Muscle Fiber Types and Training

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Keywords: muscle fiber types; muscle fiber recruitment; training; fast-twitch; slow-twitch.

HOW SKELETAL MUSCLES ADAPT
to a repeated stimulus depends, to a large extent, on the neuromuscular recruitment of muscle fibers as well as on the inherent characteristics of the muscles themselves. The specific types of fibers that make up individual muscles greatly influence the way your clients will adapt to their training programs. There is a reason why some people get bigger muscles more easily than others and why some people are able to run for much longer periods of time without fatigue. In order to design programs that will work best for each of your clients, it is important for fitness professionals to understand at least some of the complexities of skeletal muscles.

■ Types of Muscle Fibers
Humans have basically 3 different types of muscle fibers. Type I (slow-twitch [ST]) fibers are identified by a slow contraction time and a high resistance to fatigue. Structurally, they have a small motor neuron, a high mitochondrial and capillary density, and a high myoglobin content. Energetically, they have a low supply of creatine phosphate (CP), a low glyogen content, and a wealthy store of triglycerides. They contain few of the enzymes involved in glycolysis, but contain many of the enzymes involved in the oxidative pathways (Krebs cycle, electron transport chain). Functionally, type I fibers are used for aerobic activities requiring low-level force production, such as walking and maintaining posture. Most activities of daily living use type I fibers.

Type II (fast-twitch [FT]) fibers are identified by a quick contraction time and a low resistance to fatigue. The differences in the contractile properties that gives the fibers their names can be explained, in part, by a specific component of the myosin filament—the myosin heavy chain—which exists in 3 different varieties, or isoforms: type I, IIA, and IIB. In addition, the rate of release of calcium by the sarcoplasmic reticulum (the muscle's storage site for calcium) and the activity of the enzyme (myosin–adenosine triphosphatase [ATPase]) that breaks down adenosine triphosphate [ATP] inside the myosin head influence the speed of contraction among the fiber types. Both of these characteristics are faster and greater in type II fibers (12, 19).

Type II fibers are further divided into type IIA (FT-A) and type IIB (FT-B) fibers. Type IIA fibers have a moderate resistance to fatigue and represent a transition between the 2 extremes of the type I and type IIB fibers. Structurally, type IIA fibers have a large motor neuron, a high mitochondrial density, a medium capillary density, and a medium myoglobin content. They are high in CP and glycogen and have a medium level of triglyceride stores. They have both a high glycolytic and oxidative enzyme activity. Functionally, they are used for prolonged anaerobic activities with a relatively high-force output, such as running a long sprint and carrying heavy objects. Type IIB fibers, on the other hand, are very sensitive to fatigue and are used for short anaerobic, high-force production activities, such as sprinting, jumping, and lifting a very heavy object. Type IIB fibers contract about 10 times faster than type I fibers (3). These fibers are also capable of producing more power than type I fibers.
Like the type IIA fibers, type IIB fibers have a large motor neuron but a low mitochondrial and capillary density and myoglobin content. They also are high in CP and glycogen, but low in triglycerides. They contain many glycolytic enzymes but few oxidative enzymes. Table 1 summarizes some major characteristics of the 3 fiber types.

In addition to the 3 major divisions of muscle fibers, there are also hybrid forms of these fiber types, which contain mixtures of slow and fast myosin isoforms. These hybrid fibers are scarce in young people, with older adults having a greater amount of hybrid fibers.

At any given velocity of movement, the amount of force produced depends on the fiber type. During a dynamic contraction, when the fiber is either shortening or lengthening, an FT fiber produces more force than an ST fiber (12). Under isometric conditions, during which the length of the muscle does not change while it is contracting, ST fibers produce exactly the same amount of force as FT fibers. The difference in force is only observed during a dynamic contraction. At any given velocity, the force produced by the muscle increases with the percentage of FT fibers and, conversely, at any given force output, the velocity increases with the percentage of FT fibers. However, regardless of fiber-type distribution, as the velocity of movement increases, the force produced decreases (6, 10, 17, 34, 35).

There is great variability in the percentage of fiber types among people. For example, it is well known that endurance athletes have a greater proportion of ST fibers, whereas sprinters and jumpers have more FT fibers (8, 27). The greater percentage of FT fibers in sprinters enables them to produce greater muscle force and power than their ST-fibered counterparts (12). However, FT fiber percentage has a greater influence on the production of explosive maximal strength (i.e., power) than on the production of maximal strength alone (7). It seems that FT fibers are the main contributors to force production during maximal ballistic movements, such as jumping or sprinting, whereas ST fiber contribution increases as the muscle contraction time increases.

Differences in muscle fiber composition among individuals have raised the question of whether muscle structure is an acquired trait or is genetically determined. Studies performed on identical twins have shown that muscle fiber composition is greatly genetically determined (24). However, there is evidence that both the structure and metabolic capacity of individual muscle fibers can adapt specifically to different types of training. For example, heavy resistance training has been reported to cause a decrease in the percentage of type IIB fibers and an increase in the percentage of type IIA fibers (1, 18, 29). This finding seems to be a result of the change in the myosin heavy chain isoform content (1, 2), suggesting that training can cause a genetic transformation among the FT fiber subtypes.

### Table 1

<table>
<thead>
<tr>
<th>Property</th>
<th>Type I (slow-twitch)</th>
<th>Type IIA (fast-twitch A)</th>
<th>Type IIB (fast-twitch B)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contraction time</td>
<td>Slow</td>
<td>Fast</td>
<td>Very fast</td>
</tr>
<tr>
<td>Size of motor axon</td>
<td>Small</td>
<td>Large</td>
<td>Very large</td>
</tr>
<tr>
<td>Resistance to fatigue</td>
<td>High</td>
<td>Intermediate</td>
<td>Low</td>
</tr>
<tr>
<td>Energy System</td>
<td>Aerobic</td>
<td>Long-term anaerobic</td>
<td>Short-term anaerobic</td>
</tr>
<tr>
<td>Force production</td>
<td>Low</td>
<td>High</td>
<td>Very high</td>
</tr>
<tr>
<td>Mitochondrial density</td>
<td>High</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Capillary density</td>
<td>High</td>
<td>Intermediate</td>
<td>Low</td>
</tr>
<tr>
<td>Oxidative capacity</td>
<td>High</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Glycolytic capacity</td>
<td>Low</td>
<td>High</td>
<td>High</td>
</tr>
<tr>
<td>Major storage fuel</td>
<td>Triglycerides</td>
<td>Creatine phosphate, glycogen</td>
<td>Creatine phosphate, glycogen</td>
</tr>
</tbody>
</table>
Muscle contractions are initiated by impulses, called action potentials, which are conveyed by a neural cell called a motor neuron. Instead of recruiting individual muscle fibers to perform a specific task, a motor unit—a group of muscle fibers innervated by a single motor neuron—is recruited. The recruitment of motor units is controlled by neuromuscular processes, ultimately leading to the production of muscular forces. Motor neurons originate in the central nervous system and terminate in skeletal muscles. The space where the motor neuron and the muscle meet is aptly named the neuromuscular junction. At rest, sodium ions (Na⁺) are most heavily concentrated on the outside of the nerve membrane, causing it to be electrically positive, whereas the inside of the nerve, which contains potassium ions (K⁺), is electrically less positive or negative with respect to the outside. Under the influence of the neurotransmitter acetylcholine, which is released at the neuromuscular junction, the muscle membrane becomes highly permeable to Na⁺, causing Na⁺ to rush inside the membrane. As a result, the outside of the membrane becomes negative and the inside positive, reversing its polarity. This reversal of polarity is called depolarization and results in the formation of an action potential. The action potential propagates along the muscle fiber, eventually leading to muscle contraction.

Motor units are recruited along a gradient. During voluntary isometric and concentric contractions, the orderly pattern of recruitment is controlled by the size of the motor unit (specifically the size of the motor axon supplying the motor unit), a condition known as the size principle (20). Small motor units (those with a small motor axon diameter), which contain type I (ST) muscle fibers, have the lowest firing threshold and are recruited first. Demands for larger forces are met by the recruitment of increasingly larger motor units. The largest motor units (those with the largest axon diameter) contain the type IIB (FT-B) fibers, which have the highest threshold and are recruited last. Thus, if the exercise intensity is low, ST motor units may be the only ones that are recruited. If the exercise intensity is high, such as when lifting heavy weights or doing interval training, ST motor units are recruited first, followed by FT-A and FT-B if needed.

There is some evidence to suggest that the size principle could be altered or even reversed during certain types of movements—specifically those that contain an eccentric (muscle lengthening) or ballistic component—such that FT motor units are recruited before ST motor units (11, 18, 25, 28, 32). It is possible that a preferential recruitment of FT motor units, if it exists, is influenced by the speed of the eccentric contraction and can only occur using moderate to fast speeds (21, 25).

Determining Fiber Type

Since the only way to directly determine the fiber-type composition in an individual is to perform an invasive muscle biopsy test (in which a hollow needle is inserted into the muscle and a core sample of muscle fiber is extracted for examination under a microscope), some studies have tried to indirectly estimate the fiber-type composition within muscle groups of an individual by testing for a relationship between the different properties of fiber-type and muscle-fiber composition. Research using isokinetic dynamometers or electrical stimulation has yielded promising results, with significant relationships being found between the proportion of FT fibers and muscular strength or power (9, 14–16, 31). For the fitness professional or coach who does not have access to laboratory equipment, an alternative method to determine approximate fiber composition using strength training equipment may have some value in directing future training. First, establish the 1 repetition maximum (1RM) of your client for different exercises. Then have him or her perform as many repetitions at 80% 1RM as he or she can for each exercise. If he or she can only perform a few repetitions (<7), then the muscle group is likely composed of more than 50% FT fibers. If he or she can perform many repetitions (>12), then the muscle group likely has more than 50% ST fibers. If your client can perform between 7 and 12 repetitions, then the muscle group probably has an equal proportion of fibers (26). Although this method has not been proven scientifically, it offers a way for the fitness professional to assess the capabilities of his or her clients’ muscle groups. Because lifting weights in the fitness center requires the use of many muscles at once, this method only works to assess characteristics of muscle groups, rather than individual muscles. In order to determine the fiber-type composition of an individual muscle, a needle biopsy of the muscle of interest must be performed.

Implications for Training

Thinking about the task for which a muscle is used (or for which you want it to be used) will help determine how the muscle should be
trained. For example, while a basketball player or sprinter may train the gastrocnemius muscle in the calf for strength and power to improve jumping ability and speed, a distance runner may train it for endurance.

