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ATROPHY AND STRENGTH LOSS OF FOOT MUSCLES AND PLANTAR PRESSURE FOLLOWING 21-DAYS BED-REST

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SUMMARY
Immobilisation of the lower limb due to surgical treatment, orthopaedic devices and/or bed-rest may cause differential patterns of atrophy and strength loss in leg muscles. The current study has demonstrated that disuse of foot muscles during a period of 21-days bed-rest impaires foot muscle strength by 24% and decreases foot muscle volume by 8%. Changes in plantar loading were observed in the forefoot, in which intrinsic and extrinsic toe flexor muscles importantly contribute to load distribution during terminal stance of gait. Rehabilitative exercise programmes should consider the significant decrease in foot muscle function following immobilisation in order to prevent secondary failures of foot structures.

INTRODUCTION
Orthopaedic and surgical treatments require often prolonged bed-rest and/or immobilisation of the lower limb in orthopaedic devices in order to protect healing tissue. Whilst muscle atrophy may be expected as result of the injury itself, it has been demonstrated that unloading of the lower limb induces differential patterns of atrophy and strength loss in upper and lower leg muscles [1]. Bed-rest in healthy subjects is a model used by space-agencies to simulate the absence of weight-bearing activity during prolonged sojourns in microgravity environments. This model permits to investigate changes in musculoskeletal function caused by a disuse of the lower limbs. Previous studies have been showen that prolonged bed-rest induces greater atrophy of plantar flexor muscles followed by atrophy of knee extensor muscles and hamstrings, whereas, for example, ankle dorsal flexors are less involved [2]. In addition, the recovery of muscle size following bed-rest differs between individual leg muscles and may result in muscle imbalances [3]. Understanding the process of muscle atrophy during physical inactivity is important in guiding rehabilitative exercises programmes of the individual. Little is known about the changes in foot muscle volume and strength following unloading of the lower limbs. Along with the plantar aponeurosis, activities of intrinsic and extrinsic foot muscles play a decisive role in stabilisation of the different foot joints, alignment of the toes, metatarsal and mid-foot bones as well as for generation of an optimal lever arm during terminal stance of gait [4].
The nutrition countermeasures (NUC) 21-days bed-rest study at the German Aerospace Center Cologne provided the opportunity to examine the extent to which foot muscles are impaired by disuse and to investigate how plantar loading in the foot during gait is altered after immobilisation. The NUC study was originally designed to investigate the effect of a dietary countermeasure on bone demineralisation [5]. Effects on muscle volume and muscle function due to the nutrition countermeasure were not expected. We hypothesised that prolonged bed-rest decreases the size and strength of foot muscles and alters plantar loading in the foot during gait.

METHODS
Seven healthy male subjects (age: 30 ± 7 years, body-height: 1.81 ± 0.04 m, body-weight: 78.9 ± 6.4 kg) volunteered for the study. The subjects remained in a 6° head-down tilt bed-rest throughout the 21-days bed-rest period at all times and did not assume any weight-bearing posture or physical activities.
To evaluate intrinsic and extrinsic toe flexor strength, we determined the maximal plantarflexion moment at the metatarsophalangeal joints (MPJ) during maximal voluntary isometric contraction using a custom dynamometer (Fig. 1) and DaisyLab software. The subjects pushed their toes against the resistance of an angled toe-plate (MPJ: 25° dorsiflexion, ankle: neutral position) within 3-s and tried to hold a peak plateau for 2-3 s. The maximum was calculated as mean value of a 2 s-period at the peak plateau. Five valid trials were performed to obtain the best value for analysis.

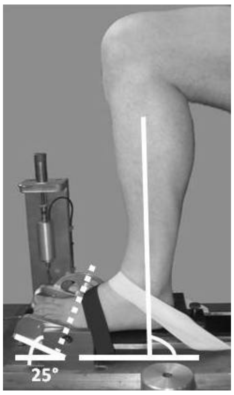

Figure 1: Dynamometer to quantify foot muscle strength.

The intrinsic foot muscle volume was measured using MRI at 1.5T with a circumferential extremity coil in a standardised foot position. Using gradient echo sequencing T1-weighted images were acquired in the coronal plane of the foot (TE = 2.49 ms, TR = 7 ms, field of view = 192 x 200 mm^2, slice thickness = 0.8 mm, spatial resolution = 0.82 mm x 0.82 mm x 0.80 mm, matrix = 240 x 250 pixels). Manual segmentation was performed using ImageJ software.

Coronal images were selected for muscle volume analysis of the foot according to anatomical landmarks between the first MPJ joint line and the first tarso-metatarsal joint line.

Plantar pressure distribution was measured by an emed-x platform (novel, 4 sensors/cm², 100Hz) mounted flush in the middle of the walkway. Using the third step method, five valid trials were collected. Contact time, peak pressure and pressure-time-integral for ten plantar foot regions and for the total foot were analysed (novel software).

Baseline data (BDC) were recorded up to seven days before bed-rest. Post bed-rest data were determined on the day of recovery from bed-rest (R+0) and on the first (R+1) and on the third (R+3) day of recovery from bed-rest. All investigations were performed in the dominant foot.

