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Long-Term Metabolic and Skeletal Muscle Adaptations to Short-Sprint Training

Implications for Sprint Training and Tapering

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Abstract

The adaptations of muscle to sprint training can be separated into metabolic and morphological changes. Enzyme adaptations represent a major metabolic adaptation to sprint training, with the enzymes of all three energy systems showing signs of adaptation to training and some evidence of a return to baseline levels with detraining. Myokinase and creatine phosphokinase have shown small increases as a result of short-sprint training in some studies and elite sprinters appear better able to rapidly breakdown phosphocreatine (PCr) than the sub-elite. No changes in these enzyme levels have been reported as a result of detraining. Similarly, glycolytic enzyme activity (notably lactate dehydrogenase, phosphofructokinase and glycogen phosphorylase) has been shown to increase after training consisting of either long (>10-second) or short (<10-second) sprints. Evidence suggests that these enzymes return to pre-training levels after somewhere between

7 weeks and 6 months of detraining. Mitochondrial enzyme activity also increases after sprint training, particularly when long sprints or short recovery between short sprints are used as the training stimulus.

Morphological adaptations to sprint training include changes in muscle fibre type, sarcoplasmic reticulum, and fibre cross-sectional area. An appropriate sprint training programme could be expected to induce a shift toward type IIa muscle, increase muscle cross-sectional area and increase the sarcoplasmic reticulum volume to aid release of Ca^{2+}. Training volume and/or frequency of sprint training in excess of what is optimal for an individual, however, will induce a shift toward slower muscle contractile characteristics. In contrast, detraining appears to shift the contractile characteristics towards type IIb, although muscle atrophy is also likely to occur. Muscle conduction velocity appears to be a potential non-invasive method of monitoring contractile changes in response to sprint training and detraining.

In summary, adaptation to sprint training is clearly dependent on the duration of sprinting, recovery between repetitions, total volume and frequency of training bouts. These variables have profound effects on the metabolic, structural and performance adaptations from a sprint-training programme and these changes take a considerable period of time to return to baseline after a period of detraining. However, the complexity of the interaction between the aforementioned variables and training adaptation combined with individual differences is clearly disruptive to the transfer of knowledge and advice from laboratory to coach to athlete.

Sprinting involves the rapid release of muscular energy to propel an athlete forward with maximum attainable velocity. Sprinting is a fundamental constituent of many activities, with the ability to move rapidly offering a considerable competitive advantage in most sports. Sprint performance has been widely tested with a variety of measures from peak, mean or total power to more simple measures such as a time trial or velocity measure over a specified distance. These measures are all strongly related and periodically will be grouped under the banner of 'performance' during this review; however, where possible the specifics of the performance measures will be discussed. Sprint performance has generally proved to be a highly reliable measure (see Hopkins and associates[1] for a review) particularly when athletes are used as study participants, with coefficients of variation of 0.9 to 2.0% being reported for sprint running and peak power (bike or treadmill). As such, significant changes in performance reported in the literature in response to training generally indicate meaningful adaptation.

To develop sprint ability, athletes and coaches often employ training methods that involve brief maximal intensity sprint repetitions of varying duration interspersed with either long or short recovery periods. Indeed, this type of training has been shown to elicit improvements in sprint performance (peak and mean power) after as little as 3 weeks of training.[2] The majority of other sprint studies have observed improvements in performance after sprint training of varying duration,[3-12] although there have been some exceptions.[13-15] Training frequency is one factor that may alter performance adaptations with one report suggesting that daily sprint training does not improve either peak or mean cycle sprint power; in contrast, training every third day significantly increased both.[16] This observation is in agreement with the general consensus among leading coaches who typically recommend about three maximal intensity sessions per week.[17,18] There is also considerable variation in the nature and magnitude of performance gains observed in response to sprint training. This variation may be caused by the different sprint-training protocols used (notably differences in volume, duration, recovery and fre-

quency), the training status of individuals and the performance assessment employed.

While sprint training has generally been shown to be effective in improving sprint performance, the effects of detraining and/or reduced training on sprint performance are not so well described. In one of the few articles on this topic Linossier and associates[19] reported that even after a 7-week period of detraining (following 9 weeks of sprint training), peak cycle speed, peak power and time to peak speed were still significantly above pre-training levels. Furthermore, the raw figures in the Linossier et al.[19] article demonstrated that both peak speed and time to peak speed were in excess of the post-training level (although not significantly) after the 7-week detraining period. It also appears that power performance is well maintained as a result of short-term detraining (14 days to 3 months)[20,21] or is improved by short periods of reduced training.[22]

The purpose of this article is to review the long-term metabolic and skeletal muscle adaptations to short sprint (<10-second) training and, where possible, reduced training and rest. Although some researchers have investigated adaptations to short-sprint training ,[3,10,12,13,19] much of the sprint training research has focussed on training involving prolonged (>10-second) sprint repetitions.[15,23,24] Therefore, the literature investigating physiological adaptations to prolonged sprint training will also be briefly discussed where relevant to determine how the nature of different sprint training protocols influences the adaptive process.

1. Metabolic Adaptation

Skeletal muscle is a dynamic tissue that is able to adapt in many different ways to an exercise stimulus. Metabolic adaptations in a muscle are associated with enhancing the ability of the muscle to produce energy. This is achieved by one or more, of three different mechanisms. First, key regulatory enzymes can increase their activity and thus the rate of energy production. Second, increasing the amount of substrate stored in the muscle can increase total energy production. Finally, the muscle can increase its ability to combat the accumulation of certain metabolites associated with fatigue.

Metabolic adaptations to sprint training, and to a certain extent the subsequent detraining adaptations, are largely dependent on the type of sprint training undertaken. Indeed, the bioenergetics of sprint exercise bouts differs markedly depending on the sprint duration and recovery intervals. Energy for muscle contraction during brief maximal exercise of 10 seconds or less is primarily derived from the breakdown of stored muscle phosphagens, such as adenosine triphosphate (ATP) and phosphocreatine (PCr), and glycolysis.[25-28] However, when short sprints are repeated or when sprint exercise lasts for up to 30 seconds, the contribution of anaerobic energy production to the energy yield decreases and a significant amount of energy is derived from aerobic metabolism.[15,26,29,30] Indeed, recent evidence suggests that 13% of energy during a 10-second sprint and 27% of energy during a 20-second sprint is generated aerobically.[27] In fact, no form of exercise can be purely anaerobic or purely aerobic.[31] Therefore, as the metabolic demands of short- and relatively long-duration sprint exercise bouts appear to be quite different, it follows that intramuscular adaptations very much depend on the nature of the sprint-training programme and that adaptations in both anaerobic and aerobic energy systems will occur in response to training.