In addition to the variations in fiber type from person to person, muscle fibers will also vary from muscle to muscle within a person. When your clients strength train, their fiber-type proportions will play a major role in the amount of weight that they can lift, the number of repetitions that they can complete in a set, and the desired outcome (increased muscular strength or endurance). For example, a client with a greater proportion of FT fibers will not be able to complete as many repetitions at a given relative amount of weight as will a client with a greater proportion of ST fibers, and therefore will never attain as high a level of muscular endurance as will the ST-fiber client. Similarly, an individual with a greater proportion of ST fibers will not be able to lift as heavy a weight as will an individual with a greater proportion of FT fibers, and therefore will never be as strong as will the FT-fiber person.

Training for endurance, strength, or power causes changes in the genetic expression of myosin ATPase and myosin heavy chains (1, 2), resulting in an altered contractile function of myosin that favors the specific demand of training. For example, depending on the specific training, FT-B fibers can take on some of the endurance characteristics of FT-A fibers, and FT-A fibers can take on some of the strength and power qualities of FT-B fibers. Heavy strength training (6–8RM) has also been shown to decrease the percentage of type IIB fibers while increasing the percentage of type IIA fibers (29). It seems that any interconversion of fibers that exists is limited to the FT fiber subtypes. There is no evidence that an ST fiber can be converted to an FT fiber as a result of training. Therefore, no matter how much sprint training an elite marathon runner performs, he will never become an elite sprinter. There is some interesting evidence from studies performed on rabbits that an ST fiber can be made to behave like an FT fiber if the nerve that supplies the ST fiber is surgically interchanged (cross-reinnervated) with one that supplies an FT fiber (4, 5), suggesting that the behavior of muscles is greatly influenced by the activity of their nerves. However, no similar studies have been performed on humans, and there is no evidence that training has a similar effect on muscle fibers. Until surgically cross-reinnervating nerves in humans is considered ethical practice, we are left with old-fashioned training.

Although the type of fiber cannot be changed from one to another, training can change the amount of area taken up by the fiber type in the muscle. In other words, there can be a selective hypertrophy of fibers based on the type of training. For example, someone may have a 50/50 mix of FT/ST fibers in a muscle, but since FT fibers have a larger cross-sectional area than ST fibers, much more than 50% of that muscle’s area may be FT and much less than 50% may be ST (26). Following a resistance-training program for improvement in muscular strength, the number of FT and ST fibers will remain the same (still 50/50), however the cross-sectional area will change (26). Depending on the specific training intensity used, the muscle may change to a 75% FT area and a 25% ST area. The change in area will lead to greater strength. In addition, since the mass of FT fibers is greater than that of ST fibers, the individual will gain mass, as measured by the circumference of the muscle.

If a relatively untrained individual trains for muscular endurance (and therefore has minimal recruitment of FT fibers), the ST fibers will hypertrophy, causing a greater relative cross-sectional area of ST fibers and a smaller relative area of FT fibers. The area of the muscle, which may have begun at 65% FT and 35% ST before training, may change to 50% FT and 50% ST following training. The endurance capability of the muscle will increase while its strength will decrease, and the individual will lose some muscle mass, again because ST fibers are lower in mass than FT fibers. In addition, long-term aerobic training is associated with a decreased cross-sectional area of all fibers to optimize oxygen uptake kinetics (23, 33). The decrease in muscle mass may be observed by a smaller circumference of the muscle.

In order to improve muscular strength, many muscle fibers need to be recruited simultaneously. Lifting heavy weights recruits the FT-B fibers, which are capable of producing a greater dynamic force than the ST or FT-A fibers. Positive changes in strength (or endurance) will only occur in those muscle fibers that are overloaded, so the FT-B fibers must be recruited in order to be trained (36). Training with a low or moderate intensity will not necessitate the need to recruit the FT-B fibers. Therefore, the training intensity must be high. But how heavy a weight and how many repetitions should you use? Muscular strength is primarily developed when using loads less than a
10–12RM (the maximum amount of weight that can be lifted 10–12 times) (36). When the aim of training is to increase maximum strength via emphasis on neuromuscular processes, at least 95% of the individual’s 1RM and 1–3 repetitions should be used. This intense type of training increases muscle force production by enhancing the neuromuscular processes responsible for the simultaneous recruitment of motor units and the frequency of their stimulation. When the aim of training is to increase maximum strength by stimulating muscle hypertrophy, multiple sets of 5–8 repetitions at 80% or higher of 1RM should be used (36) and should include systematic variation in volume and intensity (30), such as with periodized training programs (22). Hypertrophy can result either from a growth of the sarcoplasm and noncontractile proteins, or from an increase in the number of actin and myosin filaments (36). This latter type of hypertrophy leads to a greater strength potential, since the amount of muscle force production is proportional to the number of actin and myosin crossbridges. This difference in hypertrophy may be observed in the difference between Olympic weightlifters and bodybuilders—both types of individuals have muscle hypertrophy; however, the Olympic weightlifter can produce a greater force. Typically, fewer sets are used when training for muscular strength. Remember, in order to improve muscular strength, FT-B fibers must be recruited. If the aim of training is to increase muscle size (hypertrophy) and definition with moderate gains in strength, then multiple sets of 6–12 repetitions should be used (12). Bodybuilders typically use this repetition range. For maximum results, try to train your clients according to their genetic predisposition. For example, someone with a greater proportion of ST fibers would adapt better to a muscular-endurance program using more repetitions of a lighter weight. Likewise, someone with a greater proportion of FT fibers would benefit more from a muscular-strength program using fewer repetitions of a heavier weight. Of course, the client’s goals and needs should be considered as well. You wouldn’t want to train a 40-year-old female client to develop larger muscles if she does not want to develop larger muscles, even if she does have a greater proportion of FT fibers. ▲

References

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Topical Review

Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications

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(Received 7 August 2001; accepted after revision 27 September 2001)

In eccentric exercise the contracting muscle is forcibly lengthened; in concentric exercise it shortens. While concentric contractions initiate movements, eccentric contractions slow or stop them. A unique feature of eccentric exercise is that untrained subjects become stiff and sore the day afterwards because of damage to muscle fibres. This review considers two possible initial events as responsible for the subsequent damage, damage to the excitation–contraction coupling system and disruption at the level of the sarcomeres. Other changes seen after eccentric exercise, a fall in active tension, shift in optimum length for active tension, and rise in passive tension, are seen, on balance, to favour sarcomere disruption as the starting point for the damage. As well as damage to muscle fibres there is evidence of disturbance of muscle sense organs and of proprioception. A second period of exercise, a week after the first, produces much less damage. This is the result of an adaptation process. One proposed mechanism for the adaptation is an increase in sarcomere number in muscle fibres. This leads to a secondary shift in the muscle’s optimum length for active tension. The ability of muscle to rapidly adapt following the damage from eccentric exercise raises the possibility of clinical applications of mild eccentric exercise, such as for protecting a muscle against more major injuries.

All forms of exercise, if carried out vigorously enough, can become painful. But only one form of exercise, eccentric exercise, if we are unaccustomed to it, leaves us stiff and sore the next day. During eccentric exercise the contracting muscle is forcibly lengthened. One commonly encountered example of eccentric exercise is downhill walking. As we step down the slope, the contracting quadriceps muscle controls the rate of knee flexion against the force of gravity and in the process the muscle undergoes an eccentric contraction with each step. Immediately after the exercise there is no pain. This sets in several hours later and peaks at about 48 h. It is thought to result from muscle damage produced by the eccentric exercise.

An interesting and important feature is the adaptation process. A second bout of eccentric exercise, a week after the first, leaves us much less stiff and sore. The ability of muscle to rapidly adapt to the damage from eccentric exercise, to prevent further damage, provides the opportunity for a number of clinical applications.

The subject of eccentric exercise and its mechanism has been discussed previously in a number of reviews (Armstrong et al. 1991; McHugh et al. 1999; Morgan & Allen, 1999; Warren et al. 2001; Allen, 2001). Brief perusal of the literature indicates that the subject is of growing interest. The aim in this review is to focus attention particularly on some of the indicators of the damage from unaccustomed eccentric exercise and their possible mechanisms. The discussion will consider how such indicators might be used to assess the degree of protection available to an individual in the event of exposure to further eccentric exercise and how this kind of knowledge might be useful in the clinic.

Initial event

It is generally agreed that there are two prominent signs of damage in a muscle immediately after it has been subjected to a series of eccentric contractions. There are the presence of disrupted sarcomeres in myofibrils and damage to the excitation–contraction (E–C) coupling system. It remains a point of controversy which of these two represents the primary event. The view taken here (see also Morgan & Allen, 1999) is that the damage process begins with overstretch of sarcomeres (Fig. 1). The alternative view is that the starting point is damage to components of the excitation–contraction (E–C) coupling process. In a recent review, Warren et al. (2001) summarised their position by declaring that 75% or more of the decline in tension after eccentric exercise was attributable to a failure of the E–C coupling process. The remaining damage seen during the first few days after
the exercise was attributed by the authors to physical disruption of the tension-bearing elements within the muscle. So the suggestion is that most of the primary damage arises in the E–C coupling system and only a small component occurs at the level of the sarcomeres. Supporting evidence comes from the observation that in mouse muscle the post-exercise deficit in tension can be recovered with caffeine (Warren et al. 1993; Balnave & Allen, 1995). In the first of these studies, tension was recovered with 50 mM caffeine, which releases Ca\(^{2+}\) from the sarcoplasmic reticulum and leads to development of a contracture in the muscle. In the second, 10 mM caffeine was used to potentiate tension in single fibres in response to direct electrical stimulation. It was concluded that in mouse fibres changes in E–C coupling may be a major contributor to the observed fall in tension after eccentric contractions (Allen, 2001).

However all of this still leaves open the question of what comes first, E–C coupling failure or sarcomere disruption. With respect to the effects of potentiators, there does seem to be a species difference. In single frog fibres (Morgan et al. 1996) and in toad muscle (Talbot, 1997; Allen, 2001) the fall in tension could not be recovered by potentiating Ca\(^{2+}\) release.

There is a specific hypothesis for the process of sarcomere disruption (Fig. 1). It has been known for some time that the descending limb of the sarcomere length–tension curve is a region where sarcomere inhomogeneities develop (Gordon et al. 1966). It has been proposed by Morgan (1990) that during active stretch of a muscle, most of the length change will be taken up by the weakest sarcomeres in myofibrils or, more strictly speaking, the weakest half-sarcomeres. On the descending limb of the length–tension curve, these sarcomeres will become progressively weaker and when they reach their yield point, they will lengthen rapidly, uncontrollably, to a point of no myofilament overlap, where tension in passive structures balances the active tension in adjacent sarcomeres that still have myofilament overlap. This process is repeated iteratively, with the next-weakest sarcomere stretching, and so on. It is postulated that overstretched sarcomeres are distributed at random along muscle fibres. At the end of the stretch, when the muscle relaxes, myofilaments in the majority of overstretched sarcomeres re-interdigitate so that they are able to resume their normal function. A few may fail to do so and become disrupted (Talbot & Morgan, 1996). During repeated eccentric contractions it is postulated that the number of disrupted sarcomeres grows, until a point is

![Figure 1. Postulated series of events leading to muscle damage from eccentric exercise](image)
reached where membrane damage occurs. It is at this point that damage to elements of the E–C coupling machinery becomes apparent. Subsequently the fibre may die (Fig. 1).

