Rate of force development (RFD) and Co-activation

Matthew Van Dyke

Success in non-team sports ultimately comes down to which athlete can produce the most force in the time allowed during their individual competition event, while also being efficient with their movements [1-7]. The goal of a strength and performance coach at any level should be to get athletes to make the greatest performance gains in the shortest amount of time [7]. This is the idea of efficiently applying optimal periodization schemes for athletes.

Individual athletes generate maximal forces based on two elements. The first determining factor is the maximal force capabilities of individual muscles, with the second being the coordination of muscle activity by the central nervous system (CNS) [4,8]. The ability of the individual muscles to produce force is determined by specific training methods, while the force produced by the coordination of muscle activity is a learned activation pathway of the muscles for optimal safety of the organism while maintaining maximal force outputs [3].

The ability to produce force rapidly has been coined the rate of force development (RFD) and is actively sought after by every strength and performance coach. The reasoning for placing importance on improving RFD is due to the limited time available for athletes to deliver force in their competition movements [2]. The time available for force development in athletic movements is much smaller than the time needed for the body to produce maximal force, which takes up to 0.3-0.4 seconds [2,5]. Sprinting is an example of this, the ground contact time in maximal velocity sprinting is typically between 0.08 and 0.12 seconds, which is much less than the 0.3 to 0.4 seconds needed to produce maximal force [2,5]. Pre-activation plays a role in increasing force production, however maximal force still is not achieved. Pre-activation is especially important to optimize the stretch-shortening cycle. Pre-activation "increases the initial muscle stiffness and thereby improves the ability of the viscoelastic tendons to be stretched and recoil," which further improves RFD. In order to increase the force produced during sprinting, the ability to produce force rapidly becomes more important than maximal force production. Sprinting is just one example of a movement of sports; however, the majority of maximal velocity movements in sport have a time of less than 0.25 seconds to produce force. It is for this reason every coach is training with the intent to increase RFD.

Rate of force development is influenced by different training methods. Rate of force development can be trained in a biphasic manner with an early phase lasting less than 0.1 seconds and a late phase with times longer than 0.1 seconds [4,9]. The early phase is influenced primarily by neural drive while the late phase is more dependent upon the muscle cross sectional area and maximal force production capabilities [2,4,9]. Another trainable aspect that leads to improvements in RFD can be found in the viscoelastic properties of the muscle and tendon [2]. The proportion of fast and slow twitch fibers is another variable that affects RFD; however, this tends to be more genetically based and not as trainable [2].

With early and late phases of RFD affected by different processes, it is not surprising that different training methods bring about different adaptations to the two phases. Training programs that place focus on explosive strength, or high velocity movements increases early force development by increasing neural drive [1,2,4,6,9]. It appears that the early phase of force development may also be improved when the intention of training is maximal acceleration, meaning the intended velocity becomes more important than the object velocity [4,5,9,10]. Programs that focus training on high loads lead to improvements in maximal strength and maximal force development. These adaptations are involved in the late phase, as the movements allow enough time for maximal force to be developed during muscle contraction.

Maximal velocity movements in athletic competitions do not allow enough time to apply maximal force with each contraction. This does not mean that maximal force should be overlooked in the training process. Maximal force has a positive relationship with RFD, meaning as maximal force increases, so does RFD [4]. This concept justifies the thought process of athletes needing to improve maximal strength. There comes a point, however, when increasing maximal strength will no longer transfer to improved athletic performance. Once this threshold of maximal strength has been reached, the goal of coaches must be to increase the early phase of RFD. The time period of the year also determines a coach's decision on which phase to train in regards to RFD. As the competition period approaches, focus of training must be shifted to the early phase in order to optimize transfer of training, and ultimately performance.