1.1 Enzyme Adaptation

A summary of enzyme adaptations to sprint training is provided in table I. More detailed discussion of the enzyme adaptation specific to each energy system follows.

1.1.1 Phosphate Metabolism

Hirvonen et al.[25] have suggested that brief maximal exercise performance depends on the capacity for using high-energy phosphates (ATP and PCr) at the beginning of the exercise. Indeed, elite sprint athletes have showed the capacity to deplete PCr levels by over 60% during a 60m sprint.[25] Myokinase [MK; enzyme catalysing the resynthe-

Table I. Enzyme adaptations to sprint training and detraining

Study	n	Training details	Detraining period	Enzyme adaptations
Cadefau et al.[6]	13	8mo, young sprint athletes, sprint running 0-500m, plyometrics, weights etc., frequency not reported		No change in CPK, increased PK, increased PHOS
Dawson et al.[10]	9	6wk, 30-80m sprints, 2-4 min recovery, 3 × week		MK insignificant 18% increase, PHOS significant 41% increase, PFK no change, CS significant 32% decrease
Fournier et al.[32]	6	3mo, 50-250m sprints, 4 × week	6mo	Training: PFK significant 20% increase, SDH no change. Detraining: PFK 32% significant decrease, SDH no change
Hellsten-Westing et al.[33]	11	6wk, 10 sec cycle sprints, 50 sec recovery, 3 × week		PFK significant ~16% increase
Jacobs et al.[15]	11	6wk, 15 and 30 sec cycle sprints, 2.5 × week		CS significant 12% increase, PFK insignificant 16% increase, CPK insignificant 9% decrease
Linossier et al.[34]	7	9wk, 5 sec sprints, 55 sec recovery	7wk	Training: CS and CPK no change, PHOS and LDH significant ~9% increases. Detraining: CS significant 19% decrease, CPK, PHOS and LDH no significant decreases
Linossier et al.[3]	10	7wk, 5 sec cycle sprints (55 sec recovery), 4 × week		CS insignificant 14% increase, PFK significant 19% increase, LDH significant 20% increase
MacDougall et al.[23]	12	7wk, 30 sec cycle sprints, 4 min recovery, 3 × week		PFK significant 49% increase, LDH no change, CS significant 36% increase, SDH significant 65% increase
Parra et al.[16]	SP 5, LP 5	2 groups with matched total volume of work training for 2wk (SP) or 6wk (LP) at 24 or 72 hourly intervals, 15 sec sprints with 45 sec recovery		Significant increases, CPK (SP only), PHOS (LP only), PFK (both), LDH (both), CS (both), PK (SP only), see section 1.1 for further details
Roberts et al.[35]	4	5wk, 8 × 20-30s sprints (200m run), 2 min recovery, 3-4 × week.		Significant increases in PFK, LDH
Simoneau et al.[36]	19	15wk, cycle sprints at 60-90% intensity, 4-5 × week, 15-90 sec efforts	7wk	Training: CPK no change, PFK significant 26% increase. Detraining: no significant decreases
Thorstensson et al.[37]	4	8wk, 3-4 × week, 5 sec sprints on treadmill, rest 25-55 sec		MK significant 20% increase, CPK significant 36% increase, LDH no change

CPK = creatine phosphokinase; **CS** = citrate synthase; **LDH** = lactate dehydrogenase; **LP** = long programme; **MK** = myokinase; **n** = number of participants; **PFK** = phosphofructokinase; **PHOS** = glycogen phosphorylase; **PK** = pyruvate kinase; **SDH** = succinate dehydrogenase; **SP** = short programme.

sis of ATP from adenosine diphosphate (ADP)] activity has been shown to increase (~20%) after short (5-second work intervals with 25- to 55-second recovery) sprint training.[37] Dawson et al.[10] have also reported a similar (~18%), although not statistically significant, increase in MK activity after a very similar protocol of short-sprint training. Therefore, it appears that short-sprint training can elicit relatively small changes in MK activity, which may lead to enhanced ATP resynthesis and improved sprint performance. However, the link between increased ATP resynthesis and improved performance is tenuous at present. Further research examining the relationship between the magnitude of the change in MK activity, ATP resynthesis and the change in sprint performance is required to investigate this hypothesis. Longer sprints (15 seconds) have also been shown to substantially increase MK activity, although adaptation appears to be dependant on training frequency with 72 hourly sessions producing an 18% increase in activity, in contrast to 24 hourly sessions which produced a decrease of ~3%.[16] However, these results again failed to reach significance, possibly because of the small participant numbers

in the study (n = 5 per group). Furthermore, Parra and associates[16] matched only total work with either a long programme (LP), 6 weeks training at 72 hourly intervals, or a short programme (SP) of 2 weeks training at 24 hourly intervals. Such a design is somewhat flawed in that it fails to discriminate between changes caused by the training frequency difference and changes caused by the total duration of the training period, hence, a relatively slow speed of adaptation may lead to misinterpretation of the results. Interpretation of data from such a design should be examined with this in mind.

Hirvonen et al.[25] have demonstrated that the fastest sprinters in a group of seven elite male sprint runners were able to more rapidly deplete their PCr levels than the slower runners in the group. Thorstensson et al.[37] have shown that creatine phosphokinase (CPK; enzyme catalysing the breakdown of PCr) activity increased by 36% after short-sprint training (5-second sprints with 25- to 55-second recovery). However, this increase was statistically significant only at the $p < 0.1$ level in the group of four participants in this study. Similarly, 15-second sprints have also been shown to be effective at increasing CPK levels.[16] Conversely, Nevill et al.[5] have shown that PCr degradation during a 30-second sprint remained unchanged after sprint training involving a combination of short and long sprint repetitions. Other studies have shown that CPK activity does not increase after sprint training.[6,15,34] A lack of CPK adaptation after sprint training may be caused by the use of prolonged sprints in the training protocols of the studies mentioned above. Alternatively, Cadefau et al.[6] have suggested that a relatively large activity of CPK is evident in the muscle of sedentary individuals and the stress induced by a period of sprint training may not be sufficient enough to stimulate an increase in CPK activity. Therefore, although higher level sprinters are able to use PCr more rapidly than others, sprint training consisting of prolonged sprint repetitions does not appear to be able to elicit any change in CPK activity. Not surprisingly, no changes in CPK as a result of detraining have been reported either.[34] Further research is necessary to determine the effect of short-sprint training on CPK activity.