It is conceptually more difficult to envisage damage to the E–C coupling process as the primary event. Observations by Takekura et al. (2001) of abnormal t-tubular arrangements after eccentric exercise could provide the basis for such a hypothesis. Here the first step in the damage process would be t-tubule rupture. Torn tubule ends would then seal off leading to inactivation of some sarcomeres. If such sarcomeres were concentrated in particular myofibrils it could lead to a fall in tension. This tension would be recoverable by a caffeine contracture but not by potentiators. If inactivated sarcomeres were scattered at random amongst myofibrils, the situation would be very similar to that where the primary event was sarcomere disruption. That is, ultrastructurally, a non-uniform sarcomere length distribution would be observed and mechanically, a shift in optimum length for active tension would occur, in the direction of longer muscle lengths. The main difficulty with such a hypothesis is trying to account for why t-tubules should be the primary site for damage and why this only occurs at lengths beyond the optimum. The reverse sequence, beginning with sarcomere disruptions and leading to t-tubule damage, could, of course, account equally readily for the observations of Takekura et al. (2001).

Structural signs

It is no longer a matter of controversy that eccentric exercise leads to structural signs of muscle damage (Friden et al. 1981; Newham et al. 1983). Most of the evidence comes from electron microscopic examinations and these show sarcomeres out of register with one another, Z-line streaming, regions of overextended sarcomeres or half-sarcomeres, regional disorganisation of the myofilaments and t-tubule damage; for a review see Morgan & Allen (1999).

The precise details of the sarcomere disruption process following eccentric contractions remain the subject of speculation. They may involve the elastic filament titin, which anchors thick filaments to Z discs, or the structural protein desmin, which links adjacent Z discs (Allen, 2001). It is conceivable that because of small alignment errors, thick and thin filaments of overstretched sarcomeres may butt up against one another. Inactivation of some sarcomeres from damage to t-tubules may also play a part. Whatever the precise details, there is evidence of overextended sarcomeres and half-sarcomeres in muscle which has undergone eccentric contractions (see, for example, Brown & Hill, 1991; Lieber et al. 1991; Wood et al. 1993; Talbot & Morgan, 1996). It has recently been shown with permeabilised segments of single fibres of rat muscles that regions of long sarcomere lengths before an active stretch contained the majority of disrupted sarcomeres.
sarcomeres after the stretch. These disrupted sarcomeres were longer than the rest (Macpherson et al. 1996).

A diagnostic structural feature of disruption after eccentric exercise is the presence of overstretched half-sarcomeres, with the adjacent half-sarcomere contracted down to a short length (for examples, see Brown & Hill, 1991; Talbot & Morgan, 1996; Macpherson et al. 1997). This consistent feature prompted us to speculate about the arrangement of elastic filaments within sarcomeres. Titin is believed to anchor the ends of the thick filaments to the Z-line (Horowits, 1999). In a simple mechanical model of a sarcomere, if the titin was attached just to the ends of the thick filaments, the two half-sarcomeres would behave independently of one another and overstretch of one half would not be expected to lead to shortening of the other half (Fig. 2). If, however, a second elastic element is included, one that spans the full length of the sarcomere, the structural changes observed in electronmicrographs are achieved. That is, a shortened half-sarcomere facing an overstretched half-sarcomere (Fig. 2).

There is less information available about structural damage to the E–C coupling system. In a recent study of rats which had been exercised by downhill running, forelimb muscles showed a number of ultrastructural abnormalities including more longitudinal t-tubule segments, changes in disposition of triads, caveolar clusters and apposition of multiple t-tubule segments with terminal cisternae elements (Takekura et al. 2001). The disordered membrane systems were seen widely distributed throughout muscle fibres. These findings were interpreted as consistent with a hypothesis starting with focal sarcomere damage, sliding of myofibrils past one another, and eventual damage to t-tubules.

**Shift in optimum length**

Is there any evidence from the mechanical properties of muscle to support the existence of overstretched sarcomeres in damaged muscle? It has been proposed that the presence of overstretched sarcomeres increases series compliance, leading to a shift in the muscle’s active length–tension relation in the direction of longer muscle lengths (Morgan, 1990). Such a shift was first described by

![Figure 3. Changes in mechanical properties of muscle following a series of eccentric contractions](image)

**A**, disruption of sarcomeres. Computer simulated sarcomere length–tension relations. The dashed line is the active length–tension relation taken from Gordon et al. (1966). The dotted line is an exponential curve representing passive tension; the continuous line is the total tension. Tension is normalised relative to the maximum active tension. Length is given as that of a postulated muscle fibre comprising 10,000 sarcomeres with a sarcomere length of 2.5 µm at optimum length. The control curve is the continuous curve on the left. After a series of eccentric contractions 10% of the sarcomeres have their active force set to zero to simulate becoming disrupted, leading to a shift in optimum length of the total tension curve by 3 mm (continuous curve to the right). **B**, adaptation of the muscle fibre following injury from eccentric exercise. The continuous curve is the control total tension curve as in the upper panel, the dashed curve, that after the number of sarcomeres in series has been increased by 10%, without changing the length of the tendon. It has led to an increase in optimum length by 2 mm.
Katz (1939) and since then has been shown for single frog fibres (Morgan et al. 1996), whole amphibian muscle (Wood et al. 1993; Talbot & Morgan, 1996) and human muscle (Jones et al. 1997; Brockett et al. 2001). A modelled example is shown in Fig. 3A.

If it were argued that the primary cause for the tension deficit after eccentric contractions was E–C coupling failure (Warren et al. 1993), leading to a reduced but uniform Ca\(^{2+}\) release, a shift in the length–tension relation could be interpreted as indicating nothing more than a reduced level of activation, so that the muscle had to be stretched further to achieve maximum activation (Endo, 1973). There is evidence that such an explanation does not always hold. For single frog fibres (Morgan et al. 1996) and whole rat muscle (Fig. 4), examples can be found where the length–tension curves from before and after the eccentric contractions cross at long lengths (Katz, 1939; see also Brockett et al. 2001a,b). At these long lengths, the post-exercise tensions are higher than before the exercise so that here incomplete activation cannot be used to explain the change in the curve (Fig. 4).

The sarcomere non-uniformity hypothesis predicts that damage will only occur if sarcomeres are actively stretched to beyond optimum length. If sarcomere disruption and damage are especially prevalent on the descending limb of the muscle’s length–tension curve, indicators of damage should show a length dependence. That is indeed the case. For both rat and toad muscle the shift in optimum length and the fall in active tension post-contraction depended on the length range over which stretches were given (Lynn & Morgan, 1998; Talbot & Morgan, 1998).

A shift in the optimum length for active tension, in the direction of longer muscle lengths, is a rather indirect indicator of increased muscle compliance, post-exercise. Are there any other mechanical changes in the muscle, consistent with an increase in compliance? In his original account, Katz (1939) commented on the 2–3 times slower rise of the isometric tetanic tension and fall in twitch:tetanus ratio consistent with a ‘partial transformation of active contractile into passive elastic tissue’. In a recent series of experiments on the medial

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Figure 4. Activation and the length–tension relation

_A_, torque–angle relationship for the vastus intermedius muscle of the anaesthetised rat. Torque–angle curves were measured before (continuous line) and after (dotted line) a series of eccentric contractions of the muscle. Here the muscle was stretched through 27 deg in 33 ms while being stimulated at 90 pulses per second. Stretches were arranged to start 5 deg short of the optimum angle and finished at 22 deg beyond optimum. Included knee angle is the angle subtended between knee and thigh. At each length, the ratio of torque before and after the contractions has been calculated (dashed line) giving an estimate of the activation fraction (modified from Allen, 1999).

_B_, computer simulation of partial activation. The continuous curve is the fully activated sarcomere length–tension curve (Gordon et al. 1966). The dashed curve is a Hill plot (Hill, 1913), as a reasonable estimate of length dependence of the activation fraction. The dotted curve is the resulting partial activation curve. The partial activation simulation gives a realistic shift in optimum length for tension but is unable to simulate active tension seen at long lengths after a series of eccentric contractions. That is, at long lengths tension lies well below the control curve. Note also the greater fall in tension at short lengths.
Different fibre types

Many studies have considered the susceptibility of different muscles and parts of muscles to damage from eccentric contractions. A question that has been raised repeatedly concerns the vulnerability of different types of motor units in muscles of mixed, slow–fast fibre composition. Claims have been made for a predisposition to damage in slow units during locomotion (Armstrong et al. 1983; Mair et al. 1992). The reasons put forward include the low recruitment threshold and important postural roles of these motor units. When muscles of mixed, slow–fast fibre composition were subjected to maximal active lengthenings, the large, fast-fatiguable motor units were more vulnerable, it was suggested, because of their lack of oxidative capacity (Friden & Lieber, 1998), or the higher tensions generated by them (Appell et al. 1992). In another recent study demonstrating preferential fast-oxidative-glycolytic fibre damage, it was suggested that fibre phenotype or lower contractile workload might be responsible (Vijayan et al. 2001). Others have suggested a combination of factors involving both active and passive properties of muscle fibres (Macpherson et al. 1996). In a recent review, Lieber & Friden (1999) proposed that the larger amount of fibre injury in fast-glycolytic fibres after eccentric exercise was a result of the ‘increased strain and injury due to their short fibre length’.

An important feature of the sarcomere non-uniformity hypothesis is the dependence of damage on the length range over which eccentric contractions are carried out. It raises the possibility that optimum lengths for different fibre types may not be the same so that in a muscle of mixed composition, stretch of the whole muscle leads some fibres to be stretched further down the descending limb of their length–tension curve than others. In a recent study from our laboratory (J. Talbot, M. Homewood & D. L. Morgan, unpublished observation), the predominantly fast-twitch, tibialis anterior muscles and slow-twitch soleus muscles of rats were subjected to a series of eccentric contractions. The applied active stretches were carefully arranged to cover the same portion of each muscle’s length–tension relation. Shifts in optimum length for active tension were observed in both muscles, indicating that the contractions had produced damage in both fibre types. The size of the shift was not significantly different between the two muscles. The result suggested that fibre-type composition, as such, was not a determinant of the amount of damage from eccentric contractions, provided the contractions covered an equivalent range of muscle lengths.