The second determining factor of force production in athletes is the coordination of the muscle activity, as controlled by the CNS. In order to perform optimally and produce maximal force, precise coordination of the involved muscles is an absolute requirement [11]. The improvement in this coordination of the nervous system is a learned skill through training [3,12]. Training with exercises that utilize the same muscle activation systems as seen in competition will lead to increased coordination of the nervous system and increased transfer of training. When all of these nervous system qualities improve, athletes better able coordinate the activation of fibers in single muscles as well as in muscle groups.
The CNS, consisting of the brain and spinal cord, send signals in accordance to the desired action. The brain sends an action potential, or signal, down the spinal cord and through the peripheral nervous system to the desired motor neuron. The peripheral nervous system has many branches that connect the spinal cord to all parts of the body. In some cases reflexes are used. In these cases the signal originates in the spinal cord. The reflex system is used when rapid reaction is necessary.

A motor unit is composed of a single motor neuron and the muscle fibers it innervates [10]. Different motor units vary greatly in their size, force generating capacities, and their resistance to fatigue. As the number of motor units being utilized during a movement, so does the force production [10]. This is the basis of recruitment. There is a close match between the size of a motor unit and the type of muscle it contracts. For example, fast twitch fibers are innervated by motor neurons capable of firing at faster frequencies, which allows greater contraction velocities and more force production. Motor units also vary in the number of muscle fibers they innervate [10]. Smaller motor units innervate a smaller number of muscle fibers and are generally responsible for completion of more precise and finely graded movements. Larger motor units that innervate hundreds to thousands of muscle fibers are responsible for the execution of large muscle movements that do not require as much precision. An example of a small motor unit is the control of an eye muscle while focusing on an object, while a large motor unit would be the quadriceps during knee extension.

Specific neural adaptations due to training may include recruitment, rate coding, the incidence of discharge doublets, and to some extent synchronization [3,6,7,9,12]. Recruitment of muscle simply implies changing the number of muscle fibers used during a movement. Muscle fibers are recruited in a specific order according to their size. Small slow twitch endurance type fibers are recruited first with the bigger, stronger, or fast-twitch, fibers being recruited as force requirements increase. Excluding sustained submaximal activity, as the number of muscle fibers recruited increases force production also increases.

A second way to increase force production is to increase the rate at which each motor unit is activated. This is known as rate coding [3]. When the firing rate of a motor neuron increases, so does the potential for force output of the muscle. Doublets occur when two twitches are sent to the same motor neuron within 0.05 seconds of each other. Doublets increase RFD significantly and have been shown to increase in occurrence with explosive training methods [1,3]. The activation of the motor units in a more or less coordinated way is also known as synchronization. Synchronization, although shown to have less of an impact than recruitment, rate coding or doublets, can still help increase the efficiency of firing, allowing for maximal force production.

Maximal muscular force is only achieved when a maximal number of fibers are recruited during a contraction. This includes both slow and fast-twitch motor units, when rate coding is optimal to produce a fused tetanus in each motor fiber and all motor units work synchronously over the short period of the maximal voluntary contraction. Therefore, maximal muscular force is determined not only by the quantity of involved muscle fibers, but also by the extent that each fiber is activated [10]. Each of these trainable and necessary neural adaptations as well as specific methods of training are discussed in greater detail in a later section of this thesis.

Antagonist

The production of force in a rapid manner is imperative for success of most athletic performances [1-7]. In order for maximal power to be produced, intramuscular and intermuscular coordination must be at the highest possible levels. Included in the intermuscular coordination is the contraction timing and force produced by the agonist as well as the antagonist. The net torque produced at the joint is ultimately the moment generated by the agonist minus the moment of the antagonist [3,13-21]. Thus, in order for the agonist to shorten with maximal net torque, the antagonist must be allowed to stretch within reason to keep joint integrity.

During any voluntary contraction co-activation, or activation of both agonist and antagonist, occurs to some extent. Co-activation is important for joint stability; but, it also determines the net torque about the joint [3,13-21]. The ability to reduce the level of co-activation during certain phases of the muscle contraction show adaptations within the nervous system and increase desired force output [12,13,14,18,19]. This increased force will show improvements particularly during the early RFD phase which appears vital for optimal athletic performance.