1.1.2 Glycolysis

Glycolysis is initiated at the onset of exercise[38] and contributes significantly to energy production during 10 seconds of maximal dynamic exercise.[28] In fact, glycolysis may contribute between 55 and 75% of the metabolic energy production during sprint exercise lasting ~10 seconds.[27,39,40] Phosphofructokinase (PFK; enzyme that catalyses the phosphorylation of the glycolytic intermediate fructose 6-phosphate) activity has been shown to increase[3,33] or remain unchanged[10] after short-sprint training. However, a number of studies have found PFK activity to be increased after sprint training consisting of either longer-duration training repetitions or a combination of long and short sprint efforts.[6,15,16,23,32,35,41] Similarly, the activities of both lactate dehydrogenase (LDH; enzyme that catalyses the conversion of pyruvate to lactate) and glycogen phosphorylase (PHOS; enzyme that catalyses the mobilisation of stored muscle glycogen into the glycolytic pathway) have been shown to increase after sprint training involving either short (<10-second) or long (>10-second) sprint intervals.[2,10,16,23,34,35] A higher rate of glycolytic enzyme activity may be responsible for superior brief maximal exercise performance in sprint-trained athletes compared with other athletes.[42] However, Jacobs et al.[15] have shown that sprint training (>10-second sprints) caused an increase in PFK activity without an expected corresponding improvement in sprint performance. Similarly, Parra and associates[16] (also using >10-second sprints) showed significant enzymatic adaptations including PFK, PHOS, pyruvate kinase (PK; catalyses the conversion of phosphoenolpyruvate to pyruvate) and LDH in their daily training group without any corresponding increase in performance. Furthermore, Boobis et al.[43] have observed improvements in sprint performance after sprint training, even though there was no increase in energy provision from glycolysis per unit of muscle mass during sprint exercise. Dawson et al.[10] have shown that neither PFK nor

PHOS activity was correlated with short sprint performance before or after a period of sprint training. However, Tesch et al.[44] have shown that high-intensity exercise performance is significantly correlated with PFK activity and the ratio of PFK : citrate synthase (CS) activity. Therefore, although improvements in glycogenolytic capacity are often observed after sprint training, the functional significance of this metabolic adaptation remains questionable.

With respect to detraining, two studies have examined sprint detraining and its effect on glycolytic enzyme activity. Fournier and associates[32] used a 3-month training routine consisting of 50 to 250m sprints followed by 6 months of detraining, and reported a return to pre-training levels of PFK after this period. With a shorter 7-week detraining period, the results from Linossier et al.[34] are a little less clear with LDH found to still be significantly elevated over pre-training levels, although PHOS activity had almost returned to baseline. With these results coming from studies with relatively long periods in the complete absence of training, it is interesting to note that some of the metabolic adaptations have remained. Further research is required to establish the length of time enzymatic adaptations can be maintained during a period of reduced training or detraining.

1.1.3 Aerobic Energy System

The contribution of the aerobic energy system to the total energy requirement of a 10-second bout of maximal exercise is quite small (~13%).[27] However, when short sprints are repeated with short recovery or when sprint exercise lasts for up to 30 seconds, anaerobic energy production decreases and a significant amount of energy is derived from aerobic metabolism.[26,27,29,30] Not surprisingly, increases in the activity of key enzymes of the aerobic energy system [succinate dehydrogenase (SDH) and CS] have been reported after sprint training involving relatively long-duration sprint efforts.[6,15,23,45] Sprint training involving only very short sprint bouts has been shown to decrease[10] or cause no change[3] in CS activity. Similarly, training of mixed duration (50 to 250m efforts) has also demonstrated no change in CS activity.[32] However, an increase in muscle mass might mask any increase in enzyme activity as enzyme activities are usually expressed relative to muscle mass. The results from Dawson et al.[10] are interesting as these authors demonstrated that maximal oxygen uptake ($\dot{V}O_{2max}$) increased after short-sprint training, yet CS activity actually decreased. These results suggest that short-sprint training may induce differential adaptations in central and peripheral aerobic parameters. Alternatively, improving muscle strength and/or resistance to fatigue could increase $\dot{V}O_{2max}$. Clearly, adaptation of the aerobic energy system occurs after short-sprint training; however, further research is necessary to determine the exact nature of this adaptation. Not surprisingly, training using prolonged sprints has also been shown to increase $\dot{V}O_{2max}$.[5,46,47] With regard to the effect on oxygen use during sprint exercise the effects are not as clear; however, McKenna and associates[46] suggested that when gas exchange both during and immediately after sprint exercise is measured it appears that aerobic metabolism during sprint exercise is increased by sprint training using prolonged sprints.

Limited work has been conducted examining sprint detraining and the effect on aerobic enzymes, and given the lack of response of aerobic enzymes to short-sprint training this is perhaps not surprising. However, a significant decrease in CS activity was reported by Linossier et al.[34] after 7 weeks of detraining, despite CS activity not having increased significantly over pre-training as a result of the training period.

1.2 Resting Metabolites

Energy production during high-intensity exercise may be enhanced, or maintained at a high rate for longer, if there is an increase in the amount of metabolic substrate available before exercise commences.[48] A number of researchers have investigated the effect of sprint training on resting muscle levels of ATP and PCr. Both short-sprint training and sprint training consisting of longer duration

training repetitions have been reported to cause no change[3,5,10,37,41] or a decrease in resting muscle levels of ATP and/or PCr. Furthermore, resting levels of ATP in muscle have actually been shown to decrease after 6 weeks of short-sprint training.[49] This is thought to occur because of an elevation in the rapid turnover of ATP associated with regular intense exercise. Rapid turnover of ATP results in a decrease in the ATP : ADP ratio in the muscle. The decrease in this ratio is partially offset by the catabolism of ADP to adenosine monophosphate (AMP). AMP is subsequently deaminated to inosine monophosphate (IMP), which may be degraded to inosine or hypoxanthine.[50,51] As both inosine and hypoxanthine can diffuse across the cell membrane, it is possible that repeated rapid turnover of ATP, which occurs during high-intensity training, results in a loss of purine bases from the muscle. A decrease in resting ATP level has also been observed after longer duration sprint training.[52] The decrease in resting ATP after sprint training may be greater when the volume of training is increased.[49] In contrast, recent work by Parra and associates[16] reported a significant increase in PCr as a result of 2 weeks of daily sprint training. However, this result was not seen with 6 weeks of the same training at three sessions per week. It would appear that changes in ATP and PCr levels may be affected by frequency and volume of training although the majority of sprint studies have reported no significant change. Finally, it is of interest to note that although a reduction in resting ATP levels has been reported after sprint training, significant improvements in power output were still observed in these studies.[49,52] This is perhaps not surprising when it is considered that the contribution of stored muscle ATP to the energy requirements during high-intensity exercise is quite small and unlikely to limit performance;[53] rather it is the rate of resynthesis of ATP that is important. Furthermore, *in vitro* studies have demonstrated that muscle fibres can maintain isometric tension despite low levels of ATP.[54]

Increased resting muscle glycogen stores may improve time to exhaustion during supramaximal exercise.[55] However for 30-second sprint cycle performance (peak power and work) recent work examining short-term changes suggests that muscle glycogen level has no effect on performance.[56] Therefore, for short sprinting it is likely that moderate changes in muscle glycogen have no effect on performance. In response to sprint training involving long-sprint repetitions, resting muscle glycogen stores have been observed to increase.[16] However, Nevill et al.[5] have reported that sprint training consisting of a combination of short- and long-sprint efforts caused no significant increase in resting muscle glycogen level. Nevertheless, the influence of sprint training on resting muscle glycogen stores is difficult to determine without careful control of prebiopsy carbohydrate intake. Consequently, the limited research data in this area makes it difficult to establish the effect of sprint training on resting muscle glycogen stores. Clearly further research is necessary in this area.

