# Neuromuscular Adaptations Following 90 Days Bed Rest With or Without Resistance Exercise

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**INTRODUCTION:** This study examined the effects of long-term bed rest with or without a concurrent resistance exercise protocol on different muscle function indices of the knee extensors and their influence on previously shown atrophy, neural impairment, and slow-to-fast phenotype shift.

**METHODS:** Nine men underwent 90 d of bed rest only (BR), while eight men in addition performed maximal supine squats every third day (BRE). Before and at day 1 and 5 following bed rest, surface quadriceps electromyographic (EMG) activity was measured during a sustained (60-s) submaximal isometric action and rate of force development (RFD) was assessed during a maximal isometric action, both in the supine squat position. Maximal torque was measured during isokinetic knee extensions at different angular velocities before and after (day 2 and 11) bed rest.

**RESULTS:** EMG amplitude at a fixed submaximal load increased in BR, but not in BRE. The increase in amplitude during the sustained action was elevated in BR but not in BRE. RFD decreased in BR; this effect was attenuated day 1 and normalized day 5 in BRE. RFD expressed relative to maximal force was maintained in both groups. Angle-specific torque decreased equally for all velocities in BR. The decrease in isokinetic strength was attenuated day 2 in BRE.

**DISCUSSION:** Phenotype changes were not reflected in muscle function measurements, probably because they were overridden by the effects of atrophy and neural adaptation. The protective effect of resistance exercise was more pronounced in tasks similar to the training action, inferring great impact of neural mechanisms.

**KEYWORDS:** electromyography, fatigue, force-velocity, rate of force development, spaceflight.

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A transition from slower to faster muscle fiber types has been evident following hind-limb suspension in lower mammals,[37] but previous human studies have not revealed such a change, e.g., Bamman et al.[7] and Berg et al.[8] However, we showed that 90 d of bed rest provoked a clear shift toward faster myosin heavy chain (MHC) isoforms in the vastus lateralis (VL) muscle.[15] Moreover, type I fibers tended to atrophy more than type II fibers.[32,39] Such a shift could be expected to be manifested by changes in different muscle function indices, e.g., the torque-velocity relationship, rate of force development (RFD), and muscle fatigue.[5,38] Following 90-d bed rest, there was a substantial atrophy and reduced electromyographic (EMG) amplitude at maximal efforts, leading to decreased maximal strength.[3] In the present paper, we examine whether the MHC-shift in these subjects is reflected in the in vivo muscle function parameters stated above, or if it is overridden by the atrophy and neural adaptations.

The fact that following muscle unloading, maximal EMG amplitude is decreased[3,8] and voluntary force is more compromised than electrically elicited force[21] suggests that the neural drive is reduced. From studies which employed surface EMG or functional magnetic resonance imaging (MRI), it also appears additional motor units must be recruited to aid in performing a particular submaximal motor task following unloading[9,30] or spaceflight.[23] Along with these changes, the decrease in mean power frequency of the EMG signal that typically accompanies the amplitude increase during sustained fatiguing isometric actions is more pronounced following short-term

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bed rest.[31] Further, a decay in RFD at onset of a maximal isometric action has been observed.[7,24] Collectively, these reports suggest that chronic unloading provokes alterations in motor control in exercise tasks calling for modest or all-out effort.

Resistance exercise (RE) performed every third day during the 90-d bed rest period maintained quadriceps muscle volume, maximal training-specific strength, and maximal EMG amplitude.[3] The slow-to-fast phenotype shift was attenuated but present,[15] and type I fibers were not fully protected by the RE regimen, in contrast to type II fibers.[39] These data propose that the muscle would become faster.

Strength losses in non-training-specific tasks were not entirely compensated for. This has also been shown following 2 wk of bed rest[6] and suggests an important neural adaptation. Specificity following RE involves differences regarding, e.g., unilateral vs. bilateral strength,[18] single vs. multijoint actions,[34] isotonic vs. isokinetic actions,[36] and concentric vs. eccentric vs. isometric actions.[19] In the present study we further evaluated the carryover effect from a concentric-eccentric maximal RE program to other tasks involving maximal or submaximal isometric or isokinetic actions and possible effects of the phenotype shift.

The aim of the current investigation was to determine how different in vivo muscle function parameters were affected by the previously reported slow-to-fast fiber-type shift, neural adaptations, and changes in muscle size following 90 d of bed rest with or without resistance exercise. More specifically, surface EMG activity was measured during a sustained isometric action and rate of force development was measured at onset of a maximal voluntary isometric task. The force-velocity relationship was established by measuring torque at different angular velocities using isokinetic dynamometry. Given the findings mentioned above, we hypothesized that while performing a given isometric task, there would be an increased call for motor units to be activated following bed rest, and this effect would be abolished as a result of the RE protocol. We also speculated that any increase in EMG amplitude shown during sustained contractile activity would be exacerbated following bed rest, yet unaltered when resistance exercise was performed. It was further hypothesized that the RFD would be slightly enhanced following bed rest with concurrent resistance exercise, but severely impacted if no exercise was performed. When expressed in relation to maximal force, we expected an increase in both groups. Finally, we hypothesized that bed rest would result in a decreased isokinetic torque that would be greater at lower angular velocities, but that this impairment would be attenuated but still present when performing RE.

