REVIEW ARTICLE

Sports Med 2001; 31 (6): 409-425
0112-1642/01/0006-0409/$22.00/0

Neural Influences on Sprint Running

Training Adaptations and Acute Responses

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Abstract

Performance in sprint exercise is determined by the ability to accelerate, the magnitude of maximal velocity and the ability to maintain velocity against the onset of fatigue. These factors are strongly influenced by metabolic and anthropometric components. Improved temporal sequencing of muscle activation and/or improved fast twitch fibre recruitment may contribute to superior sprint performance. Speed of impulse transmission along the motor axon may also have implications on sprint performance. Nerve conduction velocity (NCV) has been shown to increase in response to a period of sprint training. However, it is difficult to determine if increased NCV is likely to contribute to improved sprint performance.

An increase in motoneuron excitability, as measured by the Hoffman reflex (H-reflex), has been reported to produce a more powerful muscular contraction, hence maximising motoneuron excitability would be expected to benefit sprint performance. Motoneuron excitability can be raised acutely by an appropriate stimulus with obvious implications for sprint performance. However, at rest H-reflex has been reported to be lower in athletes trained for explosive events compared with endurance-trained athletes. This may be caused by the relatively high, fast twitch fibre percentage and the consequent high activation thresholds of such motor units in power-trained populations. In contrast, stretch reflexes appear to be enhanced in sprint athletes possibly because of increased muscle spindle sen-

sitivity as a result of sprint training. With muscle in a contracted state, however, there is evidence to suggest greater reflex potentiation among both sprint and resistance-trained populations compared with controls. Again this may be indicative of the predominant types of motor units in these populations, but may also mean an enhanced reflex contribution to force production during running in sprint-trained athletes.

Fatigue of neural origin both during and following sprint exercise has implications with respect to optimising training frequency and volume. Research suggests athletes are unable to maintain maximal firing frequencies for the full duration of, for example, a 100m sprint. Fatigue after a single training session may also have a neural manifestation with some athletes unable to voluntarily fully activate muscle or experiencing stretch reflex inhibition after heavy training. This may occur in conjunction with muscle damage.

Research investigating the neural influences on sprint performance is limited. Further longitudinal research is necessary to improve our understanding of neural factors that contribute to training-induced improvements in sprint performance.

Sprint exercise for the purposes of this review, is defined as rapid, unpaced cyclic running of 15 seconds or less in duration at maximum intensity throughout. Single bouts of activity are sufficiently separated in time to allow full recovery between repetitions. Examples of such activity in the sporting arena include the 60 and 100m sprints in track and field and the push-start in bobsleigh.

As shown in figure 1 sprint running performance is the product of stride rate (SR) and stride length (SL) with numerous components influencing this apparently simple formula. Performance in sprint exercise has traditionally been thought to be largely dependent on genetic factors, with only relatively small improvements occurring with training.[1] Muscle fibre type has been purported to be one of the principal factors underlying sprint performance,[2] with enzymatic adaptations and hypertrophy of prime movers thought to be largely responsible for post-training improvements in performance. However, recent evidence suggests that enzymatic adaptations or changes in the muscle contractile proteins are not always associated with significant improvements in sprint performance.[3,4] Clearly other mechanisms of adaptation are required and this likely includes neural improvements. Evidence from resistance training literature suggests that significant neural adaptation can occur after training involving repeated bouts of brief, intense exercise.[5-7] Given the highly complex nature of sprint running it may well be that neural adaptations occur over a much longer period of time than has been observed in resistance training literature. As illustrated in figure 1 most of the factors affecting both SL and SR may be influenced by the nervous system. The ability of the nervous system to fully or appropriately activate skeletal muscle therefore bears closer examination.

Maximal intensity sprint exercise necessitates extremely high levels of neural activation.[8-11] Direct evidence as to whether the level of activation is altered through sprint training is uncertain as no training studies have as yet been conducted. Measurable neurological parameters such as nerve conduction velocity (NCV), maximum electromyogram (EMG), motor unit recruitment strategy and Hoffman reflex (H-reflex), however, have been shown to alter in response to physical training programmes.[6,12-17] Cross-sectional differences in these variables are also evident between sprint, untrained and endurance-trained groups.[16-19] While it is likely that neural adaptations to sprint exercise occur, whether they have a causal influence on the improvements in sprint performance is at present unclear.

Potential mechanisms for neurally influenced improvements in sprint performance include: changes in temporal sequencing of muscle activation for