In summary, sprint training appears to have little influence on resting muscle levels of ATP and PCr. However, very intense, high volume training may cause small reductions in muscle ATP, but these reductions are unlikely to affect performance. Further research is necessary to determine the effect sprint training has on resting muscle glycogen content.

1.3 Intramuscular Buffering Capacity

Glycolysis is rapid and is initiated very soon after the onset of muscle contraction during high-intensity exercise.[25,26] Consequently, a significant amount of lactic acid accumulates in the muscle during sprint exercise. The hydrogen ions formed from the dissociation of lactic acid have the potential to cause fatigue during high-intensity exercise.[56,57] A full review of buffering and its effect on sprint performance, however, is beyond the scope of this paper (see Juel,[58] Allen et al.[59] and Parkhouse and McKenzie,[60] for related reviews). Nevertheless, it is known that human skeletal muscle has the ability to offset the change in pH through the use of various buffering mechanisms, including chem-

ical buffers bicarbonate, phosphate and proteins and haemoglobin in red blood cells. Furthermore, evidence suggests that with training there are significant improvements in muscle buffering ability. Sahlin and Henriksson[61] have reported in a cross-sectional study that the muscle buffering capacity was higher in team sport athletes than sedentary individuals. Muscle buffering capacity has also been shown to be significantly increased after sprint training involving long sprint efforts[41,62] although not all studies have found such a result.[5] However, with regard to short sprinting, less is known, although Dawson et al.[10] have suggested the increased run time to exhaustion observed after short-sprint training might have been caused by improved muscle buffering capacity. Unfortunately, no study has directly measured the effect of short-sprint training on muscle buffering capacity. Furthermore, there is an absence of detraining work in this area. Clearly, further research is necessary to investigate this potentially important mechanism of adaptation.

1.4 Summary

In summary, during short-term bouts of brief maximal intensity exercise, high-energy phosphagens, glycolysis and oxidative metabolism all contribute to ATP turnover. An increase in the activity of key regulatory enzymes of these energy systems after sprint training would appear to contribute to improved sprint performance. Although a number of studies have reported increases in enzyme activities of each energy system, others have not. Both duration of sprints and frequency of training appear to influence both performance and enzymatic adaptation. Furthermore, a few studies have shown that performance improvements after sprint training are not related to increased enzyme activity. Likewise, the comparative lack of detraining data makes for inconclusive analysis particularly with regard to the time frames required for training effects to be negated both in performance terms and measurable metabolic changes. Nevertheless, with the data available, the effects of detraining appear to be very limited within the first 2 weeks, with negligible changes both in performance and enzyme levels reported. Furthermore, even 7 weeks or longer of detraining does not appear to completely eliminate the effects of the previous sprint training. Sprint training appears to have little influence on resting muscle substrate levels. However, very intense, high-volume training may cause reductions in muscle ATP, but these reductions are unlikely to affect performance improvements. Sprint training does appear to increase anaerobic capacity and muscle buffering ability. Currently, however, there is a limited body of literature on short-sprint training with studies using different qualities of enzyme analyses, different training programmes and analytical techniques providing a less than complete picture of the muscle adaptation. Hence our understanding of metabolic adaptations to sprint training/detraining and the manner in which these adaptations contribute to changes in performance is not quite complete. Further research, particularly focussing on very short-sprint training, is necessary in this area.

2. Morphological Muscle Adaptations

Skeletal muscle composition may influence sprint performance in a number of ways. Muscle contraction speed and strength are determined largely by muscle fibre type, sarcoplasmic reticulum (SR) adaptation and muscle cross-sectional area. Moreover, skeletal muscle is a plastic tissue that can be modified by a variety of exercise training interventions. The following section describes changes in muscle contractile characteristics that have been observed after sprint training and detraining.

2.1 Muscle Fibre Type

A number of different classification systems have been used to describe the different contractile properties of skeletal muscle fibres (see Pette and Staron[63] for a more complete review of this area). Brooke and Kaiser[64] devised a method that enabled muscle fibres to be categorised as slow twitch (type I), fast twitch oxidative-glycolytic (type IIa), and fast twitch glycolytic (type IIb) based on the

different pH sensitivities of their myosin ATPase activity. It was believed that myosin ATPase histochemistry was able to accurately reflect the expression of myosin heavy chain (MHC) isoforms in individual muscle fibres. However, more recent work has revealed that myosin ATPase histochemistry can only identify the predominant MHC isoform contained in individual muscle fibres.[65,66] MHC isoform expression is a major determinant of the contractile characteristics (eg. force development) of skeletal muscle.[67,68] As it has been shown that individual muscle fibres may contain more than one MHC isoform ,[66,69] myosin ATPase histochemistry may not be sensitive enough to accurately assess the contractile character of skeletal muscle tissue. More sensitive techniques such as electrophoresis and immunocytochemistry are now being used to determine the proportion of MHC isoforms contained in individual muscle fibres and the muscle as a whole. Nevertheless, histochemistry has remained widely used within training literature and as such will still be referred to in this review although its limitations are noted.

Classification of MHC subtypes in human skeletal muscle is a matter of some debate with some authors referring to the three major subtypes as I, IIa and IIb[70] while others suggest that the type IIb human MHC is more akin to rodent IIx MHC[71,72] and hence refer to it as IIx. Nevertheless, it appears there is a continuum of MHC isoforms from type I→IIb/IIx, with transitional types between the major subtypes being commonly reported.[73-75] Indications from animal and human studies indicate that type IIb/IIx MHC exhibit highest power outputs, type IIa MHC intermediate power output and type I MHC exhibits the lowest power outputs.[67,70] Recent *in vitro* analyses of human skeletal muscle suggest that MHC isoforms (I, IIa and IIb/IIx) are important determinants of the force of a fibre and power per unit area characteristics (IIb/IIx>IIa>I).[67,68] Furthermore, the maximum unloaded shortening velocity of type IIb MHC is approximately ten times faster than type I MHC.[67] Therefore, a higher percentage of type IIb MHC, or a change in MHC isoforms towards greater type IIb expression after sprint training, would be advantageous for individuals wishing to improve sprint performance.