Improving net torque about a joint can be accomplished in two ways; increase the activation of the agonist muscle(s) or decrease activation of the antagonist muscle(s). Increasing force output of the agonist muscle(s) is the most commonly understood method to increase net torque. However, methods of decreasing co-activation of the antagonists can be just as valuable to improve net torque as increasing activation of the agonists.

Strength training, when programmed correctly, has the ability to reduce the interfering effect of co-contraction between the agonist and antagonist muscles in rapid movements such as those seen in athletic competitions [14,16-19]. Proper training leads to a more efficient control of what is commonly referred to as the "ABC" pattern seen in musculature during dynamic movements [15,16,17]. The ABC pattern consists of three contraction phases. The three steps in this pattern include a large burst by the agonist muscles early in the movement phase (A), followed by a short braking burst from the antagonist muscles (B), and then a final push by the agonist muscles to complete the movement (C). As the speed of a contraction increases, so does the amount of braking force applied by the antagonists. With this in mind, the thought of decreasing the co-contraction of antagonist muscles to increase net torque becomes a relevant idea as a shorter, more succinct braking phase would mean that agonist muscle action could contribute a larger portion of the total contraction time.

The efficient coordination of the agonist and the antagonist is one of the valuable early adaptations in resistance training responsible for increases in torque production (16,20). Adaptations to co-activation appear related to skill development as highly trained athletes show different magnitudes and/or patterns of co-activation during maximal contractions or other given motor tasks when compared to novice or non-athletes (12,13,14,18,19).

Weak antagonist muscles may also limit the velocity at which contractions are completed and strengthening of antagonists often lead to increases in agonist muscle movement as well (16,24). Antagonist movements in training directly following agonist movement training led to an increase in power to a greater extent than the group training with just agonist exercises (10).This displays the importance of training both agonist and antagonist muscles, even if a sport action is considered to be dominant in just one of those muscle groups.

Transfer of Training

Another factor in force production in athletics is the transfer of training from the weight room to the competitive event. A strength and performance coach's goal must be to improve performance in the specific athletic event, not just make an athlete stronger. Increased transfer of training consists of several key factors that include increasing RFD and optimizing muscular activation patterns specific to sport.

In regards to skill learning, the expression of voluntary strength is linked to a skilled act. The primary agonist muscles used must be fully activated along with proper activation of synergistic and antagonistic muscles (14,18,19). When subjects are introduced to a new and relatively complicated strength task, excessive co-contraction of antagonist muscles may limit their ability to fully activate the agonists, leading to a decrease in net torque produced (19). Practice and training in a specific movement pattern may reduce the amount of this co-contraction, allowing for optimal torque production about the joint(s).

Improvement in athletic performance is determined by an increase in the coordination of all muscles involved in a movement and not solely on the increase of strength of the individual muscle. It is unlikely that training improvements in one movement will correlate to other movements, even within the same muscle. An example of this can be seen in the quadriceps muscles. Although quadriceps contraction is common for many movements in athletics such as jumping, cycling, and/or sprinting, the sequence of muscle activation for each of these movements differ so that a set of neural connections established as a result of quadriceps training is unlikely to help with multiple movement patterns. For this reason, it is vital to train athletes in patterns similar to the movements seen in competition. This is important for both force production and improvement in activation patterns of muscles seen in their respective event. Another method that improves transfer of training of an exercise is attempting to mimic the velocity of movements seen in competition. This is difficult as very few movements if any completed in a weight room can match the speed seen in competitive events (7). An example of attempting to improve transfer of training is comparing a heavy set bench press at 80% of an athlete's 1 repetition max (1RM) compared to a medicine ball chest pass. The medicine ball exercise is much closer to the speed and rate of force development required during an athletic movement, thus it will have a higher transfer of training than the 80% 1RM bench press.

