Training-Specific Adaptations of H- and Stretch Reflexes in Human Soleus Muscle

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ABSTRACT. The authors investigated the effect of physical exercise on reflex excitability in a controlled intervention study. Healthy participants (N = 21) performed 4 weeks of either power training (ballistic strength training) or balance training (sensorimotor training [SMT]). Both training regimens enhanced balance control and rate of force development, whereas reductions in peak-to-peak amplitudes of stretch reflexes and in the ratio of the maximum Hoffman reflex to the maximum efferent motor response (Hmax:Mmax) measured at rest were limited to SMT. The differences in reflex excitability between the training regimens indicated different underlying neural mechanisms of adaptation. The reduced reflex excitability following SMT was most likely induced by supraspinal influence. The authors discuss an overall increase in presynaptic inhibition of Ia afferent fibers as a possible mechanism.

Key words: balance, motor control, sport

Recent growing evidence indicates that not only voluntary activation properties but also reflex excitability are specifically influenced following continuous physical exercise. The gain of the Hoffmann (H) reflex has frequently been assessed as a supposed electrical analog of the monosynaptic spinal reflex arc. Differences in the ratio between the maximum Hoffmann reflex and the maximum efferent motor response (Hmax:Mmax) were found between untrained and well-trained participants (Nielsen, Crone, & Hultborn, 1993), indicating changed maximum gains of the Ia afferent system (for a review, see Schieppati, 1987). Adaptations in reflex excitability that are specific with respect to the type of training have been demonstrated. Reduced reflex excitability for power- and balance-trained athletes has been found (Casabona, Polizzi, & Percivalle, 1990; Goode & Van Hoven, 1982; Koceja, Burke, & Kamen, 1991; Kyrolainen & Komi, 1994; Maffioletti et al., 2001; Nielsen et al.; Rochcongar, Dassonville, & Le Bars, 1979). The aforementioned studies were all cross-sectional, however, and therefore they did not allow the researchers to differentiate between neural plasticity, morphologic muscular adaptations, and genetic predispositions.

One could rule out the latter predisposition by performing longitudinal studies that address reflex modulation following strength training. Reduced peak-to-peak amplitudes of quadriceps tendon tap reflexes (T-reflexes) were observed after 16 and 24 weeks of high-intensity strength training (Hakkinen & Komi, 1983, 1986) and after 24 weeks of power training (ballistic strength training [BST]: Hakkinen & Komi, 1986). However, reflex adaptations following such prolonged training periods need not inevitably be neural, because the proportion of large-diameter muscle fibers was observed to increase after several months of training (Adams, Hather, Baldwin, & Dudley, 1993; Hakkinen & Komi, 1985). It has been argued that an increased number of high-threshold large motor units (MUs) after strength training could account for the decreased reflex excitability (for a review, see Burke, 1981). Furthermore, the aforementioned results originated from measurements obtained during rest. Similar measurements that were done during an active condition showed reversed effects. Increased Hmax:Mmax ratios were observed after 12 weeks of strength training for the soleus muscle when measured during a maximal isometric ramp contraction, whereas that ratio remained unchanged when measured during rest (Aagaard, Simonsen, Andersen, Magnusson, & Dyhre-Poulsen, 2002). Because one can

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assume that changes after training durations shorter than 6 weeks are primarily neural (Moritani & deVries, 1979), that finding therefore provides the possibility of excluding major muscular adaptations. Accordingly, H-reflexes were increased after only 4 weeks of hopping training. Yet, that effect was strongly dependent on the specific motor task (Voigt, Chelli, & Frigo, 1998). Measurements in the active state impose limitations with respect to stability of the paradigm and require precise experimental control of several effects on the spinal reflex arc (for reviews, see Mistaszek, 2003; Zehr, 2002). Last, making predictions for balance training (i.e., sensorimotor training [SMT]) on the basis of those studies would be improper.

Trimble and Koecha (1994) provided evidence for reflex down-training in man during SMT. In their experimental setup, however, they used H-reflexes to perturb balance and instructed participants to suppress the H-reflexes because that helped them to maintain balance. Because that study was based on an operant-conditioning paradigm, it is not clear that the reflex adaptations reported are comparable with those observed during common physical exercises.

Thus, it is still a matter of controversy whether reflex adaptation forms part of the neuroplasticity induced by training. It was our aim in the present study to test the hypothesis that SMT as well as BST induce changes in short latency reflexes, as was indicated by cross-sectional studies.