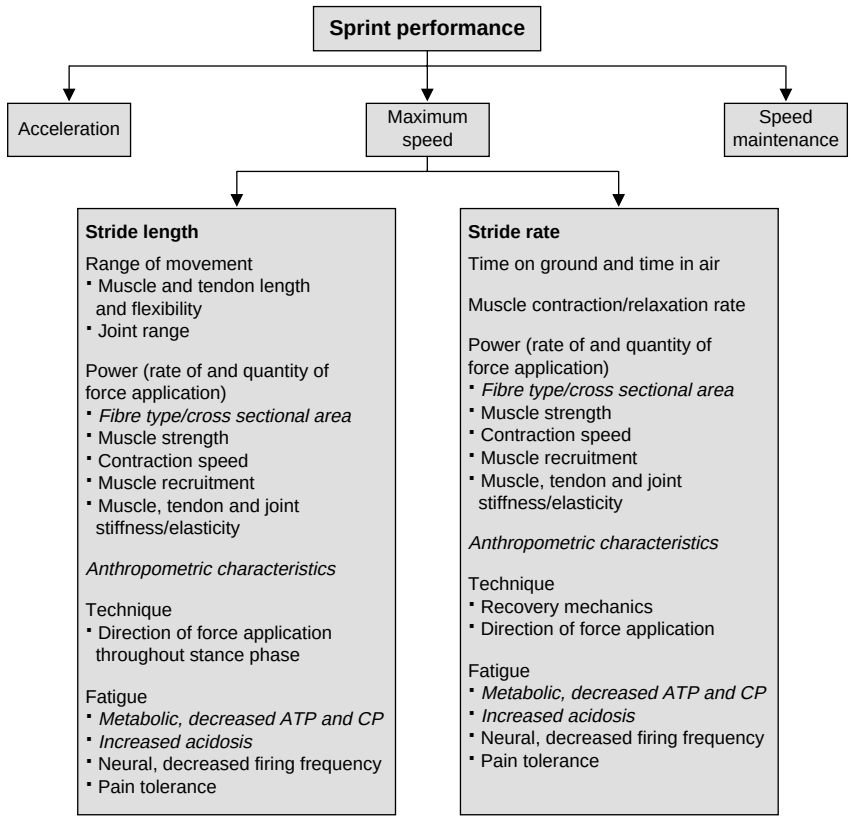

Fig. 1. Components of sprint performance. Components in italics are not neurally influenced. **ATP** = adenosine triphosphate; **CP** = creatine phosphate.

more efficient movement, altered motor unit recruitment strategies (i.e. preferential recruitment of the fastest motor units), increased NCV, frequency or degree of muscle innervation and increased ability to maintain muscle recruitment and rapid firing for the duration of the sprint activity. The purpose of this review is to discuss these mechanisms and their possible influences on performance. Where possible practical implications of the research will be discussed.

1. Muscle Activation and Recruitment Strategy

In a complex movement task such as sprinting, a multitude of different muscles must be activated at the appropriate times and intensities to maximise speed. Optimising the timing of agonist and antagonist muscle activation may allow for decreased co-contraction at an appropriate point of contraction and hence improved movement speed. Measurement of gross changes may be visible in technique modification seen via biomechanical analysis. Alternatively, more direct measurement using electromyography enables a detailed assessment of changes as a result of training.

1.1 Technique Differences

Some differences in running technique have been observed in elite and non-elite sprint performers with particular reference to joint angles (nota-

bly hip and knee flexion) and movement velocity of limb segments in hip extension.[20-22] Some of the observed differences may be the result of refinement of the neural innervation patterns or motor programmes as a result of the extensive training histories of the elite performers. Mero and Komi[23] have provided further evidence of a more developed and/or efficient motor programme in elite athletes. While no differences were found at unassisted speeds (aside from the trained being faster than untrained), the authors found that when relatively untrained individuals were towed to supra-maximal running velocities they were unable to increase SR above their normal maximum and responded to the increased speed with inefficient increases in SL. In contrast, well-trained athletes responded to such stimuli by increasing both SR and SL,[23] perhaps indicating superior neural adaptations to high intensity sprint exercise. These studies, however, were cross-sectional in nature and, as such, the relative importance of genetic and training influences remains uncertain. This question can be addressed only by a detailed longitudinal analysis of the adaptations to running technique with training.

1.2 Electromyographic Studies

The difficulties in ensuring reliable electrode placement between sessions make longitudinal studies of the changes in EMG technically challenging. As a result there are few such studies in the current literature. However, electromyographic studies investigating the temporal sequencing of muscle activation or relative activity of various muscles have reported significant changes when speed is increased to maximal or supra-maximal velocities.[24,25] Similarly, in controlled laboratory conditions, changes in the speed of muscle contraction have been reported to alter the ratio of contribution of co-agonist muscles.[26] Furthermore, relative temporal changes in muscle activation as a result of practice have been observed even in simple single arm movements.[12,27] Schneider and associates[27] reported increased use of the stretch shortening cycle (SSC) in late practice. Such an adaptation would have obvious advantages for sprinting efficiency as evidence suggests that the SSC contributes to propulsive force during running.[8] These results suggest that there is considerable scope for temporal changes in muscle activation to occur with sprint training and that such changes could enhance performance.

1.3 Fibre Type Recruitment

The ability to fully or selectively recruit fast twitch motor units has been examined under various conditions[19,28-30] and may have implications for sprint performance. A number of studies have shown evidence of preferential fast twitch fibre recruitment, particularly in eccentric exercise.[28,29] However, the results from Desmedt and Godaux[30] suggested that in rapid concentric contractions there is no evidence of selective or preferential recruitment of fast twitch units. In contrast, one cross-sectional study has demonstrated that sprint athletes may have a significantly greater ability to selectively recruit fast twitch motor units compared with endurance or untrained individuals.[19] However, Saplinskas and associates[19] limited their investigation to only one muscle (tibialis anterior) and reported little detail of the type of contraction used. Hence, it is difficult to fully assess the validity of their findings. Furthermore, the possibility of a greater proportion of fast twitch motor units in the sprint athlete's tibialis anterior may bias the result. Nevertheless, the possibility of recruiting selectively those motor units that can rapidly contract and relax remains as a potential adaptation to the demands of sprint exercise.