One cannot maximize RFD solely by focusing on a higher transfer of training and ignore maximal strength exercises. Increasing maximal force output also positively correlates with RFD [4]. Training for each parameter, maximal force output and sport specific transfer or training depends on the time of year. Training exercises typically begin with a focus on maximal force output and as the competition event or season approaches, methods of training are shifted to focus on transfer of training to maximize RFD.

The requirement for increasing transfer of training from the weight room to the competitive event must be realized in order to optimize performance. For this reason, it is necessary to train using specific exercises that utilize the similar muscle activation patterns and attempt to mimic the high velocities in sport.

Why/How co-activation occurs

Co-activation occurs to ensure joint stabilization and varies depending on both the joint angle and the forces acting on the joint [14,22-26]. The increased joint stabilization also improves movement accuracy [14,22]. Higher activity of the antagonist muscles is seen at end ranges of joint motion compared to the middle. This further depicts the importance of antagonist muscle co-activation in joint safety. The deactivation of the antagonist muscles during contraction allows for faster contraction of the muscle, leading to higher net torque [12].

During voluntary muscle contraction, the central command descending from the primary motor cortex contributes to the simultaneous activation of the agonist and antagonist muscles. The CNS will compromise maximal force production to ensure stabilization and joint integrity, particularly in situations of uncertainty in the motor task, the type of muscle contraction, when high loads are used, and at varying degrees of a joint angle [14,22-26]. There is also a sharp increase in antagonist co-activation as velocity of the movement increases. This further suggests co-activation provides the counter-torque necessary to slow down rapid movements and is useful for joint stability. Despite the knowledge of these fundamental roles of the antagonist in co-activation, little is known about the brain mechanisms underlying their control.

The origin of muscle co-activation is both supra-spinal and spinal and tends to decrease with resistance training, which allows, under certain circumstances, an increase in the desired torque developed. However, antagonist muscle co-activation also depends on the characteristics of movement. Although research shows these mechanisms are affected by both training and detraining, the origin and contribution of supra-spinal mechanisms to the antagonist muscle co-activation process have yet to be specified [27,28].

Why reducing co-activation is a goal for athletes

Athletic success is often determined by which competitor can produce the most force in the limited time allowed during an athletic movement. Due to the fact that athletic movements do not allow enough time to create maximal force outputs within the muscle, RFD becomes the most important factor in performance [2]. It is important to realize that, not only do agonist muscles produce movement, but also antagonist muscles which, during co-activation, might actually hinder torque production about the joint [12].

Net torque generated about a joint depends on the force developed by the activation of the agonist muscles, minus the force developed by the antagonist muscles. Co-activation of the antagonist may impair the athlete's ability to fully activate the agonist.

The gains in force output seen at the end of training periods have been attributed to many factors including motor unit recruitment, rate coding, and muscle hypertrophy [2,3,12]. Training and strength gains, along with the achievement of a motor skill have been accompanied by a reduction in co-activation of antagonist muscles as well [14,16,17,18]. Elite athletes in general, exhibit a reduction in co-activation compared to sedentary subjects, which supports the notion that training has the ability to reduce co-activation. The achievement of a motor skill is another method used to reduce co-activation as elite tennis players showed a significant reduction in co-activation at all angular velocities during elbow extension over recreational tennis competitors [13,18,19]. Progressive inhibition of muscular activity unnecessary for the achievement of a task has also been shown, further enforcing the achievement of a motor skill associated with reduced co-activation.

Proper training of both agonist and antagonist muscles further enhances the reduction in co-activation. It is possible that strength training causes adaptations within the nervous system that allow the agonists to be optimally activated in specific movements, leading to a better coordination of all muscles associated with movement patterns. Improving the activation and coordination of the agonists leads to either an increase in the amount of force produced in the intended direction of movement or a more efficient movement that conserves energy. The antagonists, when strengthened, will not only improve the joint stability, but are also able to stop the movement in a faster time by applying a better braking force [15,16,17,19]. This increased braking force leaves a greater amount of time within the movement for acceleration, creating higher velocity movements by the agonist muscles. By strengthening the antagonists, the ABC firing pattern can occur at a later time, leading to increased time to produce force in the concentric manner of the completed action.