Method

Participants

Thirty healthy participants with no history of neurological disorders or injuries of the lower extremities took part in the study. We randomly assigned them to one of three groups: BST (4 men and 5 women, 25 ± 3 years old, height = 1.72 ± 0.08 m, and weight = 66 ± 12 kg); SMT (7 men and 4 women, 26 ± 5 years old, height = 1.74 ± 0.09 m, and weight = 65 ± 10 kg); or control (CON; 5 men and 4 women, 26 ± 3 years, height = 1.75 ± 0.08 m, and weight = 67 ± 8 kg). Participants were not involved in any other systematic training during the experiment and had not previously performed BST or SMT. All participants gave their written informed consent before taking part in the study. The study was approved by the ethics committee of the University of Freiburg and was conducted according to the Declaration of Helsinki.

Training

The SMT group as well as the BST group trained for 4 weeks, for a total of 16 training sessions (4 sessions/week). The authors documented, surveyed, and supervised all sessions of the study. Each training session lasted for 60 min, starting with a 10-min warm-up on a bicycle dynamometer at 100 W, and ended with a 10-min cool-down on a bicycle dynamometer at 100 W. The training consisted of a circle of postural stabilization tasks: wobbling board, spinning top, soft mat, and cushion. Participants performed the exercises on their right leg, barefoot, eyes open, and hands akimbo. Balance exercises were carried out in four sets. Each set was performed on a specific training device and consisted of four trials. The trials were performed for 20 s with a 40-s rest. We allowed a longer rest of 3 min between different sets to avoid fatigue. Participants’ objective in each stabilization task was to retain balance. After 8 sessions, we intensified the training by increasing the number of sets to six.

The BST group trained according to protocols that have been used in other studies (Duchateau & Hainaut, 1984; Van Cutsem, Duchateau, & Hainaut, 1998). In each training session, the participants executed four sets of 10 ankle dorsiflexions and 10 plantarflexions, respectively. The participants were positioned with the ankle joint at approximately 100°. They performed the contractions against a load of 30% to 40% of their one-repetition maximum. We instructed participants to contract as fast as possible with maximum voluntary effort. We instructed them to rest 2 s between two contractions and to then concentrate on the following contraction. We carefully controlled that they did so to ensure high motivation. We allowed participants to rest for 2–3 min between sets to avoid fatigue. After 8 sessions, we intensified the training by increasing it to six the number of sets.

SMT was characterized by a complex activation of the muscles encompassing the ankle joint, but also of the thigh and trunk muscles, which enabled participants to gain and regain balance. Those complex activation patterns included countercontractions that occurred frequently and enabled them to stabilize the ankle joint (Nielsen & Kagamihara, 1993). In contrast to SMT, BST is aimed at movement velocity, which requires maximal activation of agonists accompanied by minimal activation of antagonists during movement execution. Therefore, almost no counteractions were observed during BST. Moreover, participants performed SMT while standing on one leg, whereas they sat during BST. Therefore, one could assume that the amount of different input was much higher for SMT than it was for BST (Katz, Meunier, & Pierrot-Deseilligny, 1988). The control group maintained their normal physical activities throughout the experimental period. The participants were not allowed to reduce or raise their daily sport activities between pre- and posttests.

Electromyography

To determine electromyographic (EMG) activity, we firmly attached Ag–AgCl surface cup-electrodes (9 mm in diameter, center-to-center distance = 2.5 cm) filled with electrode jelly to the skin over the tibialis anterior (TIB), gastrocnemius lateralis (GAS), and soleus (SOL) muscles of the right leg. The longitudinal axes of the electrodes were in line with the presumed direction of the underlying muscle fibers. The same person carefully determined all electrode positions for pre- and posttest measurements to ensure identical recording sites. We kept interelectrode resistance below 5 kΩ by means of shaving, light abrasion, and degreasing the skin. The EMG was carefully monitored for artifacts, noise, and cross-talk. EMG signals were sampled...
at 4 kHz, amplified (gain 1000), and band-pass filtered (10-1000 Hz).