In summary, differences in running technique and in muscle activation patterns have been reported among trained sprint athletes compared with controls or endurance-trained individuals. This evidence provides some support for neurally influenced changes in sprint performance, though definitive data in this area are currently unavailable. Longitudinal training studies are required to determine the influence of training on these parameters.

2. Speed and Degree of Muscle Activation

The degree of muscle activation [as measured by integrated EMG (IEMG)] is known to increase with increasing running speed.[24,25,31,32] Indeed, in well-trained athletes, muscle activation and SR may continue to increase at supra-maximal speeds.[33] However, during some maximal voluntary exercise not all individuals are able to fully activate their muscles.[34,35] Potentially, task-specific training may allow greater activation in a given activity. Indeed, some resistance training research would appear to validate this theory[5-7] with an increase in the IEMG accompanying a marked increase in strength, particularly during the initial stages of training. However, as with the examination of temporal changes in muscle activity, the complexity and explosive nature of sprint exercise means there are difficulties with accurately recording IEMG from the active musculature. Slight changes in electrode position or skin preparation from one session to another make it impossible to compare directly the amplitude of muscle activation using IEMG. To compare amplitude across sessions it must be normalised, typically by the maximum evoked potential (M-wave) obtained within each session, as has been used elsewhere.[36]

These techniques have yet to be used with sprint exercise as the large number of muscles used in sprinting make it difficult to perform studies of this nature. Despite the paucity of research directly examining changes in muscle activation, both cross-sectional and longitudinal studies[15,17,37-41] investigating sprint exercise have examined changes in other neural measures such as NCV and reflex measures which may be indicative of adaptations that allow increased neural stimulus to muscle.

2.1 Nerve Conduction Velocity

NCV is a measure of the speed an impulse can be transmitted along a motoneuron and is strongly related to muscle contraction time.[42,43] A rapid NCV is also indicative of a short refractory period.[44,45] In turn, the decreased refractory period may allow for greater impulse frequency, thereby increasing muscle activation levels.

While, NCV has been examined in a number of cross-sectional studies of different athletic populations,[17,37-41] no clear trend in its relation to performance has emerged. Some studies suggest strength and power athletes have faster NCV than endurance athletes,[17,39] while others report sprinters and jumpers have slower NCV than other groups.[17,41] Other researchers have shown that trained individuals have faster NCV than untrained individuals.[37,38] It has also been reported that no differences were evident between power and endurance groups.[40] Clearly the literature to date has left this point unresolved. Often studies use slightly different methods, and failure to correct for temperature, diurnal variation or age may account for some of the variation. Furthermore, the double stimulation technique used in these studies relies on supra-maximal stimulation at 2 sites along a nerve. The difference in transmission time to the recording site allows calculation of conduction velocity (CV). This technique, however, determines the CV of only the fastest conducting fibres.[46] An alternative method, termed the 'collision technique', determines a range of CVs from the fastest to slowest fibres for a motor nerve, by stimulating submaximally at the distal site and maximally at the proximal site.[46] Determining the potential for changes in the slower conducting fibres toward the speeds of the faster fibres may be more informative than examining only the fastest conducting axons of the motor nerve, which may already be at a maximal level. Examination of the range of NCVs may also yield a more consistent pattern in the results. To date, the collision technique has not been used for NCV assessment in a sprint-related study.

The only longitudinal study to examine changes in NCV has reported that NCV increases in response to 14 weeks of repeated 10-second cycle sprints training at 48-hour intervals.[15] No change in maximal IEMG was observed; however, IEMG was measured only in an isometric contraction. Given the specificity of performance and neural adaptations to training[47] this particular test may be insensitive

to changes in the degree of muscle activation during the sprint exercise. Thus, it is not possible to determine the relationship between NCV changes and changes in muscle activation in response to sprint training from the results from Sleivert and associates.[15]

Frequency and volume of training may also affect NCV. Research suggests that muscle adaptation and more specifically myosin heavy chain shift may vary dependent on the frequency of sprint training.[48] Similarly, neural adaptation may be related to training frequency. Some animal studies using a high daily volume of high intensity (but not sprint) exercise have reported a decrease in both axon diameter and myelination.[49,50] However, other studies have reported an increase in axon diameter in response to exercise stress.[51,52] It appears there may be a similar frequency threshold as observed in muscle adaptations beyond which exercise stress may negatively affect axon diameter, myelination and, therefore, NCV. Given the lack of data relating to sprint training and NCV, it is not currently possible to speculate what such a frequency might be, other than to suggest it would be greater than the 48-hourly protocol of Sleivert and associates.[15]

In summary, NCV would appear to differ between both individuals and different athletic populations although a lack of consistent methodology has made the results difficult to interpret. Further research using the collision technique to assess the range of NCVs across different fibres within an individual may give greater information with respect to adaptations with training. Despite the numerous studies examining NCV, its implications for improving performance would appear to be negligible as interindividual differences appear to have limited functional significance. Furthermore, recent research suggests that the relationship between NCV and muscle CV is limited,[53] perhaps a further indicator of the lack of functional relevance of NCV. However, in theory, changes in NCV may be indicative of adaptations in the nerve structure such as increased axon diameter and myelination. In turn, these adaptations may decrease the refractory period of the nerve, which would allow increased impulse frequency and potentially increased muscle activation.