Ways co-activation could be affected

Improving RFD:

Rate of force development is influenced by many factors. These factors include, but are not limited to the proportion of fast to slow twitch fibers, muscle cross-sectional area, viscoelastic properties of the muscle-tendon, and the efferent neural drive to the muscle fibers [1,2]. Rate of force development has been used to evaluate the capacity of muscles to generate force vital for athletic performance. But, as velocity of a movement increases, the muscle's ability to produce force is diminished. Because of the inverse relationship between force and velocity, sports training have typically focused on strength. Whether attention is placed on maximal strength or explosive strength during different, phases, it will certainly have decidedly different influences on RFD [4,9]. Training with short, high RFD contractions, placing a substantial emphasis on neural drive and explosiveness rather than maximal force, may work to improve the early phase of RFD [1,2,4,5,6]. Explosive muscle strength can be defined as the rate of rise in contractile force at the onset of contraction. This improvement seen with explosive training in the early phase might very well be the most important aspect to maximize in athletics where maximal contractile force is almost never reached [4].

Low resistance, 30-40% of 1 RM, with high velocity dynamic training potentially causes an earlier recruitment of large motor units, increased rate coding, increased discharge of doublets, improvements in synchronization to some extent and also changes in sarcoplasmic reticulum Ca^{2+} kinetics [1,2,3,31]. All of these factors play a vital role in RFD.

Nervous System:

Three common methods to alter motor unit recruitment, rate coding, and synchronization are aimed at improving sports skills. Each motor unit consists of a motor neuron coming from the spinal cord and the muscle fibers it innervates. When a motor neuron is activated, or recruited, impulses are distributed to all fibers within that motor unit. This is known as the all-or-none principle. By recruiting more motor units, an increase in muscular force can be accomplished. Greater force can also be created through changes in motor unit firing rates. This is known as rate coding. Synchronization allows muscles to be activated or inhibited at appropriate times in order to maximize force production through an entire movement [3].

Recruitment:

During any voluntary contraction, the orderly pattern of muscle recruitment is controlled by the size of the motor neurons. The size principle dictates that smaller motor units are recruited first as they have neurons with the lowest firing threshold [3]. As the force required by a task increases, so does the number of activated motor neurons, leading to the larger, more powerful, motor units being recruited. Motor units with the largest motor neurons also have the fastest twitch potentials. They also have the highest threshold and are therefore recruited last.

As the number of recruited fibers increases, so does the force produced by the muscle. In athletics, the ultimate goal is often to produce as much force as possible in a limited amount of time. Thus, improving the body's ability to create high forces by improved recruitment is an important aspect that cannot be overlooked.

Training the recruitment of large, explosive motor units is necessary in order to improve the force production of muscle [3,6]. This adaptation can be reached by using large, or explosive forces, in order to recruit these fast twitch type II hypertrophy based fibers.

The size principle is applicable and relatively fixed for a muscle involved in a specific motion, even if the movement velocity or RFD is altered. The threshold of motor unit recruitment is typically lower during ballistic movements due to the rapid elevation of force needed to complete the task [3]. These two elements are important details to consider in the world of athletics where almost every movement is completed with high velocity and a high rate of force development.

Although currently no techniques exist to definitively establish whether or not training elicits a true increase in motor unit recruitment, experts have argued that specific training affects motor units within a specific muscle movements, leading to a decreased threshold of the large, fast twitch motor units, while no change is seen in other movements. This demonstrates, again, that adaptations due to training are linked to movement patterns and skill development. As the body becomes proficient in a trained movement, the ability to increase force due to recruitment of fibers is improved in that specific movement pattern. This skill learning further demonstrates the importance of utilization of proper exercises and training methods to improve transfer of training.