## METHODS

### Subjects

Seventeen healthy men (26–41 yr) gave their informed consent to participate in this study. Their physical status ranged from sedentary to physically active, they had no drug addiction, and no personal nor family record of chronic or acute disease which could affect data or create a risk for the subject during the experiment. Nine subjects were confined to bed rest (BR; 32 ± 4 yr, 173 ± 3 cm, and 72 ± 5 kg), while eight men in addition performed resistance exercise (BRE; 33 ± 5 yr, 176 ± 5 cm, and 71 ± 6 kg). One BRE subject was excused from isokinetic measurements due to knee pain during this specific task. The protocol was approved by the local Ethical Committee in Toulouse (le Comité Consultatif de Protection des Personnes dans la Recherché Biomédicale (C.C.P.P.R.B.) de Toulouse I), was conducted in accordance with the declaration of Helsinki, and complied with the laws of France.

### Equipment

All training and testing was carried out in the 6° head-down tilt position on a flywheel ergometer that has been described in detail previously.[2] Briefly, by the aid of rotating flywheels, concentric and eccentric force can be produced. By keeping flywheels stationary, isometric actions can be carried out as well. Force was measured by the aid of a load cell (ELA-B2E-2KL, Entran, Fairfield, NJ), joint angles with electrogoniometers (Ergotest AS, Langesund, Norway), and data collected using a Windows™ based data acquisition system (MuscleLab™, Ergotest AS, Langesund, Norway). A screen faced the subjects to provide feedback. The isometric tests were performed in the supine squat position at a 90° knee joint angle. Unilateral (right limb) knee extensor torque was assessed using an isokinetic dynamometer (ID; Cybex 6000®, CYBEX International Inc., Medway, MA). Seated knee extensions were executed at a 90° hip angle with arms crossed and straps used to fix the chest, hip, and distal thigh.

### Procedures

The study was carried out at Space Clinic Medes at Rangueil Hospital, Toulouse, France. All subjects performed 6° head-down tilt bed rest during 90 d. This position was maintained at all times and subjects were always monitored to ensure compliance (for further details, see Alkner and Tesch[3]). In addition, BRE performed supervised resistance exercise for the knee extensor muscles every third day on the flywheel ergometer, comprising four sets of seven maximal repetitions with 2 min of rest between sets. Submaximal EMG measurements and RFD assessment were performed before (Pre) and on the first (R+0) and fifth (R+4) day of ambulation. Two familiarization sessions were followed by two test sessions during a 2-wk period prior to the bed rest. In addition, isokinetic torque was measured at different velocities prior to and on day R+1 and R+10 following bed rest. Standardized submaximal dynamic warm-up bouts on the flywheel device preceded all training and testing sessions.

Submaximal EMG measurements were performed as the subjects pushed with their feet against the platform of the flywheel device used in the fixed mode during 60 s with a load comparable to 30% of Pre maximal voluntary contraction (MVC). The same absolute level was kept for the post tests. This level has previously been proven to induce increases in EMG amplitude[28] and, by choosing this load, subjects would be able to keep the same absolute force for 60 s after bed rest as well, although working at a higher relative workload. Subjects were instructed to keep the force at its target value with the aid of the visual feedback.

EMG activity was recorded from both limbs (mm. vastus lateralis, VL; midthigh and vastus medialis, VM; about 5 cm proximal to the patella). Positions were kept for post measurements by means of marks on a plastic sheet using anatomical landmarks for guidance. Before testing, electrode sites were shaved and cleaned with alcohol. Disposable bipolar Ag-Ag/Cl surface electrodes with 25-mm interelectrode distance (Multi Bio Sensors Inc., El Paso, TX), were aligned longitudinally in the fiber direction. Reference electrodes were placed over the tibiae bone. Within the EMG acquisition system, raw EMG signals were amplified 600 times and filtered through a band-pass filter with low and high cut-off frequencies of 6 and 1500 Hz, respectively. The filtered signal was converted to a root mean square (RMS) signal using an AD536 circuit (Analog Devices Inc., Norwood, MA) with an averaging constant of 100 ms. The converted signal, RMS of the raw signal, was then sampled at 100 Hz together with force and joint angle signals using the MuscleLab system.[4] Average values were established from the 0–10 s and 50–60 s intervals. As no difference in response was found between the muscles, RMS values were averaged over the two muscles VL and VM, and also over the right and left leg. The EMG/force ratio was assessed and the difference in amplitude between the first and last 10-s period was calculated as an index of fatigue. Day to day variation (coefficient of variation; CV) for EMG amplitude was 10.3% for the first 10 s and 11.1% for the last 10 s.