2.2 Motoneuron Excitability and Reflex Adaptation

Motoneuron excitability for the purposes of this review describes how readily the motoneuron pool is activated with respect to a given input. An increase in motoneuron excitability leads to a more powerful muscle contraction.[14,54] Therefore, for sprint athletes an increase in motoneuron excitability would be advantageous with regard to performance.

Motoneuron excitability is commonly assessed using the H-reflex. The H-reflex is often regarded as a monosynaptic reflex response analogous to the tendon reflex though it is elicited by electrical stimulation of the peripheral nerve. In addition to stimulating motoneuron axons, electrical stimulation of the peripheral nerve also activates Ia afferents from muscle spindles. The Ia afferents synapse on to the motoneurons at the spinal cord level to bring about, after a brief delay, a second EMG response, which is known as the H-reflex. Examination of the relative size of the H-reflex may provide information with respect to motoneuron excitability. Interpretation of the H-reflex is complicated because the gain of the reflex can be modulated via changes in muscle spindle sensitivity through the fusimotor system or via presynaptic inhibition of the Ia afferents.[55]

Since sprint running is the basis of this review, discussion will focus on the stretch/tendon and/or H-reflexes of the leg, and, in particular, in the triceps surae muscle group where most research has been focused. While the 2 reflexes (tendon and Hoffman) do differ, their responses to interventions are generally reported to be similar though not identical.[56] The major difference is that the H-reflex is less sensitive to changes in γ-activity[57] because the muscle spindle is bypassed during direct nerve stimulation. The H-reflex has a further methodological advantage in that it is easier to test compared with a tendon reflex, particularly during high intensity ballistic activity.

Despite the substantial body of literature examining both the H-reflex and the stretch reflex, the function of these reflexes remains somewhat unclear. With respect to the physiologically significant stretch reflex, its proposed functions during gait include compensation for ground irregularities,[55] force production at the end of the stance phase[8,58] and control of muscle stiffness rather than length.[59] Its role in compensation for perturbations during stance appear limited.

2.2.1 Stretch and Hoffman Reflex in Relaxed Muscle: Effect of Sprint Exercise

In contrast, to what may be anticipated with regard to Motoneuron excitability, that is, more excitability equals better performance, cross-sectional studies using athletes trained for explosive or anaerobic events (sprinters and volleyball players) have reported decreased resting H-reflexes in both soleus and gastrocnemius muscles relative to endurance or aerobically trained athletes.[16,60,61] The authors cited either a genetic- or training-induced decreased synaptic strength of Ia excitatory intermediate motoneurons in both soleus and gastrocnemius motoneuron pools of trained individuals as a possible explanation. An alternative suggestion based on previous research is that the slower fibres within muscle contribute more to the H-reflex response.[28,62] On this basis, it was suggested that the decreased H-reflex in the explosively trained athletes might be related to a slow to fast transformation of motor units.[16,63] Indeed, Almeida-Silveira and associates[63] found both decreased slow twitch fibre percentage and decreased H-reflex amplitude as a result of a plyometric training intervention. However, contraction time is found to decrease with increasing size and force of the H-reflex[62] which would suggest that units other than slow may also be recruited. Nevertheless, an abundance of high threshold motor units may require a certain level of background EMG, or potentiation, for the reflex response to induce contraction.

The decreased resting and contraction potentiated reflexes of sprint athletes may substantiate such claims.[16,41,60] Furthermore, animal studies also suggest that a decrease in H-reflex following a conditioning period may be a result of an increase in firing threshold.[64] The use of invasive implanted stimulation and recording electrodes in the Carp and Wolpaw study[64] and resulting high quality data, adds further merit to their results.

H-reflex disappears or is unable to be recorded at high stimulation intensities because of collisions between antidromic and orthodromic reflex volleys in the Ia afferent. In addition, the antidromic firing of the motor fibres renders the motoneurons refractory to reflex input.[65] This may limit its application in the assessment of resting H-reflex of fast twitch units. A further possible explanation for decreased H-reflex in anaerobically trained muscle may relate to changes in the descending influence as a result of long term training. Elite ballet dancers have negligible reflex activity in the triceps surae muscle group.[66,67] Nielsen and Kagamihara[68] suggested that the increased chronic co-contraction of muscles in the lower limb during ballet training (and subsequent presynaptic inhibition) may lead to an enduring decrease in synaptic transmission. The 'toe-up' or dorsi flexed ankle emphasis in current sprint training[69] results in similar amounts of co-contraction of tibialis anterior with the triceps surae muscle group. This provides a possible explanation as to the decreased resting H-reflex observed in sprint athletes which is an alternative to the slow to fast transformation of motor units proposed by Casabona et al.[16]

Importantly, resting tendon tap reflexes also appear to differ between sprint and endurance groups. Koceja and Kamen[70] reported a greater reflex in elite sprint athletes than in elite endurance athletes. Similarly, Kamen and associates[71] also reported that weight lifters have a shorter reflex latency for the patella tendon tap reflex. However, there was no difference between Achilles tendon tap reflex between the power and endurance groups. In contrast, and perhaps more closely related to the prevailing H-reflex literature, Kyröläinen and Komi[72] reported diminished tendon tap reflexes in 3 out of the 4 muscles tested in power athletes versus endurance athletes. The differences in these results are somewhat difficult to explain, although details

of what the power athletes were in the Kyröläinen and Komi[72] study were limited and no reference was made to their training age or competitive status. Nevertheless, the pattern of the above results is at the very least less clear cut than in the case of the H-reflex, suggesting that a positive adaptation in the gain of the muscle spindle may occur. That is, the muscle spindle is likely more sensitive to stretch as a result of the prolonged sprint and/or power training inducing a greater reflex response.