However, it still remained to explain the reports of others concerning susceptibility to damage of fibres within the same muscle, of mixed fibre composition. In a second experiment (Brockett et al. 2001a), slow-twitch and fast-twitch motor units were studied in the medial gastrocnemius muscle of the cat. It was found that the
majority of fast-twitch units had an optimum length for tension that was shorter than the whole muscle optimum length. Conversely, slow-twitch units had an optimum length, on average, longer than the whole muscle optimum. When motor units were subjected to a series of eccentric contractions, using stretches that began at the whole-muscle optimum, all units showed a shift in their length–tension relation, indicative of damage. However, the slow-twitch units showed a smaller shift than fast-twitch units. Statistical analysis showed that a motor unit’s optimum length for tension, relative to the whole-muscle optimum, was a better indicator than motor unit type for the susceptibility of a unit to damage from eccentric exercise. The difference in optimum length of the two motor unit types was thought to be due to differences in numbers of sarcomeres in series in muscle fibres. This, in turn, was thought to relate to the role of the different motor unit types in gastrocnemius during standing and walking in the cat (Brockett et al. 2001b).

The result now needs to be confirmed for other muscles of mixed fibre composition.

Rise in passive tension

It has been known for some time that after a period of eccentric exercise there is a rise in passive tension in the muscle. For human elbow flexors this is indicated by the relaxed arm adopting a slightly flexed posture (Jones et al. 1987). When muscle stiffness was measured, this more than doubled after the exercise and remained elevated for the next 4 days (Howell et al. 1993). The immediate rise in stiffness post-exercise was postulated to result from stretch-activated Ca$^{2+}$ release (Howell et al. 1993). Other explanations have been based on shortening of parallel, non-contractile elements in the muscle (Howell et al. 1985; Jones et al. 1987). The scheme shown in Fig. I has passive tension rising from development of a local contracture in fibre segments, following a rise in myoplasmic Ca$^{2+}$, as a result of membrane damage.

Several studies have demonstrated increases in resting Ca$^{2+}$ levels in muscle fibres damaged by eccentric contractions (Balnave & Allen, 1995; Balnave et al. 1997; Ingalls et al. 1998). However, within the limits of resolution of the method, measurements showed that the rises in Ca$^{2+}$ levels were uniformly distributed along muscle fibres (Balnave et al. 1997). Perhaps the rise in Ca$^{2+}$ is sufficient to trigger a low level of activation to increase passive tension, although there is no direct evidence for this (Whitehead et al. 2001, p. 602).

When measured across the full physiological range, the rise in passive tension after a series of eccentric contractions peaks at a length close to the optimum for active tension (Whitehead et al. 2001). Measurements of work absorption by the passive muscle in response to large, slow lengthening and shortening movements showed a significant rise after the eccentric contractions. It was suggested that the increase in work absorption was a result of actively cycling cross-bridges in the damaged segments of muscle fibres (Whitehead et al. 2001). There is some structural evidence for contracted fibre segments in muscle damaged by eccentric exercise. Thus, it was found that after downhill running, fibres of soleus muscles of rats showed Z-line dissolution, A-band disruption and fibre clotting (Ogilvie et al. 1988). Similarly Friden & Lieber (1998) found that in rabbit muscle after eccentric contractions there was cytoskeletal disruption and the presence in fibres of hypercontracted regions.

Muscle swelling and soreness

Eccentric exercise is followed by sensations of stiffness and soreness the next day (Hough, 1902). The current view of the mechanism is that damage at the level of sarcomeres leads, during repeated contractions, to more extensive damage and, ultimately, to death of some muscle fibres. The injury triggers a local inflammatory response that is accompanied by some oedema. The breakdown products of injured tissues sensitise nociceptors (Smith, 1991; MacIntyre et al. 1995).

It has been suggested that a component of the stiffness after eccentric exercise is due to the swelling accompanying the damage. Thus Howell et al. (1985) suggested that delayed increases in stiffness in exercised elbow flexor muscles were the result of volume changes exerting strain on perimysial and epimysial connective tissue elements. A quantitative biomechanical model supported this view (Purslow, 1989). However, passive tension and stiffness change immediately post-exercise (Howell et al. 1993; Chleboun et al. 1998; Whitehead et al. 2001), when there is not yet any evidence of swelling. In our experiments, there was a rise in passive tension in ankle extensor muscles which reached close to its peak value immediately after the exercise. At 24 h after the exercise, when swelling peaked, there was no significant further increase in passive tension. So under the conditions of our experiments there did not appear to be a close link between passive tension rises and muscle swelling. The swelling began to subside by 4 days post-exercise.

Soreness sets in at about 6–8 h after the exercise and peaks at about 48 h (MacIntyre et al. 1995; Jones et al. 1997). The current view of the mechanism is that the tissue breakdown products sensitise nociceptors so that these respond to stimuli that are normally non-noxious. So the muscle is tender to local palpation, stretch and contraction. It has recently been proposed that a component of the delayed soreness from eccentric exercise involves large-fibre mechanoreceptors (Barlas et al. 2000; Weerakkody et al. 2001). It has been postulated that input from mechanoreceptors, including afferents of muscle spindles, is able to access the pain pathway at the level of the spinal cord.

Adaptation

We have all had the experience that the stiffness and soreness following a period of exercise become very much
less when the exercise is repeated a week later, the result of adaptation by the muscle. This has been known for some time (Hough, 1902; Friden et al. 1983; Schwane & Armstrong, 1983; Clarkson & Tremblay, 1988). However, the underlying mechanism remains controversial (for a review see McHugh et al. 1999).

In his original proposal for a mechanism for the damage from eccentric exercise, Morgan (1990) suggested that the subsequent adaptation process involved an increase in the number of sarcomeres in series in muscle fibres. As a consequence, at a set muscle length, average sarcomere length would be shorter (Fig. 3B). Therefore less of the muscle’s working range would include the region of potential instability, the descending limb of the length–tension curve. Supporting evidence comes from experiments in which rats were exercised on an inclined or declined treadmill (Lynn & Morgan, 1994; see also Lynn et al. 1998). Fibres of a muscle known to undergo eccentric contractions during downhill running, vastus intermedius, were fixed, digested and sarcomere numbers counted after a week of exercise. It was found that the mean number of sarcomeres was, on average, 11% greater in the muscles of animals which had run downhill, compared with those which had run uphill.

This proposal has been challenged (Koh & Herzog, 1998; see also Koh & Brooks, 2001). However, the contrary findings are difficult to evaluate since in that study of rabbit muscle no indication was given of the length range, with respect to the optimum length, over which the muscles were stretched, nor was it stated whether the eccentric contraction protocol used was followed by any detectable signs of disruption or damage.

Scepticism over an adaptation mechanism involving the addition of extra sarcomeres to muscle fibres centres around the time course (McHugh et al. 1999). If muscle fibres are going to be re-modelled after injury from eccentric exercise, this process must be rapid enough to be substantially complete by the end of a week following the injury. Is muscle able to undergo such a rapid adaptation process at the level of the sarcomeres? It was shown back in 1973 that immobilising a muscle in the lengthened position with a plaster cast led to an increase in sarcomere number in muscle fibres within 5 days (Williams & Goldspink, 1973). This increase was rapidly reversible. While we do not yet understand the precise details, at the cellular level, of the events that lead to the adaptation of sarcomere numbers (Goldspink, 1998; see also Wretman et al. 2001), the speed of the process is obviously sufficient to account for the adaptation observed after eccentric exercise.

Figure 5. Sarcomere length–muscle length relation
Relation between sarcomere length and muscle length for theoretical muscle fibres comprising different numbers of sarcomeres and different lengths of tendon. It is assumed that optimum sarcomere length is 2.5 µm. For a muscle fibre with 10000 sarcomeres and 20 mm of tendon (fibre no. 1) tension begins to rise at 35 mm and the optimum is reached at 45 mm (dashed line). A shift in optimum length for active tension by 5 mm in the direction of longer lengths (dashed line) can be achieved by increasing the length of tendon to 25 mm (fibre no. 2). The drawback is that active tension is not developed until the muscle is stretched to 40 mm, that is, the working range of muscle lengths has been reduced. Increasing the number of sarcomeres from 10000 to 12000, and leaving tendon length at 20 mm (fibre no. 3) produces the required increase in optimum length and leads to a smaller reduction in the muscle’s working range, where tension begins to rise at 39 mm. Increasing sarcomere number further to 14000, while at the same time reducing the length of tendon to 15 mm (fibre no. 4) produces the most satisfactory result, the required shift in optimum length by 5 mm and reduction of the working range by only 1 mm.
If the muscle adapts to the injury from eccentric exercise with an increase in sarcomere numbers in muscle fibres, what implications does this have for muscle–tendon relations? We have tried to model this situation (Fig. 5). Consider a muscle composed of muscle fibres containing 10 000 sarcomeres and 20 mm of tendon. In this model muscle tension would begin to rise when the muscle was stretched to 35 mm length, representing a sarcomere length of 1.5 \( \mu \text{m} \). Tension would peak at 45 mm or 2.5 \( \mu \text{m} \) sarcomere length. If the aim was to shift the muscle’s length–tension curve in the direction of longer lengths, to provide protection against injury from eccentric exercise, a simple adaptation would be to increase the length of the tendon by 5 mm. That would shift the optimum length by 5 mm. However, tension would be less well maintained at short muscle lengths, falling to zero at 40 mm rather than at 35 mm. If the number of sarcomeres in the fibre was increased by 20\%, without changing the length of the tendon, the requisite shift in optimum length would still be obtained but the working range of the muscle would be reduced by 3 mm. Only if sarcomere number was increased further and the tendon shortened could both a shift in optimum length and maintenance of the original working range of the muscle be achieved (Fig. 5). In practice, it is unlikely that the tendon is able to be remodelled within a week, although this remains an option for adaptation in the longer term.

Is there any evidence for a shift in the length–tension curve as a sign of a muscle’s adaptation to the injury from eccentric contractions? First, it is important to distinguish between the two kinds of shift that may occur in association with eccentric exercise. Following a period of unaccustomed exercise there will be a shift in optimum length as a result of the increase in series compliance from disrupted sarcomeres (Fig. 3A). This is followed by a second, delayed shift representing the adaptation by increasing sarcomere numbers (Fig. 3B). In amphibian muscles, within about 6 h of the injury from eccentric contractions, the initial shift in optimum length has reversed and the optimum has returned near to its pre-exercise value (Jones et al. 1997). Here we assume that following the eccentric exercise, in some muscle fibres disrupted sarcomeres will have managed, over time, to re-establish their normal pattern of myofilament inter-digitation and ability to generate tension (Talbot & Morgan, 1996). In other fibres, areas of disruption may have progressed to a more major lesion. Such damaged fibres will no longer contract and therefore cannot contribute to the shift in the length–tension curve. Both of these factors will lead to a reversal of the shift.