**H-reflex recordings.** We evoked the H-reflexes by using an 0.5-ms current pulse applied at the tibial nerve in the popliteal fossa with a constant current square wave stimulator (Digitimer Model D57; Digitimer Ltd, Hertfordshire, England). We placed a large, 10-cm × 5-cm graphite rubber anode below the patella and used a small, 2-cm diameter electrode as a cathode. We located a suitable position for stimulation by carefully moving the cathode in the popliteal fossa, whereas we monitored SOL and TIB EMGs on an oscilloscope. Last, we fixed the cathode when we obtained a stable H-reflex with minimal stimulus current and without EMG activity in the TIB muscle. We did preliminary electrode positioning while participants were standing. We ascertained the final position while participants were in the sitting position before the experiment was conducted. For calculating Hmax-to-Mmax ratios, we obtained the H-reflex recruitment curves with at least 40 stimuli. The current was progressively increased until we obtained maximum M-wave. We verified Mmax and carefully determined Hmax to get the Hmax:Mmax ratio. We elicited H-reflexes every 4 s to assure that we recovered all excited motoneurons (Sabbahi & Sedgwick, 1987).

**Stretch reflex recordings.** An ankle ergometer applied stretch reflexes (S-reflexes). Two motors controlled by a belt gear system drove the foot pedals. Participants were fixed by a snowboard binding system, with their feet resting on the rotation platform of the ergometer (100° in the ankle and 90° in the knee joint). The arbitrary rotation axis of the upper ankle joint coincided with the rotation axis of the torque platform (Figure 1A). An induced dorsiflexion movement at the ankle joint with an amplitude of 6° and a velocity of 200°/s evoked an S-reflex. In the present study, we summed at least 40 S-reflexes to calculate mean peak-to-peak amplitudes. We recorded both H- and S-reflexes while participants were sitting and their muscles were quiescent (Figure 1B and C). The procedure was described in detail by Gollhofer and Rapp (1993).

**MVC and RFDmax**

We measured maximum isometric ankle plantarflexion strength on an isokinetic system (Isomed 2000, D. & R. Ferstl GmbH, Hemau, Germany). The maximum error of the torque sensor was <0.2%. Participants were positioned on the seat of the isokinetic device, with hip and knee angles adjusted at 90° and the ankle angle at 100°. Straps on the isokinetic system fixed the waist, the thigh, and the shank, and we asked the participants to cross their arms in front of their chest. The exact position of each participant was documented and saved so that it was identical in pre- and posttests. Testing was performed only on the right leg. We allowed each participant a warm-up period of 10 min on a bicycle ergometer at 100 W, followed by three to five submaximal isometric actions in the isokinetic system, to get accustomed to the testing procedure. Thereafter, each participant performed five plantarflexions and five dorsiflexions. The first two test trials had to be performed with submaximal effort. We visually inspected the resulting torque time curves of those test trials and finally instructed participants to do the three contractions that were used for analyses. The last three trials of each set were performed with maximal voluntary effort. We thoroughly instructed participants to act “as forcefully and as fast as possible” for each of those trials.

A digital fourth-order recursive Butterworth low-pass filter with a cut-off frequency of 50 Hz filtered the torque signal. Onset of torque was determined when torque exceeded 2 Nm. Torque parameters were recorded for each of the three maximal isometric contractions and were afterward reported as mean values. We defined RFDmax as the maximal slope of the torque time curve (dT/dt). We determined time to reach RFDmax (tRFDmax) relative to the onset of torque (Figure 2A and B).

**Dynamic Postural Control**

We assessed participants’ dynamic postural control during a 40-s one-leg stance test on the Posturomed (Haider Bioswing, Pullenreuth, Germany). The Posturomed is a two-dimensional platform mounted on four springs that allows damped translatory movements in anteroposterior and mediolateral directions. The maximum range of motion in both directions is 70 mm. The mechanical constraints and the reliability of the system were described earlier (Mueller, Guenther, Krauss, & Horstmann, 2004). We measured total sway by using joystick potentiometers connected to the moveable platform. Participants stood on the right leg, with the knee bent to about 30°. Participants were instructed to stand as still as possible with hands akimbo and with their view directed to a nearby wall. Several trials helped participants to get accustomed to the measuring device. After that, three test trials were performed. Cumulative sway path was determined during a time interval of 40 s.

**Statistics**

Data are presented as group mean values ± standard error (SE). Because of the small number of participants, we calculated nonparametric tests. We assessed differences between the three groups in the pre- and posttests by using Kruskal–Wallis analysis. In case of significance (p < .05), we calculated single-group comparisons by means of Mann–Whitney rank tests for unpaired samples (two-tailed, p < .05). We analyzed the effect of the different training interventions with Friedman tests (p < .05) for paired samples. We used SPSS Version 13.0 software (SPSS, Inc., Chicago, IL) to execute all analyses.