As yet, no sprint training studies have been conducted to examine modification of the stretch reflex; however, it has been shown that it is possible to enhance the stretch reflex response using a conditioning routine consisting of multiple rapid short stretches and biofeedback on the stretch reflex response.[73,74] While this research is still somewhat removed from sprint work, clearly there is potential for the stretch reflex response to be enhanced as a result of ballistic type activity such as sprinting.

As discussed in this section, most the work using sprint athletes and reflex investigation has been cross-sectional in design, hence genetic and training-induced differences are difficult to discriminate. However, the small amount of longitudinal work in conjunction with the prevailing cross-sectional work, suggests that the H-reflex decreases in response to sprint training – reflecting a decrease in motoneuron activation either caused by changes in presynaptic inhibition or potentially linked to a slow to fast motor unit transition. Stretch reflexes, however, are either unaffected or may increase post-sprint exercise, possibly indicating an increase in muscle spindle sensitivity to compensate for the decreased motoneuron excitability.

2.2.2 Reflex Potentiation by Contraction

A common result of an isometric muscle contraction is potentiation of both H- and tendon reflexes.[75-79] This may be caused by either central or peripheral mechanisms acting on the reflex pathway. Centrally, possible mechanisms for reflex potentiation include an increase in excitability of the motoneuron pool and decreased presynaptic inhibition of Ia terminals.[75,77] Peripherally, changes in the threshold of spindles after or during contraction may also affect stretch reflexes particularly.[79]

Reflex potentiation during a steady contraction[41] has been used to directly compare sprint athletes to other populations. The results suggested that reflexes were potentiated to a greater extent in elite sprint athletes than in control participants. While at rest, the higher percentage of fast twitch or high threshold motor units in sprint athletes require greater stimulation from the Ia afferents for a reflex to be elicited. Hence, during a contraction, the fast twitch units may be closer to threshold and thus the reflex stimulus may be sufficient to elicit a response. Whether enhanced reflex potentiation has performance advantages or applications beyond indicating fibre type is debatable. However, it is possible that potentiated reflex contribution, as a result of muscle activity at and immediately before footstrike, may aid force production in sprinting.[32] Furthermore, sprint-trained athletes may potentiate the stretch reflex to a greater extent than an untrained population, giving sprinters a further performance advantage.

Immediately following the contraction there is a brief depression of the H-reflex, followed by potentiation after approximately 4 minutes.[54,80] Güllich and Schmidtblicher[54] demonstrated a greater increase in reflex response after maximal contractions in sprint and power athletes compared with controls. Notably, the increase in H-reflex followed the same time course as did enhancements in explosive voluntary force production, possibly indicating potentiated stretch reflex contribution to power output. This may be caused by an increase in post-synaptic discharge caused by an increased effectiveness of the afferent volley following a high frequency burst of impulses, in turn leading to an increased liberation of transmitter from the Ia terminals.[80] Alternatively, the post-maximal activation potentiation (PAP) may be caused by phosphorylation of myosin light chains (MLC) during maximum voluntary contraction (MVC) which renders actin-myosin more sensitive to Ca^{2+} in a subsequent twitch.[81] This PAP appears to be greatest in type II fibres and is thought to be related to their greater capacity for MLC phosphorylation in re-

sponse to high frequency activation.[81] These latter possibilities may also provide a mechanistically based explanation for the greater potentiation seen in elite sprint athletes, given the known high proportions of fast twitch muscle in such populations.[82]

The mode of contraction also strongly affects the reflex response.[83-85] The studies above generally show that H-reflex is potentiated during an isometric contraction. Similar findings have been reported with a concentric contraction.[83] Conversely, eccentric muscle actions have been reported in a number of studies to show smaller H- or stretch reflex responses compared with isometric or concentric contractions.[83-85] These results appear to be independent of the level of background EMG although the control mechanism remains somewhat unclear.[85] This has implications for sprinting which will be further discussed in section 2.2.3.

To summarise, the H-reflex is potentiated during isometric and concentric muscle action and is potentiated to a greater extent in sprint athletes. Similarly, the stretch reflex is also potentiated by isometric muscle action; the H-reflex is potentiated more in sprint athletes after contraction, and explosive voluntary muscle action follows a similar time course. The H-reflex is depressed during eccentric muscle action in the soleus muscle. These findings may have implications for both talent identification as well as pre-competition warm-up leading to performance enhancement for sprint athletes.

2.2.3 Reflex Influence on Gait: Implications for Sprinting

An extensive review of reflex function during the phases of gait is beyond the scope of this review (see Zehr and Stein,[86] for a more comprehensive review in this area). Nevertheless, reflexes and, in particular, short latency stretch reflexes have a number of important influences that are pertinent for sprint running performance. These are addressed briefly below.