In a study of human triceps surae, it was possible to identify a damage-related shift in optimum angle for torque in the direction of longer muscle lengths after a period of eccentric exercise, but this had reversed back to control values by 2 days post-exercise (Jones et al. 1997). No subsequent shift due to adaptation could be detected.

In a subsequent study of the hamstrings muscle group, a sustained shift in optimum angle was able to be demonstrated (Brockett et al. 2001a). Here, while a training effect was clearly evident, it was not possible to identify its point of onset, nor any reversal from a shift attributable to injury. Presumably in situations like this, the time course of recovery from the initial shift and the onset of the adaptation process may overlap.

In animal experiments it has been shown that fibres of vastus intermedius muscles of rats, trained to run downhill for a week, had more sarcomeres than fibres from an uphill trained group. At the end of training, in response to an acute series of eccentric contractions, beginning from the same knee angle, the downhill-trained group showed a smaller shift in optimum angle for torque than the uphill-trained group. The smaller shift was considered an indication of less damage (Lynn et al. 1998).

There is also limited evidence of an adaptation process in the opposite direction (Whitehead et al. 1998). A group of human subjects was required to carry out a period of concentric exercise with triceps surae of one leg, the other leg acting as a control. In response to a test period of eccentric exercise a week later, the concentrically trained muscles showed a larger shift in optimum, indicative of more damage, than the control muscles. It was suggested that during concentric exercise muscle fibres may lose sarcomeres, leading to a greater vulnerability to damage from eccentric exercise.

**Muscle sense organs**

While quite a lot is known about the effects of fatigue from exercise on local reflex action of muscle afferents (for a review, see Gandevia, 2001), the question of whether eccentric exercise leads to damage in muscle receptors remains an open one. Here the discussion will be restricted to the two prominent muscle receptors, the muscle spindle and tendon organ.

In an elbow position matching task, carried out after a period of eccentric exercise of elbow flexors of one arm, it was found that the exercised arm consistently adopted a more flexed position in matching the position of the unexercised arm (Saxton et al. 1995). Given that the main proprioceptors signalling limb position are the muscle spindles (Gandevia, 1996; Proske et al. 2000), the result suggests that the signal from muscle spindles had increased as a result of the exercise. That is, to get a set level of proprioceptive signal, the exercised muscle had to be stretched less than the control muscle. The current view is that the level of resting activity from muscle spindles signals the length of the muscle and, so, the position of the elbow. If as a result of the exercise some intrafusal fibres of spindles had become damaged and developed an injury contracture, this would be expected to raise the level of resting activity for a given muscle length and so explain the above results.
A second experiment by Brockett et al. (1997) employed a rather milder exercise regime and elbow flexors of one arm underwent eccentric contractions, while flexors of the other arm underwent concentric contractions, over the same period. Here there was only a small, transient drop in tension, post-exercise, suggesting minimal damage to the muscle, but over the next 48 h subjects matched the position of the concentrically exercised arm by placing the eccentrically exercised arm in a more extended position. This result was therefore the opposite of that of Saxton et al. (1995). The explanation probably relates to the difference in severity of the exercises. Mild eccentric exercise might be expected to produce some sarcomere disruption, leading to an increase in series compliance. That, in turn, would increase the length threshold for tension (Whitehead et al. 2001) and the muscle would have to be stretched further to achieve a set level of spindle discharge. If the spindles had actually become damaged the opposite result would have been obtained. These kinds of propositions should now be tested on single, identified receptors in animal experiments.

Some observations have also been made on tendon organs after a period of eccentric exercise (Gregory et al. 2001). In anaesthetised cats, the medial gastrocnemius muscle was subjected to a series of lengthening contractions and the responses of tendon organs measured to both passive and active tension changes before and after the exercise. It was found that in response to a slow stretch after the exercise, tendon organs commenced firing at a shorter muscle length. This was attributed to the rise in whole-muscle passive tension, post-exercise. In one animal, all of the sample of six tendon organs isolated signalled this increase, suggesting that the damage producing the rise in passive tension was widespread throughout the muscle. However tension threshold and tendon organ sensitivity had not changed suggesting that the exercise had not disturbed, in any way, the normal functioning of the receptors.

Clinical applications
Since eccentric exercise produces muscle damage, weakness and soreness, it raises the question of whether the mild symptoms we all experience on occasions may, at times, lead to more major injuries. A specific case in point is the hamstring tear (Brockett et al. 2001a). Clinical reports suggest that hamstring injuries occur most often as a result of eccentric contractions (Stanton & Purdham, 1989; Garrett, 1990; Kujala et al. 1997; Sallay et al. 1996). It is possible that in some elite sports such as track and field events, football and rugby, the micro-damage from mild eccentric exercise may, as a result of the demands placed on the muscle by the competitive event, progress to more major tears. If so, then a way to combat the problem would be to subject athletes to a mild eccentric exercise program to effect an adaptation that protected the muscles at risk against further damage. This proposition is currently being put to the test in our laboratory.

Another area where the adaptation effect from eccentric exercise may prove useful is with a condition known as idiopathic toe walking (equinus gait). It is a condition commonly found in children. They adopt a strongly plantar-flexed posture with their foot and walk on the heads of their metatarsals, rather than with a heel-to-toe action. Toe walking is sometimes associated with cerebral palsy but it also presents in the absence of neurological signs, in otherwise normal children. Current standard treatments designed to bring the heel down onto the ground include botulinum toxin injections into triceps surae to relax the muscle, plaster casts applied with the muscle in a dorsiflexed position, and insertion of stiff graphite plates in the shoes of afflicted children. We have recently explored the possibility of effecting an adaption in triceps surae by giving the children a specific programme of eccentric exercise of ankle extensor muscles, using a motorised foot plate. While the work is still ongoing, preliminary data suggest that, in the future, the more invasive treatments may be able to be replaced by exercise regimes (D. L. Morgan, C. Blackburn & P. Percival, unpublished observations).

Finally, Duchenne muscular dystrophy is a degenerative muscle disease related to the lack of a protein associated with the sarcolemma, dystrophin. It is know that dystrophin-deficient mice are particularly vulnerable to the damage from eccentric contractions (Head et al. 1992; Moens et al. 1993). It raises the possibility that degenerative changes in muscles of humans afflicted with the disease may be triggered by eccentric contractions. Strategies aimed at minimising such damage would be to avoid eccentric contractions altogether or alternatively embark on a programme of very mild, non-injurious eccentric exercise, in the hope of effecting an adaptation process in the affected muscles.

Concluding comments
This review has focused the discussion on the initial series of events leading to the damage to muscle from eccentric exercise (Fig. 1). In our view, the body of evidence in support of the sarcomere non-uniformity hypothesis has grown considerably in recent times and therefore a review of its current status is called for. Its appeal is that it is able to account for the length dependence of the amount of damage and for the observed differences in effects from concentric and eccentric exercise. It also helps to explain several other behaviours of skeletal muscle that have hitherto resisted satisfactory explanations (Noble, 1992; Morgan, 1994; Morgan et al. 2000). Another area of attention in the review has been the various signs of injury accompanying the damage. These may, in future, have practical applications. So to determine the extent of damage post-exercise, rather than measuring the tension deficit, which is complicated by metabolic factors, or a
shift in the length–tension relation, which requires a series of measurements of active tension, a simple, non-invasive indicator is provided by the rise in passive tension. The training effect, produced by a period of unaccustomed exercise, is important because it represents a potential means of protecting athletes against muscle injuries. It may also be helpful for other clinical conditions. Its mechanism involves the addition of sarcomeres to regenerating muscle fibres, as shown by animal experiments. Such a mechanism provides further support for the proposal that the primary injury process is dependent on sarcomere length.


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Periodization of Training for Team Sports Athletes

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Summary
Training variation and periodization is widely acknowledged as crucial to optimizing training responses. Applying periodized planning to team sports poses unique challenges due to the variety of training goals, volume of concurrent training and practices, and extended season of competition. Practical suggestions are offered in this article to address these considerations and apply periodization in training design for different phases of physical preparation for team sports athletes.

Introduction
Training variation is increasingly acknowledged as serving a key function in successful training prescriptions (13, 37, 40). Periodization offers a framework for planned and systematic variation of training parameters, in a way that directs physiological adaptations to the training goals required of the sport (6, 14, 28, 29, 34, 37). Accordingly, training studies typically find periodized training to elicit improved training responses in comparison to training groups employing a constant load throughout the study period (13, 37, 40). Consensus has thus largely been reached among researchers and practitioners that periodized training offers superior development of strength, power, body composition, and other performance variables (13, 14, 35–37, 39, 40).

The original theoretical basis of periodization was the general adaptation syndrome (GAS) proposed by Hans Seyle (30), which described the generic response of an organism to a stressor (39). According to this model, the first phase of response to any stressor is characterized as shock or alarm (6, 39). Following this is a supercompensation phase, whereby the body adapts to increase the specific capabilities affected by the particular stressor (6, 39). Over time if the stressor continues the organism may enter the terminal phase, termed maladaptation or exhaustion (6, 39). Periodization was developed with the aim of manipulating these adaptation effects, and avoiding the maladaptation phase, which could place the athlete in an overtrained state (6, 34, 39).

This GAS paradigm has since been refined and effectively superseded by the fitness fatigue model (7, 28). A key distinction is that the fitness fatigue model differentiates between the actions of a given stressor on individual neuromuscular and metabolic systems (7). The other major advancement is that the model describes a dual adaptive response resulting in both fitness and fatigue aftereffects, as opposed to the single common response described by GAS. These fitness and fatigue responses essentially work in opposition and are described as having defined characteristics, with distinct differences in both magnitude and duration (7). It is the net effect of these 2 opposing outcomes that determine the state of the athlete’s neuromuscular and metabolic system at a given time (7).

Accordingly, strength and conditioning coaches working in a multitude of team sports at various levels report adopting a periodized approach to their program design. The use of periodization was indicated by the vast majority of Division 1
collegiate strength and conditioning coaches responding to a survey of their methods (9). Similar surveys of professional North American team sports reported comparable use of periodized training design (10–12, 32). These included the National Basketball Association (90% of respondents using periodization) (32), National Hockey League (91.3% using periodization) (11), and Major League Baseball (83.4%) (12). National Football League coaches reported by far the lowest use of periodization models (69%) (10). This may be a result of the contact nature of the sport; on the issue of training periodization, one coach was quoted as saying, “Weight training in football is different than any other sport. When you have them healthy, (you) train them” (10). The fact that the data collection for this study was notably earlier (1997–1998) than the other respective surveys may also have played a part. This may in turn explain the relatively greater use of high-intensity training methods (19%) by the coaches in the sample (10), as such methods were enjoying relative popularity at the time.