2.1.1 Fibre Type Adaptations to Sprint Training

Adaptations of the contractile apparatus to a variety of training types has been previously reviewed[76] with endurance training appearing to induce a shift toward type I (IIb→IIa→I), and it was suggested by Pette[76] that both sprint and strength training induce a comparable transition. While there are certainly some similarities (i.e. often a decrease in IIb is reported) there do appear to be adaptations specific to the more anaerobic nature of sprint and/or strength training that are further discussed below. Although it should be noted that it is somewhat difficult to discriminate, with respect to adaptations, between effects of volume and the type of stimulus. At this point it should also be noted that with experimental participant numbers between 6 to 18 in the reviewed studies (table II) the results may not be completely representative of a typical population or indeed indicative of the adaptation in elite athletes or those of a mature training age.

Sprint runners have a larger percentage of type II fibres than other athletes[42] and indeed sprint performance has been strongly correlated with the percentage of histochemically typed type II fibres.[10,79-81] Furthermore, examination of the contractile nature of whole muscle using stimulated contractions in cross-sectional studies demonstrates that sprint athletes have greater rates of both force development and relaxation than what is seen in untrained or endurance trained individuals.[82,83] Such characteristics would appear to be a logical adaptation to the demands of sprint exercise, therefore, it is often assumed that an increase in type II fibre percentage would be a natural adaptation to a period of sprint training. While this is the case in some sprint-training studies (table II),[10,13,15,24] other studies have, surprisingly, shown either a decrease in the percentage of type II fibres (accompanied by an increase in the percentage of type I fibres) after sprint training[3,6,78] or no change in MHC or histochemically typed muscle composition.[11,12,77] Poten-

Table II. Summary of sprint-related studies investigating fibre type/MHC isoform changes in response to sprint training

Study	n	Training duration	Training type/frequency	Performance changes	Change in proportion fibre type/MHC isoforms
Allemeier et al.[14]	11	6wk	30 sec cycle sprints, 2-3 × week	No significant changes	Significant increase in IIa MHC (8.7%), significant decrease in IIb (5.5%), insignificant decrease in I (3.2%) [I→IIa←IIb]
Andersen et al.[4]	6	3mo	Sprint athletes, weight training and sprints (varied distances), 6 d/wk	Significant improvement in 40 and 20m sprint times	Significant fibre shift (I→IIa←IIb), observed both histochemically and electrophoretically
Cadefau et al.[6]	13	8mo	Young sprint athletes, sprint running 0-500m, plyometrics, weights etc., frequency not reported	Improvement of both 60 and 300m times	~8% significant increase in type I and insignificant decrease in IIa, b, c (I←IIa←IIb)
Dawson et al.[10]	9	6wk	30-80m sprints, 2-4 min recovery, 3 × week	Significant improvement of 40m time	~10% significant increase in FT and corresponding decrease in ST (I→II)
Esbjornsson et al.[77]	16	4wk	30 sec cycle sprints (20 min recovery), 3 × week	No significant changes in males, significant increase in peak and mean power in females	No significant changes
Esbjornsson et al.[13]	11	6 + 1wk	10 sec cycle sprints, 50 sec recovery, 3 × week for weeks 1-6, 14 × week for week 7	Insignificant (7%) increase in peak power	Significant fibre shift (I→IIa←IIb) in initial 6wk, increased type I and decreased IIb in week 7
Harridge et al.[11]	7	6wk	3 sec cycle sprints (30 sec recovery), 4 × week	Increase in crank torque at all workloads, increased time to peak tension in muscle twitch	No significant changes
Jansson et al.[24]	15	4-6wk	15 and 30 sec cycle sprints, 2-3 × week	No change	Significant decrease in type I (9%), increase in IIa (6%), insignificant increase in IIb (4%) [I→IIa→IIb]
Jacobs et al.[15]	11	6wk	15 and 30 sec cycle sprints, 2.5 × week.	No change	Significant increase (~7%) in type IIa and insignificant (~10%) decrease in type I (I→IIa←IIb)
Linossier et al.[3]	10	7wk	5 sec cycle sprints (55 sec recovery), 4 × week	Significant increase in peak force, power and velocity	Significant decrease in IIb (8.5%), significant increase in I (8%) [I←IIa←IIb]
Ørtenblad et al.[12]	9	5wk	20 × 10 sec cycle sprints (50 sec recovery), 3 × week	Increased total work and average power	No significant changes
Simoneau et al.[78]	19	15wk	Cycle sprints at 60-90% intensity, 4-5 × week, 15-90 sec efforts	Not reported	6% significant increase in type I, 6% decrease in IIb (I←IIa←IIb)

FT = fast twitch; **MHC** = myosin heavy chain; **n** = number of participants; **ST** = slow twitch; → and ← indicate the direction of the fibre type shift.

tially, those studies reporting the non-advantageous fibre adaptations may reflect their use of inappropriate training protocols, including some endurance work or only prolonged sprinting,[6,77,78] or insufficient recovery between repetitions (possibly[3,11]) or training too frequently (possibly[3,78]) or finally insufficient total training duration for measurable significant adaptation (possibly[77]). While it is not possible to make conclusive judgements with the data available it may be hypothesised that decreases in the percentages of type II fibres may be related to the training being in some way excessive or inappropriate for the individuals involved.

The mechanisms or factors that stimulate a change in muscle contractile characteristics [e.g. towards IIb/IIx (I→IIa→IIb/IIx)] are uncertain and likely

to vary between individuals. However, there are a number of studies examining physiological factors that are thought to be influential. Firstly, both stretch and stimulation have been shown to cause repression of IIb myosin in rabbit tissue, with the authors suggesting the IIb is the default gene, that is, it responds positively to a lack of stimulation, namely, rest.[84] This theory has received support in further animal research with the enforced unweighting of rat hind limbs causing a significant increase in IIb MHC isoforms in postural muscles.[85] Secondly, stimulation intensity and recovery duration between bouts of stimulation have been examined in denervated rat tissue with three different stimulation groups; low frequency stimulation (20Hz) every 15 seconds, high-frequency stimulation (150Hz – suitable for recruiting large fast twitch motor units) every 15 seconds, and high frequency stimulation every 15 minutes plus a nonstimulated control group.[86] After ~2 months of stimulation the resulting analysis found nonstimulated tissue was 79% IIa/IIx, and both low- and high-frequency stimulation every 15 seconds produced tissue that was ~90% IIa/IIx. In contrast, high-frequency stimulation every 15 minutes was only 52% IIa/IIx and 45% the more rapidly contracting IIb. Such results suggest the importance of recovery and training frequency in muscle adaptation, although a fourth stimulation group combining low frequency stimulation on 15-minute intervals may have made the results more discriminating. Nevertheless, the data suggest that recovery duration may be a factor contributing contractile adaptations. Finally, general overloading of muscle has also been examined in rat tissue via the surgical removal of synergist muscle, the remaining muscle showing an increase in histochemically typed type I fibres.[87]

In more applied sprint studies with human participants some similarities with the work of Ausoni and associates[86] can be seen. As previously suggested (table II), a number of the sprint studies that have produced a shift toward type I (I←IIa←IIb) have used protocols with sprints of excessive duration and/or frequency with short recovery periods between repetitions.[3,6,13,78] As prolonged sprinting involves a considerable amount of aerobic energy production ,[27,29,30] an increase in type I fibre percentage may indeed be a positive adaptation. Similarly, repeated sprints with inadequate recovery tax the ability of the muscle to oxidise lactate and resynthesise PCr. An increase in type I fibre percentage may benefit these two aerobic processes. Therefore, the influence sprint training has on muscle fibre type transition is likely dependent upon the nature of the sprint training protocol undertaken.