(i) *Force production*. During stance phase, evidence suggests that the stretch reflex makes a strong contribution to leg extensor EMG, aiding propulsive force.[8] The rapid time frame of the reflex contribution makes it highly applicable to its use in sprinting where ground contact is less than 100 milliseconds. Factors influencing force production via the stretch reflex include:

- Muscle pre-activity (activity before ground contact). Pre-activity occurs in numerous leg muscles that are involved in generating propulsive force, including gastrocnemius, vastus lateralis and biceps femoris during sprint running.[81] This pre-activity likely increases muscle spindle sensitivity potentiating the stretch reflex contribution.[32,79,87]
- Tendon compliance. The degree of compliance of the tendon affects force through the muscle and the resultant feedback from the muscle spindles.
- Training. It has been speculated that with strength and power training, the length feedback component that originates from the muscle spindles may be enhanced,[88] possibly improving muscle stiffness on contact (see point ii). A similar spindle/stretch-reflex moderated adaptation may occur with high intensity sprint training.[89] Unfortunately, as yet no studies have examined the effect of sprint training in a longitudinal study. However, Voight and associates[90] examined changes in both H- and stretch reflexes in response to 4 weeks of hopping training. While some adaptations were reported following the training period (notably increased soleus tendon reflex and reduced soleus H-reflex depression during hopping) no measurable changes in either gastrocnemius or soleus movement–induced stretch reflexes were observed. This lack of change in the movement-induced stretch reflex may be a function of the short period of training in the study (4 weeks). A longer period of training may be required to make significant changes in the muscle spindle gain.

(ii) *Stiffness of the tendo-muscular system*. Stiffness of the tendo-muscular system appears to be related to, and enhanced by, reflex contribution.[59,91] The stiffness of the tendo-muscular system may affect the use of the SSC with respect to storage and use of elastic energy. Stiffness of the tendo-muscular system has been strongly related to maximal running velocity and speed maintenance.[92,93]

It is likely that a stiffer system would have positive implications for running, such as, increased rate of force development at contact, resulting in decreased contact time and higher peak force. Therefore, reflex control of the stiffness of the tendo-muscular system has important implications for sprint performance. Some factors influencing stiffness regulation via reflex loops include:

- Pre-activity and co-contraction also strongly influence the stiffness of the system by resisting undue joint perturbations on contact, as well as influencing the reflex gain on the active muscles (see point iii).
- Training. As suggested in the force production section, long term training may have an affect on the gain of the resultant afferent feedback from the muscle spindles. Furthermore, if the inhibitory force feedback component via the Golgi tendon organs could be simultaneously decreased, a further increase in muscle stiffness would result.[89]

(iii) *Spinal level control of gait*. As mentioned in section 2.2.2, reflex gain is influenced by the type of muscle action. During sprint exercise it is likely that the type of muscle action regulates changes in excitability. Control of excitability in this manner may help regulate the contribution of an individual muscle to an action. For example, in the triceps surae muscle group evidence suggests that during the contact phase of sprint running the gastrocnemius muscle has an isometric muscle action, whereas the soleus is initially contracting eccentrically.[94] H-reflex changes during initial ground contact would appear to reflect this with gastrocnemius showing a potentiated reflex and soleus being inhibited.[95-97] This control of excitability, to some extent, may influence the organisation and output of individual muscles during sprinting.

A further process for control at this level is reciprocal inhibition. Co-contraction of agonist-antagonist muscle groups appears to affect the reflex activity of co-agonist muscles somewhat differently. For example, co-contraction of tibialis anterior and the triceps surae induces a decrease in soleus H-reflex but no change in gastrocnemius H-reflex.[68] Finally, it has also been suggested that movement commands are processed by stretch reflex mechanisms which improve linearity of response, hence improving control of stiffness and smoothness of action during gait.[59] This damping function prevents oscillation and jerkiness of movement.

In summary, reflexes appear to have numerous effects on gait. Although control of reflexes is influenced by descending input, adaptations to training are not yet fully understood. However, the available evidence suggests that reflexes do aid in damping undesirable oscillations in movement and do affect muscle stiffness, both of which are positively related to sprinting speed. Furthermore, sprint training may enhance muscle spindle sensitivity, which appears to enhance the stretch reflex, making an increased contribution to force production, also beneficial for sprint performance. Finally, it appears the mode of contraction of individual muscles also affects their reflex excitability, a factor that may allow for regulatory control of gait at a spinal level.

3. Neural Fatigue

Neural fatigue is potentially a limiting factor during and for a period of time following maximal sprint exercise. Fatigue of central or neural origin has been defined as an involuntary reduction in voluntary activation.[98] The actual site of neural fatigue is often difficult to establish, although there are a number of possibilities including supra-spinal failure, segmental afferent inhibition, depression of motor neuron excitability and loss of excitation at branch points. Fatigue at the neuromuscular junction may also prevent full muscle activation in sprint exercise. In this review, acute neural fatigue refers to fatigue of neural origin during or immediately after exercise, and long lasting neural fatigue refers to ongoing fatigue (minutes to days later) which may have implications with respect to training frequency and adaptation.