Such difficulties as injuries and residual fatigue underline the unique challenges of designing periodized programs for team sports athletes. Providing appropriate metabolic conditioning and physical preparation within the time allowed while athletes are concurrently required to perform high volumes of technical/tactical training, team practices, and competitive matches requires considerable planning and skill (16). Some of the considerations facing the strength and conditioning coach when carrying out this task are discussed below.

**Challenges for Incorporating Periodization into Team Sports Athletes’ Training Extended Season of Competition**

A major obstacle for coaches working in seasonal team sports is the frequent matches and extended competition period. The classical periodization models for planned training variation were developed for the competitive season in athletics (28). As a result, the classical periodized format features extended training cycles designed to progressively prepare the athlete for 1 or 2 major championships in the year (39).

The playing season for sports like football and rugby union can span in excess of 35 weeks, particularly in Europe. If coaches were to follow the classic model, training would taper considerably for the duration of the competition phase. This is clearly counterproductive for most team sports (2, 20). It has been shown that following such restrictive competition-phase repetition schemes may lead to excessive losses in lean body mass during the season, which is unfavorable for most power sports (1). Given this requirement to continue regular training over many months, achieving the necessary training variation represents a sizeable challenge.

**Multiple Training Goals**

Team sports require several disparate training goals. These may include hypertrophy, maximum strength, explosive power, metabolic conditioning, and injury prevention (16). All of these elements must be addressed in the course of the training plan. Therefore there is a need for planned variations in the training program to systematically shift the emphasis to promote these different training effects at different phases of the preparation period.

**Interaction of Strength Training and Conditioning**

When programming strength training, coaches must also take account of the interaction of the metabolic conditioning that is performed alongside strength and power training (16). The physical activity involved in technical and tactical sessions and team practices should also not be overlooked.

A major consideration when strength training and metabolic conditioning are undertaken concurrently is that a preceding bout of high-intensity endurance exercise is shown to impair the ability to perform strength training (23, 24). After conditioning, players are not able to complete the same number of repetitions with a specified load that they are capable of without having performed prior endurance exercise (24).

These interference effects are associated with conflicting hormonal responses to strength versus endurance training (23). Over time, when strength training is performed in the same day following endurance training, power development in particular is shown to be impaired (23). It appears that exercises requiring greater neuromuscular control and coordination may be more susceptible to these interference effects.

**Time Constraints Imposed by Concurrent Technical and Tactical Training**

Given the time constraints imposed by the high volumes of team practices and other skill training common to all professional team sports, the time efficiency of physical preparation is paramount. As the playing season approaches, focus inevitably shifts to tactical aspects, with a greater number of team practices to prepare for the forthcoming fixtures (19, 39). During the season, the need to maximize the effectiveness of whatever training time is allowed is greater still.

**Impact of Physical Stresses from Games**

Allowances will inevitably need to be made for players’ recovery following each match. Particularly in the days following games, this need to allow the players’ bodies to recover is likely to limit the intensity and volume of physical training they are able to perform. It has been identified that losses in muscle mass may occur during the playing season due to the high volume of physically demanding practice sessions and competitive games (1).

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In the case of contact sports in particular, consideration must also be given to the physical stresses associated with both practices and matches that result from violent bodily contact with opponents and the playing surface. Indeed, there has previously been some argument whether it is possible to maintain strength and power levels during the competitive season in collision sports such as rugby football (20). Some authors have suggested that the consequent muscle tissue damage incurred may compromise the amount and intensity of strength training players are able to perform, to the extent that strength and power levels may be diminished over time (20).

The complications outlined often represent an obstacle to effective application of periodized training in many team sports. Consequently, it is probable that many teams are not enjoying the full benefits offered by periodization strategies. Each of these issues must be managed for periodized training to be effectively implemented.

Despite these considerations, training variation remains vitally important for team sports athletes. This is important to alleviate the monotony that can otherwise affect compliance throughout a long season of training and competition (39). Taking player motivation aside, a key consideration is that it is counterproductive to train in the same way for extended periods. Short-term training studies consistently show training programs that incorporate periodized training variation elicit superior results (37, 40). Continued exposure to the same training fails to elicit further adaptation, and in time may lead to diminished performance (39). It is therefore vital to vary the training stimulus at regular intervals to prevent plateaus in training responses. Solutions to these complications must therefore be sought to enable periodized training to be incorporated into athlete’s physical preparation in a given sport.

**Solutions for Incorporating Periodization in Team Sports Training**

**Extended Competitive Season**

It is vital that strength training is maintained in-season to prevent significant losses in strength, power and lean body mass (1, 2). Periodization schemes for in-season training will necessarily differ to those applied during off-season and preseason training cycles. This will be discussed in greater detail in a later section in this article.

**Multiple Training Goals**

Due to the need to maintain different neuromuscular and metabolic training goals as well as cater for technical and tactical practice, some periodization strategies may not be appropriate for a given sport. Emphasizing a particular training goal for an extended preparatory cycle to the exclusion of other aspects of performance will tend to be impractical when training team sports players.

An example of a periodization scheme that appears less suited to the multiple training goals associated with many team sports is the conjugate sequence system (28). This is an advanced approach that aims to exploit fitness and fatigue aftereffects by consecutive overload cycles, alternately stressing one motor quality (e.g., strength) for a period then switching to overload another motor quality (e.g., speed) for the subsequent training cycle (28). Two main training goals are hence typically coupled in this approach. During the overload phase for the other motor quality in the couple (strength), a low-volume maintenance program is undertaken for the motor quality not being emphasized (speed) (28). These cycles are repeated consecutively. The fact that many team sports require a greater number of training goals than the 2 motor qualities typically addressed in this periodization format would obviously make the application of this approach difficult.

**Interaction of Strength Training and Conditioning**

The sequencing of the training day appears key to minimizing the degree to which strength and power development are compromised by concurrent metabolic conditioning work (24). When strength training is prioritized and performed before conditioning, these interference effects can be reduced (24).

This approach is shown to optimize strength training responses of professional rugby league players, to the extent that strength and power measures can be maintained during the course of a lengthy (29-week) in-season period (3). Younger (college-aged) players can even increase strength and power scores during the playing season when adopting this approach (3).

**Time Constraints Imposed by Concurrent Tactical Training**

The issue of limited training time may be addressed by optimizing the time efficiency of concurrent training. A useful strategy is to incorporate different approaches to combine practice and physiological training effects in a single session. Particularly in-season, speed development and agility work can be included in team practice sessions (39). Similarly, plyometric work can be incorporated into strength training sessions late in preseason and in-season, for instance, by combining them in the form of complex training.

A good example of combined physiological and technical training involves the use of game-related methods for metabolic conditioning (17). The skill element involved encourages coaches to continue metabolic conditioning via the use of game-related conditioning methods when the training emphasis shifts to skills practice and game strategy (15). Continuing metabolic conditioning in this form during the playing season is likely to allow cardiorespiratory endurance to be better maintained in-season.

The tactical metabolic training (TMT) format allows conditioning drills to be modeled upon competition demands for
the given sport (17, 27). In this way, structured plays can be used according to the work : rest ratios observed from competitive games (27). Therefore, technical and tactical practice drills can be used as a means for metabolic conditioning (17).

An alternative approach is to use skill-based conditioning games, featuring modified rules based upon the playing area and technical and tactical demands of the sport (15, 17). Indeed, these have been suggested to offer the most effective and time-efficient means for metabolic conditioning in many sports (21). Skill-based conditioning games likewise offer the benefit of concurrent development of decision making and communication under competitive conditions and when fatigued (15, 17).

**Impact of Physical Stresses from Games**

Scheduling of training in-season and during late preseason when competitive matches are being played should take appropriate measures to tailor training sessions to the physical status of that player. In the day(s) immediately following the game, strength training will necessarily be limited to light recovery workouts, implemented alongside acute recovery practices. Similarly, the strength and conditioning coach should be prepared to modify the workout scheduled for a given day in the event the player reports to training with a diagnosed acute injury that will preclude them from performing certain exercises on that day.

**Approach to Periodization**

It has been suggested that undulating nonlinear periodized approaches are more viable when planning the training year for team sports (14, 39). However, at some phases of the training year, particularly off-season and preseason, approaches similar to the classical periodization model do still have application, which is explored further in the next section. This classical approach has been termed “linear” on the basis that it is characterized by progressive increases in training intensity with simultaneous reductions in training volumes throughout extended preparatory training cycles (39). As has been asserted previously, this is in fact a misnomer (38). By definition any periodization scheme is nonlinear, involving (nonlinear) variation in training parameters both within and between training microcycles.

All intensity prescriptions in the example given in the next section are repetition maximum (RM) values, which is similar to the undulating periodization described by Stone et al. (34). The player is expected to lift the maximum load they can handle with proper form for the assigned number of repetitions on any given training day. The only exceptions to this are ballistic lifts (e.g., jump squat) during power cycles, for which maximal power percentage RM values are used.

The use of RM loads is a departure from the classical approach as described by Stone et al. (34), which typically features light and moderate days of submaximal intensity at percentages of RM values. Some authors assert that the inclusion of lighter percentage RM workouts within the training microcycle is necessary to avoid neural fatigue and potential overtraining (18, 33). While this may be the case for power athletes, due to the differences in their respective training I would argue the same might not hold true of team sports players. This contention is based upon personal experience of implementing a year-long training macrocycle, using RM loads throughout, with elite senior professional rugby union football players without noting any ill effects. Our observations indicate the players experienced gains in strength and power in this period.

Furthermore, meta-analyses of dose–response relationships for competitive athletes indicate that training studies with a mean training intensity of 85% 1RM (=6RM load) are found to be most effective for increasing strength (25). This points to the importance of operating close to their RM loads when training competitive athletes. In support of this is the superior strength (6RM bench press) and power (40-kg bench throw) gains noted with elite junior athletes performing 6RM training, in comparison to a group matched for overall load and volume who performed 3 repetitions at 6RM load for twice the number of sets (8).

The practice of using light (percentage RM) workouts has grown predominantly out of training for power athletes (38), such as weightlifting. Practitioners and researchers with a background in the competitive lifting events have contributed much to the field of strength and conditioning and many programs in use today reflect this (14). Strength and power are the primary goals for power athletes and therefore workouts to develop strength and power form the bulk of their training. In contrast, team sports players must also develop strength-endurance and power-endurance, among other training goals. The frequency of strength/power training for power athletes will also typically far exceed that experienced by team sports players. In terms of exercise selection, variations of the snatch and clean and jerk lifts as well as the squat and deadlift necessarily feature prominently throughout all phases of preparation for power event athletes (26). Again this is not the case for team sports players, who must develop strength and power for a much greater variety of training movements and experience a much broader selection of training exercises as a result (16). Certainly team sports players would rarely be asked to perform Olympic style lifts more than twice a week, which would limit the impact of neural fatigue on lifting technique.