**Results**

**MVC and RFDmax**

MVC remained unchanged from pretest to posttest (SMT, 54 ± 10 Nm vs. 58 ± 9 Nm, p = .128; BST, 58 ± 5
FIGURE 1. (A) Stretch reflexes (SR) were recorded while participants sat with both feet attached to motor-driven footplates. In pre- and posttests, participants were instructed to cross the arms over the chest, look straight ahead and relax. Soleus (SOL), gastrocnemius, and tibialis muscles were quiescent during the measurement. (B) Grand mean curves (thin lines, ± standard deviation) of 40 pedal movements for 1 exemplary participant are displayed. Onset of stretch was determined when the pedal position signal exceeded a value of 90.1°. (C) The pedal movements induced fast dorsiflexions at the ankle that induced SRs. Latency of the SR was determined from the onset of pedal movement to the onset of SOL electromyographic (EMG) activity, which was assigned visually. The peak-to-peak reflex amplitude was calculated from grand mean curves as the difference between the highest and lowest EMG values.
were reduced after SMT ($-28\% \pm 9\%$, $p = .041$; Figure 4B); the reduction was accompanied by reduced root mean square (RMS) values ($-24\% \pm 9\%$, $p = .045$; Figure 4C), whereas no changes occurred after BST ($p = .139$ and .767, respectively) and in the CON group ($p = .515$ and .214, respectively). There was a difference between the groups when we compared S-reflex amplitude changes ($p = .004$) and RMS changes ($p = .020$).

**Electrically Induced H-reflexes**

Following SMT, Hmax:Mmax ratios were reduced ($-19\% \pm 7\%$, $p = .021$; Figure 5), whereas no significant changes were observed following BST ($p = .374$) and in the CON group ($p = .594$). The observed reductions in Hmax:Mmax ratios in the SMT group were exclusively related to reduced maximal H-reflexes ($-24\% \pm 6\%$, $p = .021$). No changes in maximal M-waves ($5.8 \pm 0.5$ mV vs. $5.4 \pm 0.4$ mV, $p = .213$) were found for SMT. No significant difference between groups was observed when we compared Hmax:Mmax ratio changes ($p = .063$). Differences in Hmax:Mmax ratios were found between SMT and BST and between SMT and CON for the postraining measurement (Figure 5).

**Discussion**

After 4 weeks of either SMT or BST, increased RFD and improved dynamic balance control could be observed. After SMT, both peak-to-peak amplitudes of S-reflexes as well as Hmax:Mmax ratios were decreased, whereas both mechanically as well as electrically induced reflexes remained unchanged after BST. The results suggest clearly different adaptations for SMT than for BST. We next discuss the results with respect to the underlying neural mechanisms.

**Possible Mechanisms That Could Account for Reduced Reflex Sensitivity Following SMT**

In SMT, the participants were practicing various balance control exercises in the one-leg stance. One strategy to improve balance is to coactivate the respective muscles encompassing the joint complexes of the lower extremity. According to the literature, the coactivation is accompanied by acute reductions in H-reflex excitability (Llewellyn, Yang, & Prochazka, 1990; Trimble & Koecke, 2001). Most interesting, in both of the cited studies, the depression of H-reflex excitability was not accompanied by any reduction in SOL background EMG. Therefore, the authors suggested that reciprocal inhibition could not be responsible for the reduction of reflex amplitude. As a matter of fact, Nielsen and Kagaishi (1992) were able to show that during the performance of unstable postural tasks, reciprocal inhibition was depressed significantly. However, the absence of reciprocal inhibition in combination with a possibly high fusimotor drive during balancing tasks would lead to facilitated S-reflex gains. Therefore, the motor system may have to use reflex control via descending pathways to prevent an impending deterioration of motor performance because of
exaggerated reflex activity. That hypothesis is supported by findings from Nielsen and Kagamihara (1993), who showed that presynaptic inhibition (PSI) of Ia afferents is actually increased during cocontractions of SOL and TIB muscles while individuals perform postural tasks. In their experiments, the authors reduced PSI by stimulating the femoral nerve. That stimulation could induce monosynaptic facilitation of SOL H-reflexes either before plantarflexions or before cocontractions at the ankle joint. Net facilitation increased 50 ms before plantarflexions, whereas it decreased before cocontractions. The authors concluded that depression of reciprocal inhibition accompanied by increased PSI of Ia afferents emanates from a central command; the command is initiated when cocontractions are required to stabilize a joint during a balance task. Furthermore, evidence has been produced that with increasing postural demands, PSI increases as well (Katz et al., 1988). Consequently it has been argued that PSI allows a differential modulating effect of afferent input depending on the functional role of a respective antigravity target muscle.
results of those studies support the concept that descending modulation of PSI may be a common mechanism for compensating for enhanced afferent input during postural tasks, possibly to avoid exaggerated feedback gains by enhanced S-reflexes.