3.1 Acute Neural Fatigue

In a typical 100m running event fatigue will be manifested in a slight decline in speed towards the

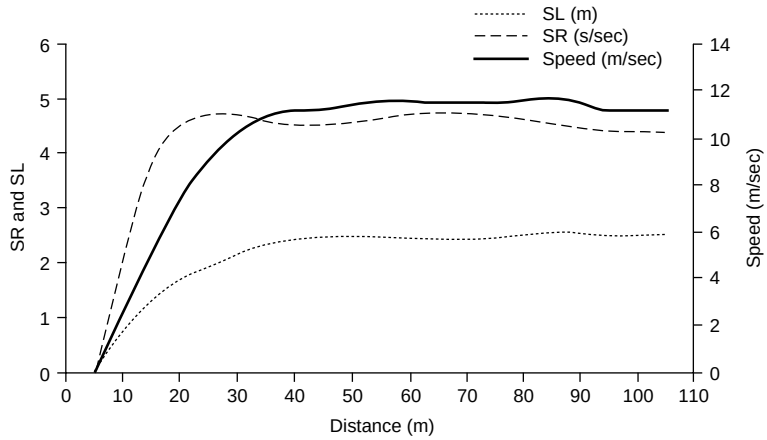

Fig. 2. Variation of speed stride rate (SR) and stride length (SL) during the course of an elite 100m sprint performance (average data from male finalists of the 1991 World Championships 100m final).[21]

later stages of the race. As shown in figure 2, typically this will be evident via a slight decrease in SR as an athlete fatigues. Part of the cause for the declining SR may be of neural origin. As suggested in figure 1, changes in technique, altered recruitment and changes in firing rate are all components with neural influence that can affect SR.

Fatigue of neural origin occurs in maximal intensity exercise within a few seconds of maximal exertion.[99,100] Much of the work in this area has been laboratory based using either animals and dissection techniques, or using electrical stimulation, EMG and nerve blocking techniques – allowing assessment of single motor units in humans.[99] The results from this research indicate that acute neural fatigue is evident, particularly in fast twitch motor units with short contraction times and high axonal CV. Similarly, rate of tension development is affected by neural fatigue.[101] Miller and associates[101] reported that rapid voluntary isometric contractions of adductor pollicus and tibialis anterior muscles slowed significantly within the first minute of exercise. In contrast, electrically evoked contractions became more rapid (twitch stimulation) or did not change (tetanic stimulation). However, slow twitch motor neurones responded continuously to prolonged voluntary drive at rates sufficient for full fusion.[99] So while slow motor neurones and the units they activate may be continuously activated, the use of these motor units may be limited in sprint exercise. The mechanisms for this slow twitch versus fast twitch discrepancy are uncertain, although in a prolonged contraction there is a general decrease in the efficiency of the central drive, which primarily affects motor units with high (force) thresholds, that is, fast twitch. There may also be a selective increase in the threshold of such units. Some high threshold units fire mainly phasically in prolonged contractions compared with low threshold units which, once recruited, continue to fire as long as their critical tension is maintained.[99]

At a whole muscle level during progressive fatigue contraction-relaxation slows, which reduces the need for high activation.[102] Therefore, to maintain optimal force output activation rates should decrease over time. As suggested above, individual motor units display rate reduction profiles tailored to their contractile and fatigue properties. However, recovery from decreasing activation levels is rapid with individuals able to momentarily decrease tension and then making a maximal acceleration to reach electrically evoked tension levels. The likely heavy recruitment of fast twitch fibres in sprinting[103] may result in a substantial degree of acute

neural type fatigue obvious via decreased activation particularly during the latter part of a 100m sprint.

The site of neural fatigue is somewhat uncertain and a number of mechanisms may contribute to fatigue. However, a relatively recent technique, transcranial magnetic stimulation, allows direct stimulation to the motor cortex. During sustained isometric MVCs transcranial magnetic stimulation produces an increase in twitch force within 15 seconds of beginning the MVC.[100] This indicates a less than optimal output from the motor cortex, which leads to less than maximal activation of the skeletal muscle.[100] Whether similar effects occur in maximal but more complex ballistic tasks such as sprinting is uncertain, although not an unreasonable proposition. This suggests that towards the end of a similarly maximal 15-second sprint, output from the motor cortex may be less than optimal and potentially performance limiting.

The inability to maintain maximal activation over 10- to 15-second periods has not been overlooked by leading coaches. Indeed, a maximum velocity drill termed 'ins and outs' uses alternating phases of maximal and marginally submaximal velocity running as a means of improving the top-end speed of an athlete.[69,104] For example, following a period of acceleration an athlete sprints at maximal SR and intensity for 10 to 20m ('in' phase) followed by an 'out' phase of 5 to 20m of marginally less than maximal intensity, in an effort to allow the nervous system to recharge. Potentially this may enable the high (force) threshold units to be re-accessed in the subsequent 'in' phase. This may be repeated 2 to 3 times so race length distances can be covered. This method purportedly allows athletes to run at absolute maximal neural intensity for the 'in' phases rather than being unable to maintain the maximal activation once fatigued from the acceleration phase. Indeed, race models similar to the above are used by some coaches to optimise performance.