Variation within microcycles does remain crucial for team sports players—particularly at the elite level—from the point of

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view of guarding against overtraining. However, rather than incorporating light workouts, this may be achieved by the use of different RM assignments within the training week. Limiting the number of workouts in the training week for a given body part and appropriate scheduling of whole-body, lower-body, and upper-body workouts may similarly be used to provide the necessary recovery (5). Exercise selection between workouts both within and between microcycles can further serve to vary the training stimulus and thereby avoid neural fatigue.

**Practical Example of a Periodized Training Scheme for a Team Sports Athlete**
The degree of variation in training loads and volumes will depend on the age and experience of the player. Elite players are capable of tolerating higher training stress; hence training intensity and volume remain close to their upper ranges for a large part of the training year (39). Furthermore, elite athletes will tend to require a greater degree of variation to optimize the effectiveness of their training (28, 37).

**Off-season**
For the purposes of clarity, the off-season phase for team sports will be defined as the period prior to the start of structured technical and tactical practices. Whether the strength and conditioning coach actually has the luxury of a supervised off-season period when the players report back after the postseason break tends to depend on the willingness of the coaching staff to delay the start of practices to allow him to do so. As a consequence of the length of the playing season, particularly in European soccer and rugby football leagues, this is not always the case.

Due to the long season of competition, it is vitally important to allow a period of active rest following the end of the playing season (39). Similarly, in view of the length of time players are engaged in supervised training it makes sense to allow players to undertake the early initial part of the off-season training unsupervised and off-site, before calling them back into full-time supervised training. This will undoubtedly have a psychological benefit by limiting the monotony of the training ground environment.

Exercise selection during the off-season will be characterized by general strength training exercises for overall development. It is important from the point of view of training variation that lifts that are considered sport-specific are not used exhaustively throughout the duration of the training year (39). General strength exercises, such as machine exercises and single-joint exercises for upper and lower body, do still have merit, particularly at this phase of the athlete’s preparation (31). Similarly, cross-training methods and recreational sports are applicable to maintain body composition and metabolic conditioning (39). Plyometrics, speed work, and agility training are not performed at this time but are reserved for later training phases. A sample off-season mesocycle is presented in Table 1.

**Preseason**
As alluded to previously, preseason for team sports is a time when technical and tactical practices are concurrently scheduled. As a result, physiological training sessions must be planned in the context of the other training and practices players are required to perform. From this point of view, it is therefore vital that scheduling is carried out in collaboration with the coaching staff.

Within the constraints of concurrent training and practices, scheduling of weekly training will follow fitness fa-
tigue effects, up until the beginning of warm-up matches towards the end of preseason. Specifically, workouts early in the week emphasize complex core lifts while the athletes are fresh, whereas fatiguing workouts are performed at the end of the week to allow the athlete the weekend to recover (7).

As suggested earlier in the article, a modified version of the classic format for training periodization still has application during this phase of training for

<table>
<thead>
<tr>
<th>Table 2: Preseason Mesocycle</th>
</tr>
</thead>
</table>

**HYPERTROPHY 1**

<table>
<thead>
<tr>
<th>Microcycle training parameters</th>
<th>Example whole-body workout (10 RM; 3 Sets)</th>
<th>Lower-body workout (12RM; 4 Sets)</th>
</tr>
</thead>
</table>
| Frequency: 4 per week: 2(1)* upper-body 1(2)* whole-body 1 assistance  
*Week 2 frequency in brackets | Parallel back squat  
Incline dumbbell bench press  
Dumbbell lunge  
Barbell bench row  
Back extension  
Wide-grip dips  
Dumbbell split squat | Front squat  
Single-leg knee extension  
Dumbbell step up  
Single-leg hip extension  
Dumbbell split squat |
| Intensity: 8–12RM (all lifts)  
Volume: 3–5 sets | | |
| Rest:  
Short rest (<60 s) between lifts  
Core work (~2 min) between sets | | |

**STRENGTH 1**

<table>
<thead>
<tr>
<th>Whole-body workout (6RM; 3 Sets)</th>
<th>Upper-body workout (8RM; 4 Sets)</th>
</tr>
</thead>
</table>
| Frequency: 4 per week: 2(1)* upper-body 1(2)* whole-body 1 assistance lift  
*Week 2 frequency | Stop clean  
Bench press  
Push press  
Bent-over barbell row  
Parallel back squat  
Single-leg knee extension | Incline dumbbell bench press  
Wide-grip chins  
Dumbbell shoulder press  
Narrow-grip dips  
One-arm dumbbell row  
Dumbbell bicep curl |
| Intensity: 6–8RM multijoint lifts  
8RM assistance lifts  
Volume: 3–5 sets | | |
| Rest:  
Multijoint lifts 2–3 min  
Assistance lifts 60 s | | |

**HYPERTROPHY 2**

<table>
<thead>
<tr>
<th>Whole-body workout (7RM; 3 Sets)</th>
<th>Upper-body workout (10RM; 5 Sets)</th>
</tr>
</thead>
</table>
| Frequency: 5 per week: 2 whole-body 2 upper-body 1 assistance | Snatch pull  
Wide-grip dips  
Front squat  
Wide-grip chins  
Dumbbell split squat  
Dumbbell shoulder press  
Romanian deadlift | Bench press  
Cable fly  
Barbell bench row  
One-arm dumbbell row  
Dumbbell shoulder press  
Dumbbell upright row |
| Intensity: 7–10RM (all lifts)  
Volume: 3–5 sets | | |
| Rest:  
Short rest (<60 s) between lifts | | |

Note: RM = repetition maximum.
team sports. According to this classic model, exercise selection features a progression from general strength exercises to sport-specific lifts as the player advances through preseason training cycles (39).

Another suggested amendment to the classical model during the preseason preparation phase for team sports athletes is to shorten the duration of the respective cycles to a maximum of 3 weeks (19). In turn, these shorter mesocycles can be rotated in sequence (in the traditional order), culminating in a peaking cycle prior to the start of the playing season (19). The relative emphasis in terms of length and number of each mesocycle will be determined by the requirements of the sport (19). For example, in a sport that is reliant on lean body mass, the hypertrophy cycles will be relatively longer and will feature more prominently. Conversely, sports in which excessive hypertrophy is counterproductive will similarly favor strength and particularly power cycles.

A further suggested manipulation is to incorporate day-to-day variation, by varying prescribed RM loads during each respective training week. This allows variation on multiple levels—both within and between microcycles, which has been suggested to favor optimal training responses (37).

The volume of plyometrics, speed, and agility work will vary depending on the training cycle. These training modes will feature most prominently in strength-
and power-oriented cycles as the preseason progresses. The format of metabolic conditioning may consist of a combination of TMT drills and skill-based conditioning games early in the preseason (17). The emphasis will shift to a greater emphasis on conditioning games later in the preseason as training time is more restricted and there is a greater demand for technical and tactical practices. An example of a preseason mesocycle with sample workouts from each cycle is presented in Table 2.

**In-season**

Undulating nonlinear periodization models are typically suggested for in-season training (39). The rationale for this is that these methods may be better suited to maintain the athlete close to their peak throughout an extended season of regular competitions.

It has been identified that average training intensity should be maintained above 80% 1RM in order to maintain strength levels during the course of a playing season (20). High loads (≥80% 1RM, or ≥8RM) are implemented 2 days per week for multijoint lifts. This loading scheme is shown to maintain, or even increase, strength levels throughout the playing season in American football (20).

Similarly, a training frequency of 2 days per week is often recommended for training during the competitive phase (39). In accordance with this, the majority of strength and conditioning coaches in professional leagues typically report strength training twice per week in-season (10–12, 32). However, these recommendations for in-season training need not be excessively restrictive. A range of training frequencies and training parameters are possible that will maintain average training frequency and intensity within the ranges recommended. Players may train between 1 and 3 times per week at various times in the season. Likewise, a variety of intensity prescriptions may be used at different phases, while still maintain-

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**Table 3a**

Four-week (3:1) In-season Summated Microcycle

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 3</th>
<th>Week 4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>8RM; 3 Sets</td>
<td>7RM; 3 Sets</td>
<td>6RM; 4 Sets</td>
<td>4RM; 3 Sets</td>
</tr>
<tr>
<td>1</td>
<td>Wide-grip chins</td>
<td>DB front raise</td>
<td>Parallel back squat</td>
<td>Clean + press</td>
</tr>
<tr>
<td></td>
<td>DB triceps</td>
<td>DB hammer curls</td>
<td>Incline DB bench</td>
<td>Bench press</td>
</tr>
<tr>
<td></td>
<td>DB lateral raise</td>
<td>DB shoulder press</td>
<td>One-arm DB row</td>
<td>Parallel back squat</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Knee extension</td>
<td></td>
<td>B/over barbell row</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>DB step up</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Stop snatch</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Bench skips</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Ballistic push-up</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Narrow-grip chins</td>
</tr>
</tbody>
</table>

**Table 3b**

Three-week (2:1) In-season Summated Microcycle

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Week 1</th>
<th>Week 2</th>
<th>Week 3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7RM; 3 Sets</td>
<td>6RM; 4 Sets</td>
<td>4RM; 3 Sets</td>
</tr>
<tr>
<td>1</td>
<td>Front squat</td>
<td>Power clean</td>
<td>Snatch</td>
</tr>
<tr>
<td></td>
<td>Bench press</td>
<td>Alternate box hops</td>
<td>Resisted knee drive</td>
</tr>
<tr>
<td></td>
<td>Wide-grip chins</td>
<td>Parallel back squat</td>
<td>Ballistic push-up</td>
</tr>
<tr>
<td></td>
<td>DB shoulder press</td>
<td>Bench press</td>
<td>B/over DB raise</td>
</tr>
<tr>
<td></td>
<td>Narrow-grip dips</td>
<td>One-arm DB row</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>6RM; 2 Sets</td>
<td>5RM; 3 Sets</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Clean + split jerk</td>
<td>Jump squat</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cable cross-over</td>
<td>Push press</td>
<td></td>
</tr>
<tr>
<td></td>
<td>DB upright row</td>
<td>Incline DB bench</td>
<td></td>
</tr>
<tr>
<td></td>
<td>DB Bicep curl</td>
<td>Narrow-grip chins</td>
<td></td>
</tr>
</tbody>
</table>

**Table 3c**

Two-week (1:1) In-Season Summated Microcycle

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Week 1</th>
<th>Week 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>6RM; 4 Sets</td>
<td>4RM; 3 Sets</td>
</tr>
<tr>
<td>1</td>
<td>Snatch pull</td>
<td>Jump squat</td>
</tr>
<tr>
<td></td>
<td>Bench press</td>
<td>Single-leg drop jump</td>
</tr>
<tr>
<td></td>
<td>Stop clean + press</td>
<td>Incline DB bench</td>
</tr>
<tr>
<td></td>
<td>Parallel back squat</td>
<td>Wide-grip chins</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>5RM; 3 Sets</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Power clean</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Alternate box hops</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Wide-grip dips</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Narrow-grip chins</td>
<td></td>
</tr>
</tbody>
</table>

Note: DB = dumbbell; B/over = bent-over; RM=repetition maximum.
ing average training intensity above 80% 1RM.