Measurements of RFD were carried out on the flywheel device. With the aid of the feedback screen, the subject pushed isometrically on the platform with a load comparable to 20% of Pre MVC. This force level was kept by the subject until a command from the operator and then he pushed immediately with maximal force. By starting at 20%, we aimed to minimize time delays due to compliance in the device and in the musculotendon complex.[24] Two trials were allowed and, if the force differed more than 5%, or if obviously unsuccessful in generating force quickly, additional trials were performed. The force/time ratio was calculated for five different time points (0.1, 0.2, 0.3, 0.4, and 0.5 s) from the start of the action. The trial which produced the best RFD value was chosen for comparison, provided that force was within 5% of MVC. Moreover, unsuccessful attempts where the force curve did not show a continuously increasing force were disregarded. Day to day variations (CV) of the rates were 11.8, 8.8, 6.9, and 6.4% at the time points 0.2, 0.3, 0.4, and 0.5 s, respectively. The 0.1-s time point was excluded from the statistical analyses due to a too large CV (19.1%).

In the isokinetic device, measurements of MVC were performed at a 120° (60° below horizontal) knee joint angle and of concentric torque at 30, 60, 90, 120, 180, and 300° $\cdot$ s$^{-1}$ (0.52, 1.05, 1.57, 2.09, 3.14, and 5.24 rad $\cdot$ s$^{-1}$) in the range of a 90°–180° knee angle. In any mode, two trials were allowed and if peak force differed more than 5%, additional trials were performed. Verbal encouragement but no visual feed-back was provided. Angle-specific torque was established at 120° for each action. Day to day variation (CV) prior to bed rest ranged from 6.6–12.1%. Isometric torque and concentric peak torque averaged over all velocities have previously been reported together with eccentric peak torque.[3]

MHC distribution, MVC, and muscle volume data were previously published[3,15] and are presented here in brief for reference. Muscle samples were obtained from the right VL using a percutaneous needle biopsy technique about 1 wk prior to and on day 84 during bed rest. The MHC isoform profile for each fiber was determined by dissecting individual fibers under a microscope and subjecting fibers to sodium dodecyl sulfate-polyacrylamide gel electrophoresis. An average of 110 $\pm$ 5 fibers (3-4 mm in length) were studied from each of the pre and post bed rest muscle samples. For a detailed method description, see Gallagher et al.[15]

MVC was tested using the flywheel device in the same position as in the tests described above. Two trials were allowed and, if force differed more than 5%, additional trials were performed. Subjects were asked to increase force smoothly and maintain maximal force for about 3 s. Verbal encouragement but no visual feedback was provided during this test. Force and EMG was averaged over the 1000-ms time window showing the highest mean force.

Muscle volume of the mm. quadriceps femoris was measured before and on day 89 during bed rest using MRI. The quadriceps muscle was circumscribed in repeated cross-sections and the muscle volume was subsequently calculated (for further details, see Alkner and Tesch[2,3]). In addition, using the same set of MR images, the thickness of the subcutaneous fat layer was measured. Guided by scout images and anatomical landmarks, the sites for EMG electrode placement were identified and the underlying fat layer subsequently measured using a Windows-based software program (Scion Image Beta 4.0.2 for Windows, Scion Corporation, Frederick, MD).

### Statistical Analysis

Values presented are mean $\pm$ SD. Using the software Statistica™ (StatSoft, Inc, Tulsa, OK), a repeated measures, two-factorial ANOVA was employed to detect group*time interaction. If a significant interaction was seen, planned comparisons were performed to detect where the differences occurred. Changes over time were determined for each group. Differences between the relative changes of the torque at different isokinetic velocities were detected using repeated measures ANOVA. Bonferroni corrections were made for each level of comparison. Statistical significance was set to $P < 0.05$.

Degrees of freedom were 15 in flywheel measurements and 14 in isokinetic measurements, 1 for group effects, 2 for time effects, and 6 for velocity effects. CV was calculated by dividing the SD by the mean and multiplying by 100 for each individual [(SD $\cdot$ mean$^{-1}$) $\cdot$ 100] and averaging across subjects.

## RESULTS

The effects of unloading with or without resistance exercise on EMG amplitude during the sustained submaximal isometric action are illustrated in **Fig. 1**. In BR, EMG/force was greater for both time windows following bed rest (0–10 s: R+0: 70%, $F = 18.3$, $P = 0.0007$; R+4: 60%, $F = 12.4$, $P = 0.0031$; 50–60 s: R+0:

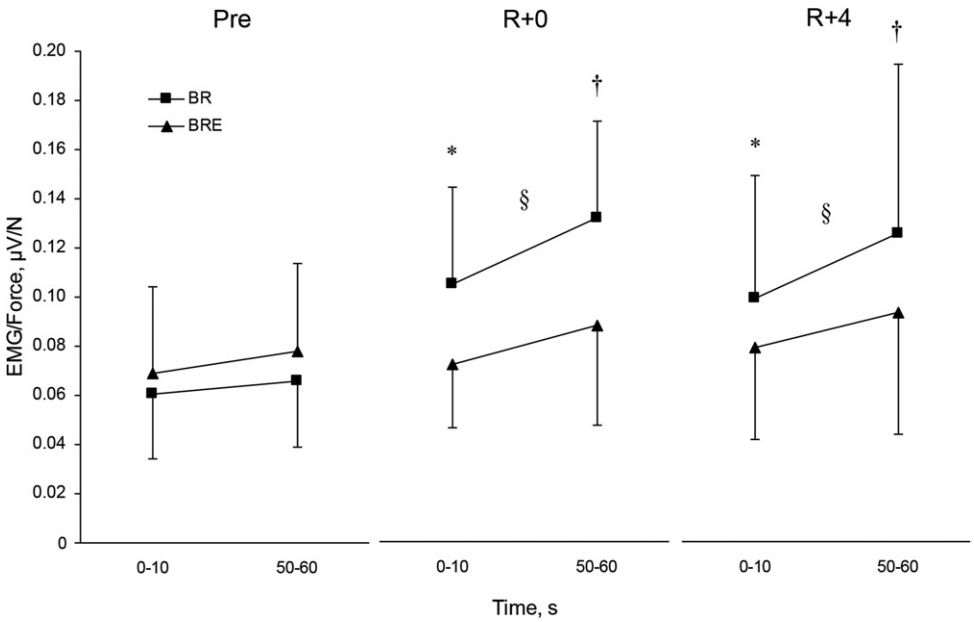


**Fig. 1.** EMG amplitude averaged over vastus lateralis and medialis bilaterally and divided by force. Data points are mean ± SD and are averaged over the first (0–10) and last (50–60) 10-s periods of a 60-s sustained contraction at 30% of maximal voluntary contraction as measured before bed rest. BR = bed rest only, BRE = bed rest and resistance exercise countermeasure, R+0 = first day of ambulation, R+4 = fifth day of ambulation. *Denotes a change ($P < 0.05$) from Pre in EMG amplitude for the first 10-s period; †denotes a change ($P < 0.05$) from Pre in EMG amplitude for the last 10-s period; §denotes a change ($P < 0.05$) from Pre in increase between the two time periods.

95%, $F = 31.5$, $P < 0.0001$; R+4: 88%, $F = 16.4$, $P = 0.0011$), while BRE did not show such a change (0–10s: R+0: $F = 0.542$, $P = 0.473$; R+4: $F = 0.766$, $P = 0.395$; 50–60 s: R+0: $F = 3.447$, $P = 0.0831$; R+4: $F = 0.773$, $P = 0.393$).

The increase in EMG/force over the 60-s isometric action was greater (R+0: $F = 8.20$, $P = 0.0183$; R+4: $F = 12.7$, $P = 0.0028$) compared to pre in BR, as illustrated by the steeper slope of the curve. BRE did not show a greater increase over time following bed rest (R+0: $F = 3.553$, $P = 0.0789$; R+4: $F = 0.357$, $P = 0.559$).

EMG amplitude of the first 10 s, expressed relative to EMG during MVC,[3] amounted to 20, 57, and 41% in BR and 23, 29, and 28% in BRE on days Pre, R+0, and R+4, respectively. The force produced (30% of Pre MVC) was 467 ± 116 N in BR and 462 ± 62 N for BRE, corresponding to 56% and 48% of MVC in BR and 40% and 38% in BRE, on day R+0 and R+4, respectively.