Rate coding:

The recruitment of the motor units is just one way the nervous system can adapt to create higher force by a muscle. Force is both a function of the number of motor units activated and the amount of force produced by each activated fiber. The nervous system has the ability to increase the frequency of the signal to a motor unit which leads to an increased rate of firing within that same motor unit [(3)]. This is known as rate coding. When multiple signals, or twitches, occur in a muscle fiber, the tension it produces is increased and overall muscle force is potentially greater.

In general, recruitment is more important than rate coding for early recruited motor units and low loads, while rate coding becomes important as force production nears maximal voluntary contraction [(3)]. Rate coding however, is the primary source when rapid intramuscular tension is required to overcome a load. In small muscles, the majority of motor units are recruited at a level of force less than 50% of the maximal force capability of the muscle [(10)]. Once those motor units are recruited, rate coding plays the major role in the further development of force [(3)]. In larger muscles, such as the deltoids and biceps, recruitment of motor units appears to be the primary mechanism for increasing force development up to 80% maximal force output. In the force ranges of 80 and 100% of maximal force output, an increase rate coding is almost exclusively responsible for increasing force output.

The ability to increase rate coding of a motor unit is important in the production of doublets, which are two twitches sent to a motor unit within 0.05 seconds of each other. Doublets result in a significant increase in RFD as tension produced by a doublet is much greater and develops much more rapidly than if the two twitches were produced on their own. Doublets have the highest occurrence when a high RFD is needed or the speed of contraction is high [(3)].

Although researchers have yet to demonstrate motor unit excitability in a definitive way, it has been argued training increases rate coding [(3)]. An increase in firing rate of trained muscles is a possible mechanism of improvement in neuromuscular performance as an increase in force production is seen. Improvements in rate coding enhance the magnitude of force generated, which is done by increasing the amount of the force produced by the recruited fibers. As more force is produced, the ability to improve RFD is also enhanced, which leads to a rapid increase in force development.

Training effects on rate coding have been demonstrated by sprinters that show the highest motor unit firing frequency in rapid dorsiflexion, leading to the idea that rate coding, like recruitment is a learned skill specific to training [(3,)].With the knowledge that athletic success relies heavily on RFD, it should be a goal of strength and performance coaches to increase the number of doublets produced via specific high intensity training. This can potentially be accomplished by using ballistic type exercises prompting adaptations to motor unit firing frequencies.

Synchronization:

Another way the nervous system can increase force production is the synchronization of motor units. Synchronization refers to the activation of each motor unit in a more or less synchronous manner, with more synchronization leading to increased force outputs [(3)]. In most contractions, motor units function asynchronously to produce a smooth, accurate movement. Although recruitment and rate coding are the two major factors in RFD, there is some evidence that in elite power and strength athletes, motor units are activated synchronously during maximal voluntary efforts [(3)].

Motor unit synchronization occurs when two or more units are activated concurrently more frequently than expected for independent random processes, which leads to an augmentation of force and increases RFD [3]. The adaptation of the nervous system leading to increased synchronization assists with co-activation of muscles within a movement, leading to increased intermuscular coordination, and ultimately increased RFD [3,12].

Intermuscular coordination describes the appropriate activation, in both magnitude and timing, of the agonist, synergist, and antagonist muscles during a movement. In order for a movement to be optimally efficient, the agonist activation must be supplemented by increased synergist activity and decreased co-activation of the antagonist muscles. Only when these steps occur, with precise timing and level of activation of the appropriate muscles, will the flow of power through the kinetic chain be optimal [3]. The ability to transfer power through the body in a coordinated fashion, like every other adaptation, is a learned skill through training. The utilization of exercises that have high transfer of training to the competitive event will optimize synchronization, leading to the realization of success in athletics [7].

Motor Learning:

Motor learning is based on the principle of skill development. Skill development, at the most basic level, is the ability of the body to adapt to a stimulus and adjust its response in order to achieve optimal results for that specific task. The accomplishment of any task, especially athletic ones that require high forces and velocities, require proper coordination. This coordination of movement can be improved through the proper training stimulus.

