plantarflexion. Thus differences between footstrike patterns may explain the higher LG activation seen in the barefoot condition throughout the stance phase. Given that plantarflexion is associated with higher eccentric loads on the calf during the landing phase, gradual exposure would increase calf strength without damaging the muscle or tendon. Thus, longer co-activation may be a beneficial adaptation to barefoot running.

Pre-intervention TA activity played a dominant role in preparing the foot for ground contact noted by the lower MG:TA pre-activation ratio. Implying that before barefoot training greater dorsiflexion was observed when barefoot. However, TA pre-activation was lower after the barefoot training and MG pre-activation increased, suggesting an increase in plantarflexion prior to ground contact. Increased plantarflexion may predispose runners to ankle injury, as the ankle mortise becomes less stable and may increase risk of ankle injury. However, if neuromuscular control and strength are concomitant with barefoot training this may prove beneficial to running performance. Greater barefoot pre-activation LG:TA and MG:TA indicates an increased grastrocnemii activity suggesting increased plantarflexion at ground contact. It is understood that increased activation of plantar flexor muscles is necessary when barefoot to attenuate the heel impact with the ground, this adaptation is suggested to improve the stretch shortening cycle and facilitate better storage and elastic compliance of muscles. Lower barefoot LG:TA ratio during weight transfer prior to barefoot training maybe

due to rocker effect of the shod condition, which aids in forefoot roll-off suggesting decreased anterior musculature activity is required to propel the body forward. Whereas, when barefoot required active activation of the TA to propel the body forward into the next gait cycle.
During weight acceptance post-training LG:TA was higher when barefoot. This resulted from an increased LG activation, suggesting increased lateral stabilization of the lower limb as a result of a greater midfoot or forefoot strike pattern. Improved co-activation during the stance is vital, as lower limb joints are at their most vulnerable to the increased forces at initial ground contact. Further, the purpose of antagonist torque is to augment the ligament function in order to maintain joint stability and equalize the pressure distribution on the joint surface.
Lastly, an inverse relationship between joint stiffness and muscle co-activation index was found. This was only found in barefoot pre-training and over the entire study in the shod condition. Increased ankle joint stiffness was largely a result of a rearfoot strike and subsequent decreased ROM. Thus, this landing configuration required a shorter co-activation to transfer the foot through stance and it appears that shod ankle stiffness may not accurately require sustained co-activation to stabilize the joint in the sagittal plane.

CONCLUSION: Barefoot running training resulted in a longer co-activation of LGTA and a decreased joint stiffness at the ankle. Additionally, barefoot running relies on the simultaneous co-activation of agonist-antagonist muscle pairs whereas shod running utilizes joint stiffness through a decreased ROM to stabilize the ankle joint. Although the relationship between joint stiffness and co-activation is equivocal, it is suggested that co-activation at a joint is not necessarily to create a stiffer joint but instead to create a stable joint. Barefoot training may assist in rehabilitating ankle injuries as it promotes an even distribution of joint stiffness and lower ankle stiffness when compared to shod running.

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