Muscle fibre type changes may also be related to the frequency of training. Esbjornsson et al.[13] found that 6 weeks of sprint training (repeated 10-second sprints) performed three times a week induced a significant increase in the percentage of IIa muscle fibres. In contrast, when participants trained twice daily for an additional week, an increase in the percentage of type I muscle fibre was observed; however, this trend was not in evidence in all participants and no changes in performance (peak or mean power) were reported. Furthermore, as histochemical rather than the more sensitive electrophoretic techniques were used, strong conclusions are difficult to draw from the study. A follow-up study with similar methodology, although perhaps with more sensitive performance measures and direct examination of MHC adaptation, would be a worthwhile addition to the literature. Nevertheless, such research illustrates that potentially the frequency of sprint training may influence the nature of changes to muscle contractile characteristics. The substantial increase in volume in the overload week of the Esbjornsson et al.[13] study, however, confounds whether changes are caused by excessive volume of training or excessive frequency of training. Nevertheless, the time frame for such changes appears to be rapid, with significant adaptation possible within a week.[13,87]

In those papers where electrophoretic techniques have been used, there typically appears to be a bidirectional shift towards IIa (I→IIa←IIb).[4,14] Anderson and associates[4] used well trained sprint ath-

letes and reported such a shift (I→IIa←IIb) with a 17.6% increase in type IIa MHC isoforms in response to three months of contemporary (including resistance training) sprint training using both histochemical and electrophoretic techniques. In parallel with the MHC adaptations both acceleration times (0-20m and 0-30m) and maximal velocity (flying 20m) improved during this period. Similarly, Allemeier et al.[14] observed an increase in the percentage of type IIa MHC and a concomitant decrease in the percentage of type IIb MHC after 6 weeks of sprint training involving prolonged (30-second) sprint cycle bouts. A nonsignificant decrease in type I MHC (3.2%) was also in evidence in their results. Interestingly, no change in histochemically determined fibre type was evident after sprint training in this particular study, perhaps showing the lack of sensitivity of this technique. In comparison to the Anderson et al.[4] study, the smaller increase in IIa MHC numbers (8.7%) in this study[14] was reflected in no significant change in sprint performance (peak or mean power) and is perhaps a function of the shorter training period (6 weeks *vs* 3 months).

Individual differences and genetic predisposition to different types of training would also appear to influence muscle fibre adaptations in response to sprint training.[13,19,71] Linossier and associates[19] reported two subgroups with essentially opposite fibre type adaptations (histochemically typed) to sprint training with short recovery. Although their participants were reportedly 'untrained' it is conceivable that there may have been substantial differences in the levels of conditioning among individuals at the commencement of training, which may have influenced adaptations. However, an equally plausible suggestion is that the genetic makeup that maintains a high proportion of type IIb MHC isoforms despite a heavy training load may be what discriminated these subgroups, and potentially such a genotype may also distinguish elite sprint performers from the sub-elite. Similarly, such a genetic profile may explain the shift to fast twitch characteristics seen among certain individuals in the work by Esbjornsson and associates.[13] Furthermore, this may give some explanation to the high training frequency and still positive contractile adaptation seen with the elite sprint athletes examined by Anderson and associates[4] (table II). Again, further use of electrophoretic technique studies would be advantageous in the majority of these studies to more closely examine individual adaptations to sprint training regimens of different types.

2.1.2 Fibre Type Adaptations to Rest and/or Reduced Training

As has been discussed in section 2.1.1 and table II, somewhat in contrast to the potentially beneficial shift toward the explosive type IIb/IIx fibres, the common adaptation to most sprint-training interventions is a decrease in type IIb/IIx MHC and an increase in type IIa MHC (often I→IIa←IIb). Interestingly similar responses have been reported in response to resistance training.[20,88,89] Type IIa MHC are more fatigue resistance than type IIb MHC and may be a more suitable adaptation for the sprint athlete as the ability to resist fatigue, even during very short sprint bouts, can play a role in determining sprint performance. Alternatively, if the IIb default gene theory of Goldspink and associates[84] is considered the shift to type IIa MHC observed after most sprint-training research interventions may be because these training protocols allow insufficient rest and may not optimise muscle fibre adaptations.

Anecdotal evidence suggests that short-sprint performance is usually greatest following a period of rest or reduced training such as a competition taper. Recent research assessing changes in the muscle contractile elements may provide some clues as to some of the mechanisms involved. Indeed, Anderson and Aagaard[20] have recently demonstrated the overshoot phenomenon whereby the percentage of type IIb/IIx MHCs can actually increase *beyond* pre-training levels when resistance training is followed by a 3 month period of inactivity. Furthermore, no significant differences in dynamic power from 0 to 15 seconds were found when comparing the post-training and post-detraining results. That is, this power measure did show a significant and rel-

atively long lasting, improvement from pre-training and it is likely that sprint performance would respond similarly. It is also notable that dynamic power after 15 seconds (15 to 40 seconds) declined back to pre-training levels as a result of detraining, perhaps indicative of the probable difference in responses of short (<10-second) and prolonged (>10-second) sprinting to detraining. Similar fibre type shifts have also been reported in other studies as a result of detraining.[73,85,90] This overshoot phenomenon may explain why other researchers have found that performance decrements in short sprint and power tests are often negligible despite substantial periods of detraining and likely decrements in enzymatic adaptations and cross-sectional area.[19,20,36] Similarly, large increases in rate of torque development in a stimulated twitch have been reported as a result of a detraining period following 8 weeks of resistance training.[91] Additionally, in the most extreme example of human detraining (i.e. long-term deafferentation via spinal cord injury fibre) composition changes are most radical with almost a complete shift toward IIb being commonly reported.[75] These types of results and the noted adaptation toward IIb with rest has prompted some elite sprint coaches/scientists to advocate interspersing phases of recovery with those of high training load in an effort to maintain the high IIb status of untrained muscle.[92] Clearly, further research is necessary to quantify how best to schedule training and recovery cycles to optimise skeletal muscle and performance adaptations to sprint training.