In the RFD measurements (**Fig. 2A**), BR showed a decrease in force increment over time over all time points (R+0: 46–47%; 0.2 s: $F = 47.0$, $P < 0.0001$; 0.3 s: $F = 70.3$, $P < 0.0001$, 0.4 s: $F = 68.3$, $P < 0.0001$; 0.5 s: $F = 60.1$, $P < 0.0001$; R+4: 38–43%; 0.2 s: $F = 31.8$, $P < 0.0001$; 0.3 s: $F = 41.0$, $P < 0.0001$; 0.4 s: $F = 41.6$, $P < 0.0001$; 0.5 s: $F = 43.7$, $P < 0.0001$) while BRE demonstrated a decrease only at R+0 (20–29%; 0.2 s: $F = 17.9$, $P = 0.0007$; 0.3 s: $F = 19.9$, $P = 0.0005$; 0.4 s: $F = 11.2$, $P = 0.0044$; 0.5 s: $F = 11.5$, $P = 0.0040$), but not at R+4 (0.2 s: $F = 1.10$, $P = 0.310$; 0.3 s: $F = 1.29$, $P = 0.273$; 0.4 s: $F = 0.692$, $P = 0.418$; 0.5 s: $F = 0.801$, $P = 0.385$). The decrease was greater for BR at all time points except 0.2 s at R+0 (0.2 s: $F = 2.63$, $P = 0.12$; 0.3 s: $F = 6.26$, $P = 0.0244$; 0.4 s: $F = 10.4$, $P = 0.0056$; 0.5 s: $F = 8.13$, $P = 0.0121$), and at all time points at R+4 (0.2 s: $F = 9.62$, $P = 0.0073$; 0.3 s: $F = 12.7$, $P = 0.0028$; 0.4 s: $F = 14.6$, $P = 0.0017$; 0.5 s: $F = 15.1$, $P = 0.0015$). When normalized to maximal force (**Fig. 2B**), no group effect was seen ($F = 2.7355$, $P = 0.119$) and neither group showed any change (BR: R+0: 0.3 s: $F = 3.26$, $P = 0.0911$; 0.4 s: $F = 3.88$, $P = 0.0676$; 0.5 s: $F = 5.34$, $P = 0.0355$; R+4: 0.3 s: $F = 2.39$, $P = 0.142$; 0.4 s: $F = 0.0937$, $P = 0.764$; BRE: R+0: 0.3 s: $F = 3.57$, $P = 0.0782$; 0.4 s: $F = 1.50$, $P = 0.239$; 0.5 s: $F = 1.64$, $P = 0.219$; R+4: 0.2 s: $F = 1.10$, $P = 0.310$; 0.3 s: $F = 0,0129$, $P = 0.911$; 0.4 s: $F = 0.094$, $P = 0.764$; 0.5 s: $F = 0.0321$, $P = 0.860$), except that a reduction was seen in BR at time points 0.2 s (R+0: 5%, $F = 47.0$, $P < 0.0001$; R+4: 6%, $F = 31.8$, $P < 0.0001$) and 0.5 s (R+4: 14%, $F = 7.80$, $P = 0.0137$), and in BRE at 0.2 s (R+0: 10%, $F = 17.9$, $P = 0.0007$). The values for rate of force development from 0–0.2 s averaged for BR was 3525 $N \cdot s^{-1}$ prior to bed rest and 1915 and 2178 $N \cdot s^{-1}$ at day R+0 and R+4, respectively, following bed rest. For BRE these values were 3643, 2589, and 3377 $N \cdot s^{-1}$, respectively. For the time period 0–0.3 s, the values were 2818, 1493, and 1706 $N \cdot s^{-1}$ for BR and 2917, 2168, and 2708 $N \cdot s^{-1}$ for BRE. For the time period 0–0.4 s the values were 2367, 1281, and 1408 $N \cdot s^{-1}$ for BR and 2384, 1917, and 2253 $N \cdot s^{-1}$ for BRE. For the time period 0–0.5 s the values were 2029, 1065, and 1166 $N \cdot s^{-1}$ for BR and 2024, 1577, and 1900 $N \cdot s^{-1}$ for BRE.

In the isokinetic measurements, BR showed a decrease in angle-specific torque (120°) for each velocity from Pre to R+1 (46–52%, $F = 118$, $P < 0.0001$) and R+10 (25–32%, $F = 0.273$, $P = 0.610$; **Fig. 3A**). The decrease in BRE was less than for BR at R+1 (25–32%, $F = 8.77$, $P = 0.0103$), but similar at R+10 (20–27%, $F = 0.273$, $P = 0.610$). No significant interaction was detected for the changes for the different velocities ($F = 0.6757$, $P = 0.6695$), as also illustrated by the normalized curve (**Fig. 3B**).

Both BR and BRE showed a significant decrease in MHC I fibers of 15–16% while only BR had a decrease in MHC IIa fibers (−14%). Both BR and BRE had more hybrid fibers following bed rest and the increase was greater ($P < 0.05$) in BR (+29%) than in BRE (+12%). For the individual hybrid fiber types, there were increases in MHC I/IIa, IIa/IIx, and I/IIa/IIx fibers in BR, but not in BRE.[15]

MVC decreased significantly by 45% at R+0 and 36% at R+4 in BR, but was maintained in BRE. Quadriceps muscle volume decreased 18% in BR while BRE showed no change ($P > 0.05$). There was a group difference for both parameters.[3] The thickness of the subcutaneous fat layer was unchanged at all electrode