Whereas an immediate increase in descending control of PSI can explain the reduced reflex excitability that was observed during unstable stance (Chalmers & Knutzen, 2002; Koceja, Markus, & Trimble, 1995; Koceja, Trimble, & Earles, 1993; Nielsen & Kagamihara, 1993; Trimble & Koceja, 1994, 2001) and during unstable walking (Llewellyn et al., 1990), the question remains: Why did a 4-week SMT produce reduced S- and H-reflex amplitudes as demonstrated in the present study? Such an observation would imply long-term effects on reflex gain similar to the findings of Trimble and Koceja (1994, 2001). They provided evidence for a “down-training” of the SOL H-reflex in humans after several sessions of H-reflex-induced perturbations.

All of those results are in line with the observations of Wolpaw and O’Keefe (1984), who demonstrated that monkeys were able to modulate the amplitudes of S-reflexes according to the requirements of a specific task. In their experiment, brief extensions of the elbow elicited S-reflexes in the biceps brachii muscle. Wolpaw and O’Keefe immediately calculated the absolute value of biceps EMG in the S-reflex interval, and gave a liquid reward only if the monkey’s S-reflex value was in a specific range. Two different phases of adaptation could be distinguished. An almost immediate tonic supraspinal influence on the Ia afferent system occurred within the first 6 hr of training, which could have been caused by operant conditioning. After the first day, a slower change occurred during the following 2 months; the change was assumed to reflect long-term plasticity. Wolf and Segal (1996) were able to show similar plasticity in human biceps brachii training participants received feedback about their S-reflex responses and were rewarded for reflex peak-to-peak amplitudes below the baseline value. After 24 training sessions, each consisting of 250 stretches of the biceps brachii muscle, peak-to-peak amplitudes of S-reflexes were reduced by 24%. It is an important fact that the onset time of stretch is unpredictable and that the spinal S-reflex occurs before any other possible response. That finding implies that a change in descending activity that modulates reflex response strength must be
continually present. The theory of the descending control of PSI is reinforced by the results of some experiments that clearly showed that the modification of reflex gain depended on the existence of the pyramidal tract (X. Y. Chen & Wolpaw, 2002; Wolpaw & Lee, 1989). In line with our observations, those adaptive mechanisms are most likely to also occur following SMT. In conclusion, we suggest that an increase in PSI on Ia fibers caused by supraspinal input could account for reduced reflex excitability following SMT in SOL motoneurons in the present study.
Why Did SMT But Not BST Reduce Reflexes?

Despite the fact that BST did not cause any reflex adaptations, the training nevertheless increased RFD and the level of balance control. That change could be the consequence of more efficient recruitment patterns and also of an increase in motoneuron discharge rate (Van Cutsem et al., 1998). Furthermore, it is known that recruitment thresholds and firing rates of motoneurons depend on the amount of afferent input (Grande & Cafarelli, 2003), which was quite different between the two training regimens. In addition to the differences in BST and SMT concerning the amount of afferent input, the diverse complexity of the tasks may have been responsible for differences in recruitment patterns and, therefore, in reflex adaptation. Perez, Lungholt, Nyborg, and Nielsen (2004) could not show any recruitment gain in motor evoked potentials when they applied transcranial magnetic stimulation on TIB anterior following unskilled voluntary ankle dorsiflexions and plantarflexions. They demonstrated that during skill training, however, the same type of ankle movement produced a significant increase in recruitment. Therefore, they suggested that recruitment changes of the leg motor areas were related to the level of difficulty of the motor task. As a consequence of body stabilization during training, SMT was a more complex task than was BST. On the basis of the aforementioned reasons, it can be argued that recruitment was different in both tasks and that that difference may explain the absence of reflex reduction following BST.

Furthermore, balance tasks frequently occur in everyday life, whereas BST demands a very specific muscle activation pattern. Therefore, we suggest that the adaptational processes caused by BST can be seen only in the trained task (Aagaard et al., 2002; Voigt et al., 1998), whereas SMT causes a more general adaptation that one can also determine during rest.