2.2 Muscle Fibre Size

As can be seen in table III sprint training alone does not appear to increase muscle fibre size significantly during short-term (6 to 7 weeks) training periods often despite performance improvements.[3,14] However, significant increases (5 to 16%) in both type I and type II fibre area have been observed after sprint training ranging in duration from 8 weeks to 8 months.[6,8] Similarly, significant increases in total muscle volume have been reported following 9 weeks of sprint cycle training.[19]

With prolonged detraining, muscle fibre cross-sectional area would be expected to decrease, particularly in fast twitch muscle, which has been shown to hypertrophy as a result of sprint training. Indeed, as shown in table IV, among elite power trained athletes some decrease in cross-sectional area has been shown in fast twitch muscle within 14 days of detraining, despite no decrement in jump performance.[21] In contrast, a swim training taper involving a reduced total volume of work but an increased proportion of sprint work elicited a significant 24% increase in type IIa cross-sectional area.[92] How-

Table III. Changes in muscle fibre cross-sectional area with sprint training

Study	n	Training duration	Training type	Performance change	Change in fibre diameter/muscle size
Allemeier et al.[14]	11	6wk	30 sec cycle sprints, 2-3 × week	No significant changes	6-12% insignificant increase
Cadefau et al.[6]	13	8mo	Sprint running 0-500m, plyometrics, weights etc.	Improvement of both 60 and 300m times	~8-16% significant increase*
Harridge et al.[11]	7	6wk	3 sec cycle sprints (30 sec recovery), 4 × week	Increase in crank torque at all workloads, increased time to peak tension in muscle twitch	3.7-6.7% insignificant increase
Linossier et al.[3]	10	7wk	5 sec cycle sprints (55 sec recovery), 4 × week	Significant increase in peak force, power and velocity	Significant ~10% decrease in ST diameter,* ~6% decrease in FT diameter
Sleivert et al.[8]	8	14wk	10 sec cycle sprints (120 sec recovery), 3 × week	Significant improvement in power output from 0-15 sec in cycle sprint	~5-10% significant increase*

FT = fast twitch; **n** = number of participants; **ST** = slow twitch; * indicates $p < 0.05$.

ever, more global measures of muscle volume have shown no decrease in total muscle volume following 7 weeks of detraining, with quadriceps and muscle volume remaining significantly elevated over pre-training values.[19]

From the aforementioned results cross-sectional area would not appear to be the greatest determinant of sprint performance particularly with regard to short-term adaptation. Hypertrophy in conjunction with maintenance or improvement of the type II/I ratio, however, would be expected to improve power and thus sprint performance in the longer term and would appear from the physique of current elite sprint athletes to be a prerequisite to success at the top level. Nevertheless, this is not an easy goal to achieve, with the majority of the literature on highly hypertrophied/body builder human participants demonstrating slower contraction/relaxation characteristics than untrained individuals[94] and a strong MHC or fibre type shift away from IIx/IIb toward IIa and possibly type I.[94,95] In addition to the negatively altered contractile properties, the additional bodyweight from excessive hypertrophy is also likely to be performance limiting for weight-bearing sprint modalities such as running.

2.3 Sarcoplasmic Reticulum Adaptations

The SR plays a critical role in muscle contraction and its actions may strongly regulate both muscle rate of contraction and relaxation. Release of Ca^{2+} from the SR is believed to permit muscle contraction to occur by exposing the active sites on the muscle actin and allowing myosin to attach. Subsequent reuptake of Ca^{2+} occurs on the completion of the contraction via an active transport system (Ca^{2+}-ATPase) that pumps Ca^{2+} from the cytosol back to the SR.[96] A greater development of the SR allows the release and reuptake of the Ca^{2+} to occur more quickly, hence the size and structure of the SR is crucial to muscle contraction and relaxation rate (RR).

Evidence suggests that muscle twitch contraction time is inversely related to the fractional fibre volume of the SR.[97] It is known that fast twitch muscle contains approximately twice the volume and area of terminal cisternae as slow twitch muscle[98] and has faster reuptake of Ca^{2+}.[99] Furthermore, fast twitch fibres have 5 to 7 times greater Ca^{2+}-ATPase enzyme density and consequently such fibre has the ability to rapidly fluctuate force levels.[100] However, recent research suggests that fibre type alone does not determine SR structure (including Ca^{2+}-ATPase isoform density) and volume

Table IV. Changes in muscle fibre cross-sectional area with sprint detraining

Study	n	Detraining/ taper duration	Prior training type	Performance change	Change in fibre diameter/muscle size
Hortobágyi et al.[21]	12	14d detraining	Power training, well trained athletes	No change in vertical jump (insignificant increase in counter movement jump and drop jump)	6.4% decrease in FT area (significant*) 5.2% decrease in ST area (insignificant)
Linossier et al.[19]	7	7wk detraining	5 sec cycle sprints for 9wk	No decrease in peak speed or peak force after detraining (still significantly greater than before training)	~9% increase in type I, ~4% decrease in type IIa, ~13% decrease in IIb (all insignificant). No change in total muscle area
Trappe et al.[93]	6	21d taper	Swimming training including sprinters	4% increase in swim performance, increase in swim bench power, increase in shortening velocity of type I and IIa, increase in peak force of IIa fibres	24% increase in IIa fibre CSA,* no change in type I CSA

CSA = cross-sectional area; **FT** = fast twitch ; **n** = number of participants; **ST** = slow twitch; * indicates $p < 0.05$.

in muscle fibre.[12] It is known that there can be adaptation of the SR in response to training (see Green[100] for a more comprehensive review in this area); however, the body of work on adaptation to sprint training is very limited.

Ørtenblad and associates[12] have provided indirect evidence to suggest that the volume of SR in muscle may increase as a result of 5 weeks of sprint training, without causing concurrent changes in MHC isoform composition. In addition to improved sprint performance (notably increased work, rate of power development and average power per kg), their results showed a significant increase in the rate of Ca^{2+} release. However, the rate of relaxation was unchanged as was SR Ca^{2+}-ATPase capacity. Nevertheless, analysis of the Ca^{2+}-ATPase isoforms demonstrated large significant increases in ATPase isoforms of both slow and fast calcium pumps, providing some evidence to suggest a slow to fast transformation might be occurring. However, the immunoblot technique used to assess changes in the Ca^{2+}-ATPase isoforms does not assess functionality, which may account for the large increase in Ca^{2+}-ATPase isoforms and no change in RR or SR Ca^{2+}-ATPase capacity. It is possible the time frame for these isoforms to reach a fully functional state and thus for the change in volume of isoforms to be reflected functionally via an increased RR and increased SR Ca^{2+}-ATPase capacity, may have been longer than the duration of the study. Similarly, rapid changes in SR function such as Ca^{2+} uptake capacity and Ca^{2+}-ATPase (and associated changes in force production) have been reported in response to denervation[101] or long-term stimulation[102] and have been shown to occur more rapidly than MHC changes or histochemically typed fibre adaptation. As with the Ørtenblad et al.[12] study these results suggest that there is a different time course in the development of SR and MHC adaptations in response to training and that the rapid adaptation to a training stimulus may be caused by SR-mediated changes in Ca^{2+} release.