It has been shown that low volume/high intensity in-season programs may not be sufficient to maintain lean body mass in power sports athletes, specifically American football players (1). A novel approach suggested for such team sports players that are reliant on strength and power involves multiple mini-microcycles. This method comprises hypertrophy, strength, power, and peaking cycles of short duration (2 weeks) performed in series (1). This (8-week) series can be repeated throughout the length of the playing season. Hence, this approach is essentially a condensed version of the traditional classical periodization format.

The “summated microcycles” approach would also appear to offer a framework for in-season periodized training (28). Variations of this approach have been successfully applied in both rugby union and rugby league (2). This format involves a steplike increase in volume load (the product of loads lifted and repetitions completed) followed by a pronounced taper. Classically, the summated microcycles format operates around a 4-week cycle, with the final week of the 4-week cycle acting as an unloading week, featuring a pronounced taper in volume load. This is designed to accommodate the time course of the physiological processes underlying training adaptations and fatigue effects (28). These cycles are repeated in series at greater or lesser relative intensities. This basic pattern can be repeated over an extended period, to create a wavelike pattern in lifting intensity and training volume.

It is proposed that the summated microcycles approach may be altered in order to tailor respective microcycles to the fixture list. Specifically, the length of each summated microcycle can be modified according to the competitive games in the period. Important matches and games against particularly strong opponents represent natural times to taper training in-season; likewise, periods with many games concentrated into a short space of time will obviously require reduced training frequency. In both these instances there will necessarily be the unloading week at the end of the summated microcycle, in order to allow players to enter these matches in peak condition. Hence, depending on the timing of these games the summated microcycle may range from 2 to 4 weeks in length, always concluding with an unloading week. Therefore, microcycles may feature a 1:1, 2:1, or 3:1 ratio of loading to unloading weeks.

Table 4
In-Season Overload Week

<table>
<thead>
<tr>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>8RM; 3 Sets</td>
<td>7RM; 4 Sets</td>
<td>7RM; 3 Sets</td>
<td>6RM; 3 Sets</td>
</tr>
<tr>
<td>Narrow-grip chins</td>
<td>Front squat</td>
<td>Ballistic push-up</td>
<td>Clean + split jerk</td>
</tr>
<tr>
<td>Dumbbell shoulder rotation</td>
<td>Bench press</td>
<td>Dumbbell shoulder press</td>
<td>Bench skips</td>
</tr>
<tr>
<td>EZ bar triceps</td>
<td>One-leg hip extension</td>
<td>Wide-grip chins</td>
<td>Incline Dumbbell bench</td>
</tr>
<tr>
<td>Dumbbell hammer curl</td>
<td>Bent-over barbell row</td>
<td>Narrow-grip dips</td>
<td>One-arm Dumbbell row</td>
</tr>
<tr>
<td>Dumbbell upright row</td>
<td>Knee extension</td>
<td>Dumbbell upright row</td>
<td></td>
</tr>
<tr>
<td>Narrow-grip dips</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: RM = repetition maximum.

Conversely there will generally be opportunities during the season for more intensive strength training, such as during fixtures in lesser competitions, or midseason breaks for international matches. In sports with an extended competitive season, such as is seen in European soccer and rugby leagues, this may be necessary to maintain physiological adaptations. This will tend to be the case particularly in collision sports that are reliant upon high levels of lean body mass, strength, and power. At appropriate times midseason, mini (1-week) overload microcycles may therefore be implemented. Table 4 features an overload week to be used for shock elevations in volume-load mid-season that may be necessary for team sports that fulfill the relevant criteria.

Weekly scheduling of workouts in-season is dictated by the dual need to allow the player to recover from the previous
match and avoid excessive residual fatigue at the end of the week in preparation for the next game. This is true of all the variations of the in-season microcycles (see Tables 3a–3c).

During the season, plyometric training will predominantly be integrated into strength training sessions by employing complex training methods. Team practices as well as matches will also provide a plyometric training element (19). Dedicated speed training sessions may be performed on alternate weeks in-season. Agility training can form part of these dedicated biweekly speed sessions. Specific agility work may also be incorporated into warm-up for practice sessions (39). For reasons of time efficiency, metabolic conditioning in-season will likely be almost exclusively in the form of skill-based conditioning games (17).

**Conclusions**

By definition, periodization concerns variation of training. As a result, it seems unlikely that a single optimal periodized training scheme exists that will elicit superior improvements when applied for extended periods. Rather, it seems probable that a range of periodization strategies implemented in combination will produce the best results throughout a long-term training cycle (28).

Some evidence for the benefit of combined use of different periodization approaches was observed in the superior strength gains during initial stages of training in strength trained subjects with a daily undulating periodized (DUP) model, in comparison to a linear periodization group (29). This was attributed to the novelty of the DUP scheme for the subjects, whose previous training had been characterized by classical linear periodization (29). Equally the attenuated training response and reports of training strain in the latter part of the study period in the DUP group indicates that the continued use of this scheme may similarly be counterproductive (29).

This is not to say that the DUP approach in the above example can be concluded to be superior. What the findings of Rhea et al. (29) illustrate is merely that continuing reliance upon a single periodization method may produce attenuated training responses. This is particularly likely to be the case for elite players, who will have far greater training history than the subjects in the study by Rhea et al. (29).

Hence, the best approach would appear to be to strategically combine periodization methods. Periods in the off-season and pre-season without competitive games will undoubtedly allow different approaches to periodized training to those that will be conducive for adequate recovery when matches are scheduled. Periodization schemes implemented in each training mesocycle throughout the training year should be selected based upon what best fits the needs of the respective phase of training (28).

The degree of training variation required appears to be specific to the training experience of the individual (28). More basic periodization schemes are sufficient for younger players, who do not require or benefit from the same multi-layered variation employed with senior athletes (4, 22, 28).

**References**

16. Gamble, P. Physical preparation of


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**Gamble**

Paul Gamble is Strength and Conditioning Coach and Sports Scientist at Heriot Watt University in Edinburgh, United Kingdom.
<table>
<thead>
<tr>
<th>MONTH</th>
<th>WEEK STARTING</th>
<th>PHYSIOLOGICAL GOALS</th>
<th>MICROCYCLE PHASE</th>
<th>VOLUME &amp; INTENSITY</th>
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**PHYSIOLOGICAL GOALS**

- Muscular Metabolic Conditioning

**SESSIONS PER WEEK**

- Training Group Splits

**PERIODIZATION MODEL**

- Linear

**REP RANGES (Core Lifts)**

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**Olympic/Platform Lift**

- Core Lower
- S/L Lower
- Posterior Chain
- LB Functional
- FSC Plyo
- SSC Plyo
- UB Hor Push
- UB Hor Pull
- UB Vert Push
- UB Vert Pull
- UB Assistant
- UB Functional

**Shoulder Complex**

- FSC Plyo
- SSC Plyo

**Positional (Forwards, Halves/Utilities, Outside Backs)**

**Morphological needs (Strength/Power or Hypertrophy)**

- Linear
- 1-2 per week 4 x week (1 x LB, 1 x UB, 2 x TB)- LB & TB after field session 2-4 x week (TB sessions + 1 x UB session)- individual basis

**VOLUME & INTENSITY**

- NOVEMBER  DECEMBER  JANUARY  FEBRUARY  MARCH
- Giant Sets- 4 exercises per station, 3 sets at each station x 4 stations

- Metabolic Conditioning Circuit Incorporating Body Weight and Weighted Exercises

All players will be given individualized strength programs to complete over the break based on their weaknesses and areas in need of improvement.
# 2 WEEK GENERIC HYPERTROPHY PROGRAM

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**Superset**

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### 2 WEEK GENERIC HYPERTROPHY PROGRAM

**INTERMEDIATE:**

**Time under Tension**

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## RUGBY UNION
### STRENGTH PERIODISATION MODEL

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### GAMES
- **Core Sets & Reps**
  - **Hypertrophy**
    - 2 X UB, 1 X LB, 1 X POWER/RFD, 1 X Functional
  - **Strength/Hypertrophy & Speed Power**
    - 1 X UB, 1 X LB, 1 X POWER/RFD
  - **Hypertrophy Power/Focus**
    - 1 X UB (Recovery), 1 X TB Strength/Power, 1 X Power/RFD

### TRAINING PHASE
- **SPP 1**
- **SPP 2**
- **IS 1**
- **IS 2**
- **IS 3**
- **IS 4**

### TRAINING FOCUS
- **Max Strength/Hypertrophy & Speed Power**
- **Strength/Hypertrophy & Speed Power**
- **Hypertrophy Strength/Maintenance/Speed Power/Focus**
- **Power Focus**
- **Hypertrophy/Power**

### TRAINING SPLITS
- **Core Sets & Reps**
  - **Hypertrophy**
    - 5 X 8 @ 85.5%
    - 5 X 5 @ 85.5%
    - 5 X 5 @ 85.5%
    - 5 X 5 @ 85.5%
    - 4 X 10 @ 76.5%
    - 4 X 8 @ 83.5%
    - 3 X 10 @ 74.5%
    - 4 X 10 @ 83.5%
    - 3 X 8 @ 85.5%
    - 3 X 5 @ 85.5%
    - 2 X 8 @ 85.5%
    - 3 X 6 @ 85.5%
    - 2 X 5 @ 85.5%
    - 4 X 6 @ 85.5%
  - **Strength**
    - 5 X 5 @ 85.5%
    - 5 X 5 @ 85.5%
    - 5 X 5 @ 85.5%
    - 4 X 8 @ 83.5%
    - 4 X 5 @ 83.5%
    - 3 X 6 @ 85.5%
    - 3 X 5 @ 85.5%
    - 2 X 8 @ 85.5%
    - 2 X 5 @ 85.5%
    - 4 X 6 @ 85.5%

### HYPERTROPHY PERIODIZATION

### STRENGTH PERIODIZATION