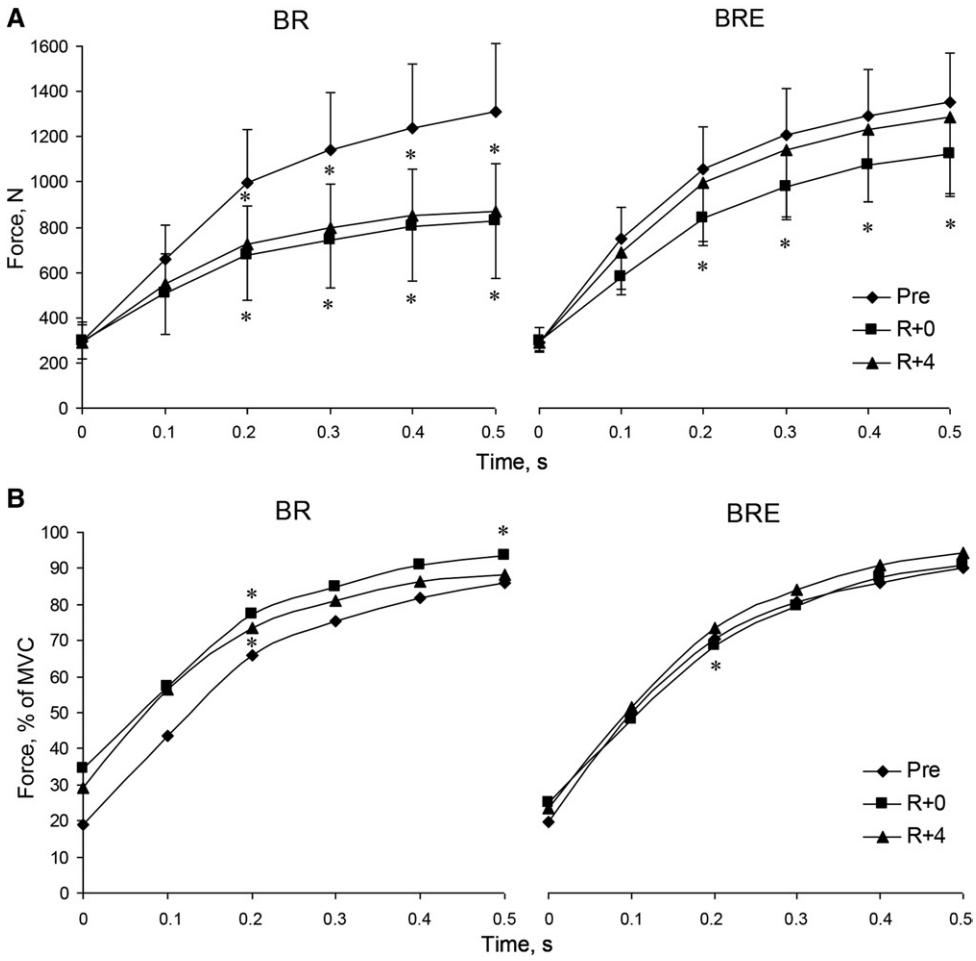


**Fig. 2.** Force produced from a starting level of 20% of Pre MVC at five different time points during a rapid isometric action. Data points are mean. A) Absolute values ± SD and B) values relative to maximal force. BR = bed rest only, BRE = bed rest and resistance exercise countermeasure, R+0 = first day of ambulation, R+4 = fifth day of ambulation. *Denotes a change ($P < 0.05$) in slope (from 0 to respective time point) from Pre (no statistics were performed at the 0.1-s time point).

sites for both groups ($F = 0.570$, $P = 0.462$). Thus, any effect on the EMG amplitude due to altered thickness of the subcutaneous layer[13,29] can be excluded.

## DISCUSSION

The present study showed decrements in muscular performance following 90 d of bed rest were diminished by resistance exercise with the protective effect of resistance exercise being more pronounced in tasks similar to the training action. In contrast to our hypothesis, phenotype changes were not reflected in muscle function measurements in either group.

As reported earlier, single muscle fiber properties are, to a large extent, determined by MHC composition.[10] A correlation between MHC composition and in vivo muscle function has been evident when studying individuals with different phenotype profiles, e.g., Andersen et al.[5] and Thorstensson et al.[38], but is not consistently found.[14,20] A concurrent change in MHC composition and the in vivo force-velocity relationship has also been evident following detraining after chronic RE.[5] It has been proposed that unloading-induced changes in fiber type composition will be reflected in muscle function, but previous human unloading studies have not provoked any significant phenotype shift, e.g., Berg et al.[8] We got a unique opportunity to study this relationship, as a more extended unloading intervention, i.e., 90 d of bed rest, induced a slow-to-fast fiber type shift.[15] However, the present data provides no evidence that this shift impacts in vivo muscle function. Hence, it appears that these effects were overridden by the atrophy and neural adaptations.

A substantial atrophy was caused by 90 d of bed rest[3] and, as a consequence of this, it is highly likely that additional high-threshold, fast motor units must be recruited[16] in order to exert a given force. Thus, the finding of an elevated EMG amplitude during a sustained submaximal action suggests there was a greater muscle involvement to produce this given force, and further agrees with data from the vast majority of previous studies which have investigated the effects of unloading[9] or spaceflight.[23] Similarly, with use of functional MRI,[30] it has been shown that a greater portion of the quadriceps muscle must be involved following 5 wk of unloading to accomplish a certain knee extensor task. The fact that maximal EMG amplitude decreased,[3] yet EMG for a given submaximal isometric action increased, would suggest that these changes truly reflect altered muscle activation induced by bed rest.