Comparison of Our Results and Reflex Adaptations Reported in Cross-Sectional Studies

Reductions in reflex excitability compared with those of controls were reported in cross-sectional investigations of track and field athletes; soccer, tennis, and volleyball players; as well as dancers (Casabona et al., 1990; Goode & Van Hoven, 1982; Koceja et al., 1991; Kyrolainen & Komi, 1994; Mattioli et al., 2001; Nielsen et al., 1993; Rochonggar et al., 1979). Because of their design, however, those studies could not show that the differences were caused by a specific training regimen. In cross-sectional studies, it is not possible to exclude the influence of genetic factors. Longitudinal training studies are rare, and their results are inconsistent. In most of those studies, training regimens with emphasis on strength have been used. Hakkainen and Komi (1986) reported a reduction in tendon tap (T) reflexes after 24 weeks of jump training, whereas Voigt et al. (1998) showed enhanced H-reflexes following 4 weeks of hopping training. Hakkainen and Komi (1983, 1986) found that T-reflexes were reduced following strength training, whereas increased H-reflexes were reported after 12 weeks of strength training (Aagaard et al., 2002). Thus, reflex adaptations were tested during rest in some studies (Hakkainen & Komi, 1983, 1986), whereas tests were applied during the active condition in others (Aagaard et al.; Voigt et al.). The heterogeneity of those results makes it impossible to draw a general conclusion concerning the underlying mechanisms and their interactions. The present results indicate that balance training (SMT) is able to reduce reflex excitability. That effect was previously assumed from the results of the cross-sectional studies in dancers (Goode & Van Hoven, 1982; Koceja et al.; Nielsen et al.). Peak-to-peak amplitudes and RMS values of S-reflexes, as well as Hmax:Mmax ratios, were significantly reduced after SMT; as a result, group differences between the training groups were observed (Figures 4 and 5). In the present investigation, those neural adaptations could not be shown for BST. We therefore suggest that reduced reflex excitability following prolonged power training interventions reported in cross-sectional studies (Casabona et al.; Kyrolainen & Komi; Mattioli et al.; Rochonggar et al.) is likely associated with muscular rather than neural adaptations. Genetic predisposition or a shift in muscle fiber distribution could explain reduced T- and H-reflexes because it is well known that high-threshold motor units are less easily excited by Ia afferents than are small and low-threshold motor units (for a review, see Burke, 1981).

Functional Implications

Down-training of SOL S-reflexes after SMT may be relevant for the rehabilitation of spasticity—for example, in spinal cord injured participants, who show high S-reflex activity even during the stance phase and only minor depression during the swing phase (Yang et al., 1991). Therefore, exercises that help to normalize S-reflex behavior could improve their walking pattern. Y. Chen et al. (2005) provided some experimental data that support that suggestion. They examined the interaction of H-reflex conditioning and locomotion in rats. Down-conditioning resulted in smaller H-reflexes even during the stance and swing phases of locomotion. Because the walking pattern remained unaffected, Y. Chen and colleagues concluded that additional compensatory plasticity ensured normal locomotion. In pathological situations, when locomotion is already impaired, therapists might use exercises that modify spinal reflexes to restore effective locomotion and improve functional capacity. It was demonstrated previously that spinal reflex conditioning of biceps brachii muscle is possible in patients with partial spinal cord injuries (Segal & Wolf, 1994). One may speculate whether an SMT would show similar effects for triceps surae muscles. Furthermore, down-regulation of spinal reflexes accompanied by enhanced balance control and increased RFD of the stabilizing muscles may be of importance with regard to the prevention of injuries or falls, especially in the elderly (Mynark & Koceja, 2002).
Conclusion
The authors demonstrated that physical exercise can reduce S- and H-reflexes. They suggest that the reduction in reflex excitability following SMT results from an overall increase in PSI on Ia afferent fibers. It is assumed that input from supraspinal centers modulates the PSI to depress S-reflexes caused by balance tasks. Results of investigations concerning operant conditioning in monkeys and down-training of reflexes during motor learning have confirmed that assumption. On the basis of the observation that reflex gain is altered exclusively after SMT but not after BST, the authors conclude that the underlying neural adaptations differ between the training regimens. According to the present results, neural adaptations could be responsible for the investigated reduction in reflexes that have been observed in balance-trained athletes. On the other hand, in athletes who perform explosive muscle contractions, a reduction in reflexes is most likely the consequence of genetic predispositions or is associated with a shift in muscle fiber distribution after a sustained BST over several months.

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