RESULTS AND DISCUSSION

The disuse of foot muscles throughout 21-days bed-rest caused a 24% decrease in foot muscle strength (BDC vs. R+0). The foot muscle strength remained reduced by 20.4% on the third postbed-rest recovery day (BDC vs. R+3) (Fig. 2).

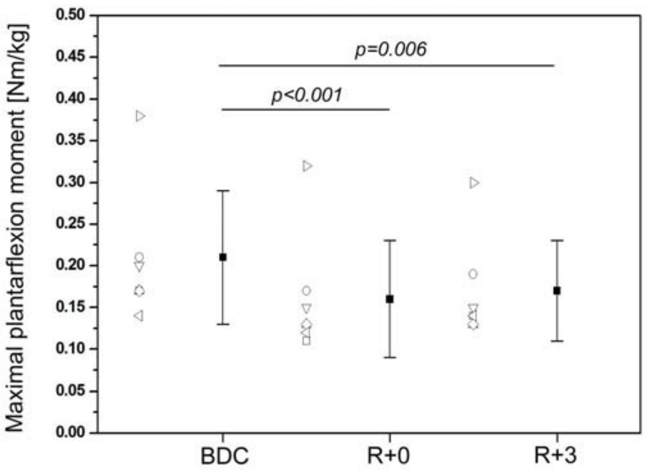

Figure 2: Values and mean values with standard deviations of the maximal plantarflexion moment (normalised to body-weight) at the metatarsophalangeal joints before bed-rest (BDC), on the day of recovery from bed-rest (R+0) and on the third (R+3) postbed-rest recovery day.

For technical reasons the intrinsic foot muscle volume could be quantified for only five subjects. It revealed an 8.5% decrease from 63.21 ± 6.13 cm³ to 57.85 ± 7.29 cm³ (p=0.043) following bed-rest (BDC vs. R+1).

Pressure distribution variables during gait for the total foot remained unchanged regarding peak pressure (BDC: 680.4 ± 241.5 kPa, R+3: 650.2 ± 155.5 kPa), contact time (BDC: 695.7 ± 63.4 ms, R+3: 679.4 ± 46.4 ms) and pressure-time-integral (BDC: 238.2 ± 46.0 kPa·s, R+3: 232.0 ± 36.2 kPa·s). At the ten plantar foot regions plantar pressure distribution changed solely in forefoot (Table 1). An increase in plantar peak pressure by 7% was found in central metatarsal region (BDC vs. R+3, p=0.018) following bed-rest.

Intrinsic and extrinsic foot muscles act as a single functional unit during the stance phase of gait and stabilise the medial longitudinal arch in the foot [5]. At terminal phase of stance, the resulting ground reaction force acts distal to the MPJ and generates a dorsiflexion moment at these joints, which is counteracted by an internal plantarflexion moment of the intrinsic and extrinsic toe flexor muscles as well as of tension of plantar aponeurosis. Cadaver models simulating terminal stance have been demonstrated, that impaired extrinsic toe flexor strength induces higher loads in the metatarsal region [4]. It can be inferred that the decreased strength of toe flexor muscles after bed-rest may account for the increase in peak plantar pressures in the central metatarsal region in the current study. Impaired foot muscle strength may substantially alter the mechanical load of metatarsal and mid-foot bones as well as of passive structures and plantar soft tissues in the foot. Foot muscle atrophy is a common clinical complication of the diabetic neuropathic foot and was associated with increased plantar peak pressure and ulceration risk in the forefoot. Foot muscle strength has been showen more than 50% lower in diabetic neuropathic patients [7] compared with the current postbed-rest findings. Thus, the effects of impaired foot muscle function regarding a redistribution of plantar pressures may much higher in those patients.

CONCLUSIONS

Prolonged bed-rest induces significant atrophy and strength loss of foot muscles and redistribution of plantar pressures in the forefoot. Rehabilitation programmes should consider the impaired foot muscle function following immobilisation in order to prevent secondary failures of foot structures.

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Table 1: Mean values and standard deviations of plantar pressure distribution variables at the forefoot: relative contact time (% roll-over process), peak pressure (kPa) and pressure-time integral (kPa·s) at the baseline data collection before bed-rest (BDC) and on the third postbed-rest recovery day (R+3)

	Contact time (% ROP)		Peak pressure (kPa)		Pressure-time integral (kPa·s)	
	BDC	R+3	BDC	R+3	BDC	R+3
Metatarsal 1	80.4 ± 2.6	78.7 ± 1.5 *	377.3 ± 201.9	347.8 ± 177.0	105.6 ± 41.5	94.9 ± 37.2 *
Metatarsal 2	82.8 ± 2.2	81.3 ± 1.0 *	427.4 ± 161.0	457.4 ± 163.6 *	118.8 ± 40.4	117.5 ± 41.7
Metatarsal 3, 4, 5	85.2 ± 1.8	84.0 ± 1.6	477.6 ± 261.2	469.1 ± 187.2	138.4 ± 55.2	139.9 ± 52.8

* Statistically significant differences between BDC and R+3 (p<0.05)