In applied sprint literature it has been reported that EMG activity in the skeletal musculature increases with increasing running speeds.[31-33] Therefore, maximal intensity speed training is probably the most stressful type of running on the nervous system. Studies using EMG in high intensity running of longer than the 15-second definition of this review (200 and 400m events) showed an increase in muscle activation during fatigue,[105] indicating that peripheral rather than central mechanisms are causing athletes to slow down. The submaximal intensity pacing strategy employed by athletes in these events allows for the increase in activation. In contrast, sprinting as defined for the purposes of this study (in this case the 100m), showed decreased muscle activation by between 4.9 and 8.7% after the accelerative phase of the race, possibly because of fatigue at the neuromuscular junction and/or a decreased firing rate.[105] Again, the drop out of high (force) threshold units such as the IIb fibres may be a further reason for the decrease in activation, with the declining activation an attempt to optimise output and minimise fatigue. Fatigue distal to the neuromuscular junction is also an obvious cause of the decreased activation. The sub-elite individuals used by Mero and Peltola,[105] decreased velocity after attaining maximal speed to a greater extent than the decrease in maximal EMG, giving a further indication of the magnitude of fatigue distal to the neuromuscular junction. Such data are yet to be collected on elite individuals.

A final possible cause of acute neural fatigue is a decrease in reflex sensitivity. As suggested in section 2.2, the stretch reflexes appear to contribute to propulsive force output during running.[8] A decrease in reflex sensitivity has been observed as a result of large volumes of traumatic SSC exercise, although this is yet to be examined during sprint exercise.[106,107] However, by-products from maximal intensity exercise such as lactate are known to act on group III and IV muscle afferents, which may inhibit reflex pathways, potentially limiting the SSC contribution to propulsion as lactate reaches a certain level.[108,109] Furthermore, even relatively small changes in reflex sensitivity may diminish the quality of sprint performance.

3.2 Long Lasting Neural Fatigue

In contrast to acute fatigue, long lasting neural fatigue is fatigue of neural origin that continues for some time after the cessation of exercise. Neural fatigue and its time course for recovery has been cited by leading coaches as a factor to be considered when assessing the timing and frequency of maximal speed sessions.[110,111] However, specific research to assess such claims, is yet to be conducted. This lack of research may be largely because of the difficulty in assessing maximal activation in a task as complex as sprinting. While interpolated twitch has recently been used during contractions other than isometric[112-114] to assess the degree of voluntary muscle activation it is unlikely to be suitable for a skill as complex as sprinting. Nevertheless there are a number of previous studies that do have implications for sprint training.[107,115-119]

Muscle damage can affect neural function at certain levels, with both reflex changes and disruption of the electrical excitability of the muscle membrane potentially causing changes in the EMG signal for up to 7 days after trauma.[115,116] Extensive muscle damage has been reported following sprint running even among trained individuals,[117] particularly among type IIb muscle fibres. Research suggests that the IIb fibre is the fastest and strongest type of muscle fibre[120,121] giving it obvious applicability to sprint performance. Given that the IIb fibre, in particular, may sustain damage during maximal sprint training it is likely that full recovery of the ability to appropriately activate IIb fibres would be required for a subsequent maximal speed session to be optimal. As with the acute situation, muscle damage has long term implications with respect to reflex changes and muscle activation, with type III and IV muscle afferents being affected by chemical agents associated with muscle pain.[109] Stretch and H-reflex values have shown full recovery after long distance running, and may take up to 4 days for complete recovery.[107] While sprint exercise is of much shorter duration, the more intense SSC exercise may mean that similar effects can occur with a much smaller total volume of exercise. Furthermore, activation of the type III and IV muscle afferents may provide increased inhibitory drive to the α-motoneuron pool, resulting in performance decrements in the SSC exercise.[118]

Evidence of muscle damage causing prolonged neuromuscular dysfunction after eccentric exercise is evidenced by poor proprioception and perceived exertion difficulties experienced by individuals after eccentric exercise.[119] Changes in position sense and perceived force generation would likely adversely influence an athlete's technical model and again make maximum velocity type training in such a state less than optimal.

To summarise, fatigue of neural origin clearly affects force production and sprint performance, hence, training drills have been developed to accommodate its influence. The origin of neural fatigue is likely multifaceted with much of it occurring via afferent feedback loops either as a result of muscle damage or peripheral fatigue. Evidence suggests that centres at least as proximal as the motor cortex are directly affected. Fatigue of the neuromuscular system also has implications for recovery and training frequency during high intensity training periods. Coaches and athletes currently assess muscle fatigue from training using anecdotal evidence, such as, performance and muscle soreness, in a effort to measure response to a training stimulus. These measures may also be indicative of the neural response to training stimuli. Further research is required to optimise both the understanding of neural fatigue and the development of training regimens that accommodate its effects.

4. Conclusion

The nervous system and its state of training is an integral component with respect to sprint performance. While the current state of research is less than comprehensive, it can be concluded that:

(i) Relative sequencing of muscle activation during gross movement changes with practice, therefore, sprint technique is modifiable

(ii) NCV may increase in response to a period of sprint training

(iii) Excessive training may result in negative adaptations with respect to NCV, reflex responses and proprioception
(iv) Stretch reflex appears to aid force production during sprinting
(v) Stretch reflex output is trainable
(vi) Acute fatigue during sprinting may have a neural component though this would appear to be strongly influenced by metabolic changes in the muscle
(vii) Longer lasting fatigue as a result of sprint exercise may also have neural implications with respect to reflex output and proprioception and, therefore, sprinting technique.

There are implications for optimising sprint training from the above points; however, these have to be weighed against metabolic and contractile adaptations to training, with the combination of these factors ultimately determining the performance outcome.

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