Despite the fact that many muscle systems are characterized by intrinsic strategies, these patterns of activation do not always result in the most effective activation sequence [3,14,20,30]. This is especially important in high velocity actions because they have the potential to lead to inefficient activation sequencing [3,30]. Proper training however can improve this process. After four weeks of resistance training, the level of input to the spinal motor neurons associated with a particular degree of muscle activation or joint torque is lower than before training [30]. This shows that a smaller input is required to achieve the same muscle activation post-training. It seems reasonable to conclude that training programs engage muscles in patterns of activation that are not promoted by intrinsic strategies of control and may actually promote flexibility in subsequent recruitment patterns.

Motor learning may also play a role in the reduction of antagonist muscle co-activation [14]. In novel or complex tasks, co-activation by antagonist muscles is often excessive, but has been shown to decrease with practice [12,14,18,19]. Lower antagonist co-activation is associated with a high skill level or familiarity with a movement, meaning with enough practice it is a learnable skill [12,14,18,30]. Data also suggests that higher scores in strength tests resulting from training programs largely reflect acquisition of skill [10,14,30]. The development of a specific skill, once again, leads back to the importance of training programs having a high transfer of training. Thus, allowing the learned skills, ranging from nervous system adaptations to increased force output, to be transferred from the weight room to competition [3,30].

Myelination:

Myelination of axons is increased with the development of skill [30]. Skill development leading to an increase in myelination is the result of countless hours of practice, which forces the brain to fire the electrical impulses through specific neural circuits to the motor units of the muscles [30]. The increased number of signals continuously sent through a specific group of neural circuits leads to an increase in myelin, which wraps the axon of nerves and acts to speed up conduction velocity [30)].

Myelin is a white, fatty substance that coats axons throughout the brain and human body. Axons act as wires, carrying electrical signals along billions of chains of nerve fibers, relaying messages from the CNS to the peripheral nervous system and then back again [30]. As the amount of myelin increases, the proficiency of the skill correlating with that specific neural circuit is increased as well.

Every task, thought and action that the human body and brain perform is a learned skill or reflex circuit [30]. The basic ideas regarding neural transmission can be broken into three points. The first is that every movement, thought, or feeling is a precisely timed electrical signal traveling through a chain of neurons, otherwise known as a circuit of nerve fibers allocated together to perform a task. Second, myelin is the insulation that wraps many of those nerve fibers and increases the signal strength, speed, and accuracy. Finally, myelination is important for skill development. The more a particular neuronal circuit is fired, the more myelin insulates that circuit. These stronger, faster signals lead to more fluidity throughout an athlete's thoughts and movements [30].

Studies have shown that a physiological adaptation of the brain to learning a new skill is the addition of myelin around the neural circuits responsible for that specific skill [30]. A second study showed that myelination could be inhibited by blocking of specific neural circuits. These two studies show stimulation of neural circuits, such as those observed in thoughts and movements, is a requirement to increase myelination [30].

The increased neural conduction velocity can become significant when RFD is important, as seen in competition. This, again, shows the importance of using exercises and high velocity movements that have a high transfer of training to an athletic event. This high transfer is vital as only the neural circuits trained, will show increased myelination. Increased myelination is important to athletes as it has the ability to lead to increased RFD of the agonist or may lead to less co-activation of antagonist muscles [30]. Increasing myelination due to a specific movement calls for better methods to determine how athletes should train and practice. The next step is to find ways to maximize the amount of neural input each athlete processes while competing in their specific event. If this feat is achieved, coaches would have the ability to speed up the development and learning process, which would ensure athletes reach their full potential [30]. There are many theories considered valid that may be responsible for the increase in myelination as this research is still in its infancy and is highly theoretical [30].