The increase in EMG amplitude that progresses during a sustained submaximal action is considered an established indicator of muscle fatigue.[12] Similar to us, Portero et al.[31] showed augmented fatigue response in plantar flexors when subjected to a sustained action at the same absolute load following 4 wk of bed rest. The augmented fatigue response accords with the shift toward more fatiguing fibers. However, BRE that also exhibited a slow-to-fast phenotype shift showed no increased fatigability, suggesting other factors to be of greater importance. The substantial atrophy seen in BR was abolished in BRE. Hence, while BRE produced the submaximal force with essentially unchanged muscle mass, BR produced greater relative force following bed rest (pre: 30% vs. post: 56% of MVC), with probably greater reliance upon fast fibers,[16] inducing aggravated fatigability and giving rise to increased surface EMG amplitude (present study and

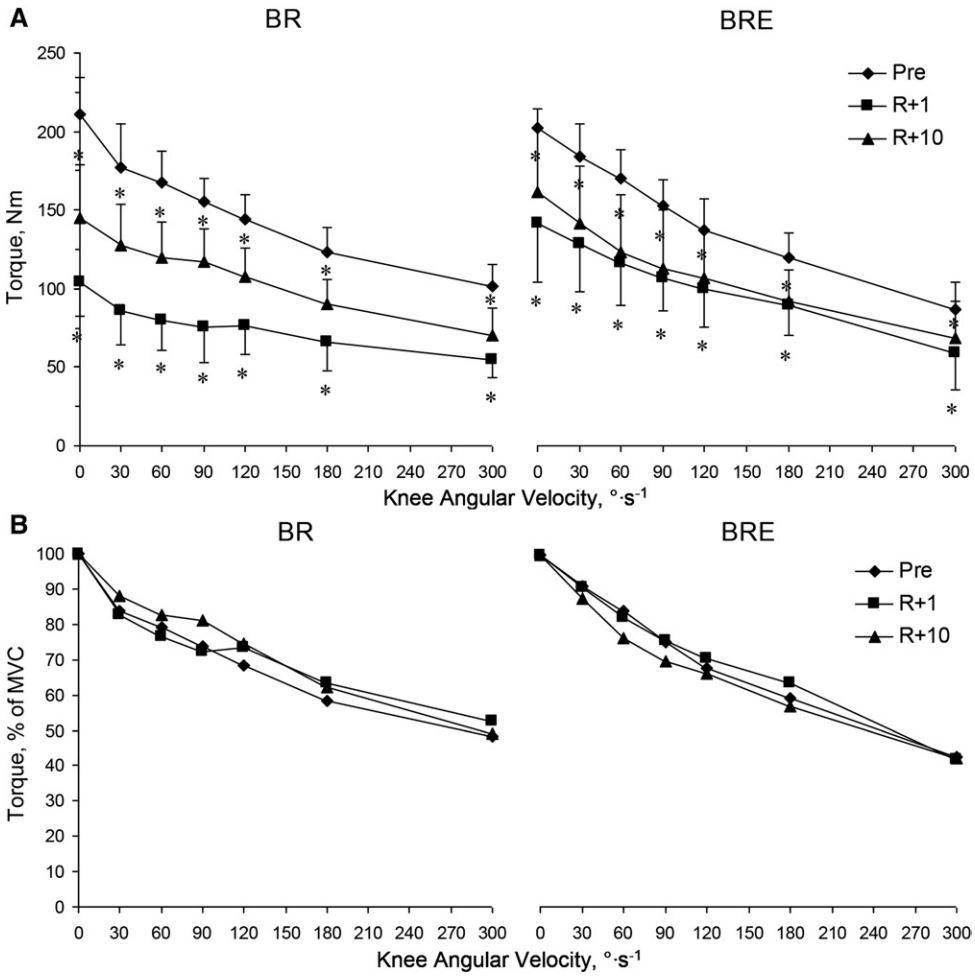


**Fig. 3.** Torque velocity relationship obtained in unilateral knee extensions with an isokinetic dynamometer. Values are mean for torque at a 120° knee angle. A) Absolute values ± SD and B) values relative to maximal isometric force. BR = bed rest only, BRE = bed rest and resistance exercise countermeasure, R+1 = 2nd day of ambulation, R+10 = 11th day of ambulation. *Denotes a change ($P < 0.05$) in torque from Pre.

Berg and Tesch[9] and Koslovskaya et al.[23]). The fact that the resistance exercise countermeasure abolished such an effect implies that the order and magnitude of motor unit recruitment, as well as fatigue susceptibility, were essentially unchanged in BRE.

For evaluating possible influence of fiber type shifts, it can be argued that we should have measured the fatigue response for the same relative load following as before bed rest. However, our intention was to study how a certain task could be performed following the simulated spaceflight, i.e., could the resistance training intervention maintain the ability to perform the same prolonged fatiguing action following the intervention. This was shown to be true and is of great importance when designing training programs for astronauts.

RFD normalized to MVC, which could be expected to increase with more fast contracting fibers remaining unaltered. This is concordant with some fragmental data on the plantar flexors[21] and even decreased normalized RFD has been present.[22] The possible effects of the phenotype shift might be offset by the decrease in specific force and power of the fibers,[39] decreased conduction velocity,[33] and/or by an impaired ability to recruit fast motor units. The absolute decrease in RFD of the knee extensors accords with results from previous short-term bed rest studies.[7,24] Such a reduction has also been shown for the plantar flexors following space simulation.[21] Though not thoroughly examined in long-duration studies, the protective effect of resistance exercise on RFD during unloading has been inferred[7,22] and is commensurate with the increased RFD noted in response to resistance exercise programs.[1] The ballistic actions performed here initially call for maximal or close to maximal motor unit activation[27] and clearly emphasize fast motor unit recruitment. However, our data cannot reveal whether the reduced RFD could mainly be attributed to the global muscle loss, including that of fast twitch fibers, reduced conduction velocity due to muscle fiber atrophy and modified membrane properties,[33] or failure to bring fast motor units into action. BRE, which maintained muscle mass and maximal strength, decreased in RFD at R+0 but equaled pre levels at R+4. This clearly suggests a failure to activate the motor units immediately after bed rest that was recovered 4 d later. Evidently, the present training regimen maintained the ability to perform this particular task quite well.