In view of the aforementioned results, SR and Ca^{2+}-ATPase-mediated adaptations may also explain contractile changes as a result of a period of tapered training. Trappe and associates[93] have provided evidence showing increased peak force in type IIa fibres and dramatic increases in maximal unloaded shortening velocity in both type I and IIa muscle fibres as a result of tapered training (with an increase in sprint emphasis).

Such results suggest that sprint training and or reduced training may be able to modify contraction characteristics/performance of skeletal muscle without or before changes in MHC, and may account for some positive performance adaptations seen in table II, despite negative or no measurable change in fibre type adaptations. Further work is required to establish the time frame for these adaptations and the resilience of such adaptations to detraining.

2.4 Muscle Conduction Velocity as a Further Indicator of Structural Change?

Muscle conduction velocity (MCV) is defined as the speed of impulse/action potential transmission from the motor point along the length of a muscle. MCV has been correlated with muscle half relaxation time, rise time and twitch torque; however, it is likely that these correlations come about as a function of MCV being largely determined by muscle fibre type and fibre cross-sectional area.[103,104] As such, measurement of MCV or changes in it may be only indirectly related to changes in muscle contractility and, more directly, reflect muscle fibre type (notably membrane excitability characteristics) and diameter. Nevertheless, MCV provides an indication of the contractility of a relatively large portion of the entire muscle, whereas fibre type and cross-sectional area are usually determined using a highly localised muscle biopsy sample. Furthermore, the non-invasive nature of the MCV technique makes it a potentially more useful measure for muscle level adaptations to training.

To date, only one cross-sectional study has been conducted using sprint athletes as participants.[103] This study demonstrated that MCV was significantly faster in sprint-trained athletes than in endurance-trained athletes. In addition, muscle conduction ve-

locity was strongly correlated (r = 0.84) with muscle fibre type measured histochemically from biopsy samples. Furthermore, the ability of MCV to discriminate between the two athletic groups was greater than the ability of muscle fibre type measurements to discriminate between the two groups. That is, the slowest sprinter's MCV was greater than the fastest endurance athlete's MCV; while the same pattern was seen in the biopsy data, this level of clear group discrimination was not. This raises the possibility that sprint performance may be more closely related to MCV than the previously demonstrated association between sprinting and muscle fibre type.[82] However, further research is needed to substantiate this possibility.

The research into longitudinal changes in MCV as a result of sprint training consists of one article with limited statistical analysis. However, Bianchi and associates[105] reported a consistent trend of increased MCV as a result of sprint training, particularly in individuals with a very low initial performance. Similarly, a 16-week resistance training study[106] reported a 3.5% increase in MCV although this was not statistically significant. As discussed in section 1.2 and presented in table I, a number of sprint studies have reported a significant increase in type II muscle fibres as a result of sprint training. Such changes and the possible increase in cross-sectional area of the muscle as a result of sprint training make it likely that MCV would increase with appropriate sprint training. Perhaps duplicating the methodology of one of the aforementioned sprint studies showing significant changes, and substituting MCV for biopsy work, is the next step in determining the usefulness of MCV measurement as a means of monitoring adaptation of skeletal muscle to training.

The paucity of MCV research is even more evident in detraining with sprint or structured power training/detraining studies being unavailable. Despite the lack of sprint-related work, cross-sectional work with spinal cord patients has demonstrated an increase in MCV as a result of such extreme detraining/inactivity.[107] Interestingly, this result parallels an increase in type IIb/IIx MHC despite considerable muscle atrophy. This highlights the importance of the fibre contractile characteristics in determining MCV.

2.5 Summary

From this section detailing skeletal muscle fibre adaptations to sprint exercise, a number of factors are apparent. Firstly, sprinters have high percentages of type II muscle fibres and these proportions are well correlated with sprint performance. This fibre composition is modifiable via sprint training with most sprint training studies reporting a bi-directional shift to IIa (I→IIa←IIb/IIx). Training frequency, within session recovery time, duration of sprint repetition and general load on a muscle appear likely to influence contractile adaptations. A prolonged period of sprint training induces muscle hypertrophy, particularly in fast twitch muscle. Sprint training further modifies the muscle contractile characteristics via increased development of the SR, not necessarily in conjunction with changes in MHC isoforms. Individuals exhibit differences in these adaptations to similar training regimens presumably because of genetic differences or previous training history discrepancies. Detraining eventually causes a shift in muscle fibre composition towards type II; however, atrophy will also occur after prolonged disuse, hence balancing this trade off is the challenge for individual athletes and their coaches. Finally, MCV is a non-invasive indicator of muscle fibre type and fibre diameter, which in future may prove useful in optimising training regimens with respect to muscle fibre adaptations.

3. Conclusions and Applications

At the most basic level it can be concluded that short-sprint performance can be improved by sprint training in the modality being measured. The precise characteristics of training that result in the greatest improvements in performance remain uncertain and likely will differ substantially between individuals. However, optimal fibre type/MHC adaptations may be induced for most individuals, with a less frequent and lower volume of total training than

that seen in most contemporary programmes. As such, rest and recovery would appear to be undervalued training variables. Furthermore, it would appear that some changes in MHC isoforms begin to be expressed within a week of heavy (I←IIa←IIb/IIx) or perhaps tapered training (potentially I→IIa→IIb/IIx). Although positive adaptations to MHC profiles may occur in response to reduced training, lower training volume and frequency may not be optimal for enzymatic, SR and muscle fibre size adaptations. Nevertheless, the effects of detraining on short sprint or power performance as well as metabolic adaptation appear less severe than may have been reasonably anticipated. Longer sprints (>15 seconds), however, may be more adversely affected by detraining than short sprints. These factors have important implications for the design of the annual training plan for a short sprint athlete. In many cases athletes and coaches are often attempting to concurrently maximise adaptations that seem to require increased volume of training (enzymes, muscle fibre size, SR adaptation, enzymatic adaptation) and those that may respond optimally to reduced training (i.e. MHC composition). This may not be the most effective approach, although currently it is not possible to suggest with any certainty as to what the optimal short sprint training protocol is and likely it would differ substantially between individuals.

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