Isokinetic torque decreased uniformly over the different angular velocities, illustrated by the unaltered normalized curve (Fig. 3B). In agreement with the other functional data, this suggests no major impact of the MHC shift. As discussed for the RFD measurements, possible effects of the phenotype shift might be offset by the decrease in specific power of the fibers,[39] and/or by an impaired ability to recruit fast fibers. It can be argued that faster angular velocities, closer to maximal unloaded speed, are necessary to demonstrate such an effect.[26] Indeed, following 3 wk of one limb unloading, a less prominent decrease was shown at higher angular velocities (180°–500° · $s^{-1}$), but no fiber-type data was available.[35] Considering data from studies of similar duration, e.g., Bamman et al.[7], a significant phenotype-shift is unlikely, suggesting other mechanisms are responsible for the velocity-specific changes demonstrated in that study. In the present study, our data clearly suggest a uniform decrease in isokinetic strength over all velocities due to muscle atrophy and impaired neural drive.

In BRE, muscle function changes were abolished in some tasks but not in others, which is probably explained mainly by

neural adaptations according to the principle of specificity. The effects of resistance exercise are known to be most significant in tasks performed during training. In this particular 90-d bed rest study, maximal concentric-eccentric bilateral squats were effective in preventing decreases in force and power in training-specific tasks. In contrast, however, and despite maintained muscle size, unilateral knee extension torque assessed by means of isokinetic dynamometry was compromised. Similar findings have also been evident following short-term unloading[6,7] and is not surprising since previous resistance exercise studies have shown that bilateral training elicits less of an increase in unilateral compared with bilateral strength[18] and that strength increases resulting from multijoint training are modest when assessed in a single- rather than multi-joint performance task.[34] It is also known that resistance training programs producing marked hypertrophy and increases in the weight that can be lifted promote no or a minute increase in maximal isokinetic torque.[36] In contrast, performance or muscle activation in other nontraining specific actions, i.e., sustained nonmaximal and maximal isometric actions, were found to be essentially unaltered. These data might appear somewhat puzzling, but may be explained by similarities in training and testing modes as all were performed as bilateral, multi-joint actions performed in the supine squat position. Moreover, both isometric multi-joint tests were performed at hip and knee joint angles that were within the range of that executed in the training mode. A significant portion of the training action, i.e., the transition from eccentric to concentric action and the subsequent early acceleration, was actually performed at a very low angular velocity close to an isometric action.[2] This accords with previous data that increases in response to chronic resistance exercise are most evident at joint angles[25] and angular velocities[11] applied during training. Task specificity is generally attributed to neural adaptations and even if the underlying mechanisms remain to be elucidated, we have shown that a dynamic resistance exercise program, emphasizing maximal voluntary concentric and eccentric actions, can transfer effects to different types of isometric actions calling for different motor unit recruitment strategies.[17]

The drop in isokinetic torque, which was greater in BR than BRE at R+1, did not differ between groups at R+10. The quick recovery in BR, as also seen in BRE for RFD, strongly suggests a neural adaptation. It can appear confusing that BRE increased RFD between post measurements with no change in BR while the opposite was true for the isokinetic measurements. It can be speculated that for RFD, the training effect allowed a quick recovery in BRE, while 4 d of ambulation was insufficient for recovery in BR. In contrast, 10 d of ambulation was enough to increase isokinetic torque. No improvement in this less training-specific test was seen in BRE and, also, torque did not significantly differ between groups at this time, though a presumable difference in muscle size indicates other compensatory mechanisms that remain to be elucidated.

In summary, the slow-to-fast phenotype shift was not reflected in muscle function alterations following 90 d of bed rest. Thus, fiber type shifts seem to play a minor role in explaining muscle function alterations following long-term muscle unloading and the effects are probably overridden by atrophy and neural adaptations. It can only be speculated that bed rest or spaceflight of even longer duration might induce such a great phenotype shift that it will affect muscle function. The resistance exercise program employed here, emphasizing coupled concentric and eccentric actions performed with maximal effort, did not only prevent atrophy and decreased maximal training-specific strength, but also performance and activation in tasks calling for different motor unit recruitment strategies performed in the training specific position. Performance in tasks more diverse from the training actions was, however, not fully maintained, which may suggest that additional training tasks are necessary to keep the quadriceps muscle function fully intact. Still, despite the long intervention period, resistance exercise performed every third day prevented or attenuated changes of muscle function, which is promising for future long-term space missions.

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