Muscle Contraction:

Skeletal muscle consists of numerous fibers, or muscle cells. Each fiber is made up of many parallel myofibrils, which consist of longitudinally repeated units called sarcomeres. Sarcomeres in turn include thin and thick filaments. Thin filaments consist primarily of actin, while thick filaments are made up of myosin. The actin and myosin filaments partially overlap each other, this becomes important to allow activation to occur. The myosin, or thick, filaments have a small projection called cross-bridges. Cross bridges, once activated attach to the actin filaments and produce tension within the sarcomere.
The force produced by a muscle is the result of activity within the muscle subunits, which include the sarcomeres, myofibrils, and muscle fibers. The amount of tension produced by a sarcomere depends largely on the total number of myosin heads available for cross-bridge links to actin filaments.

The sliding filament theory of muscle contraction describes how the cross-bridge projections attach and detach from actin filaments causing muscles to generate tension. It is also important to understand all steps occurring within the muscle during contraction as each of these steps requires a fixed amount of time. As the velocity of contraction increases, a decrease in the number of cross-bridge attachments is seen. This plays a factor is RFD, which is of paramount importance in athletics.

Although never been shown experimentally, there is speculation that improvements in both speed of occurrence and coordination in the sequence of muscle contraction steps is possible. During a muscle contraction, many fibers undergo the same contraction steps. With the possibility that those steps can be improved, the entire process is primed to create more power and/or produce power more efficiently. The adaptation of each individual step may be extremely small, but any increase in any step may, ultimately, lead to a bigger influence on contraction speed. It is important not to overlook the possible improvement within each of these steps when determining possible reasons for increased RFD (3).

These common steps occur in every muscle contraction within the body.

1. Muscle contraction is initiated by a signal from the alpha motor neuron
2. Action potential releases acetylcholine
3. Acetylcholine binds to motor end plate, releasing sodium, which enters the cell and depolarizes the muscle fiber
4. This depolarization reaches the T-tubules, which release stored calcium from the sarcoplasmic reticulum into intracellular space
5. Calcium binds with troponin-C on the actin filament, moving tropomyosin and exposing actin's binding sites
6. Myosin heads attach to the binding site now exposed on actin filaments
7. Myosin heads tilt, locking the actin filament in place and generating tension in the active muscle fibers
8. Myosin heads detach from actin when ATP binds to globular head
9. ATP is split by ATPase into ADP and an inorganic phosphate, releasing energy
10. Energy release re-cocks the globular head of the myosin filament, priming it for another attachment to actin

Muscle Spindles:

Muscle spindles act as neuromuscular regulators within the body, as the stretch or force on a muscle changes, muscle spindles relay information to the brain via an afferent pathway. If muscles are stretched by an external force, these intrafusal fibers are also subjected to being stretched. Stretching causes an increase in muscle spindle discharge, leading to efferent signals back to the extrafusal fibers of the muscle attempting to return it to its initial length in spite of the load initially applied to the muscle. The muscle spindle is ultimately concerned with controlling changes in muscle length rather than force it might generate.

Forced lengthening of a muscle during the eccentric phase of the stretch-shortening cycle causes a mechanical deformation of the muscle spindles, which activates a reflex mechanism (3). When movements are done quickly, the stretch reflex increases extrafusal muscle stimulation, often resulting in increased contraction force during the concentric phase and ultimately contributes to enhanced maximal power (3). In athletics, the ability to involve muscle spindle activity may have a positive effect on RFD.

Golgi Tendon Organs (GTO):

Golgi tendon organs (GTO) act as neuromuscular inhibitors. These receptors are sensitive to forces developed in the muscle. If muscle tension increases sharply, the GTO reflex causes an inhibition of muscle action. This leads to a decrease in muscle tension in order to prevent the muscle and/or tendon from incurring damage.

Golgi tendon organs are found within most large tendons and provide negative feedback to the CNS when muscle tension reaches beyond a dangerous threshold. When this threshold is reached, efferent signals to the muscle are inhibited reducing activation of the muscles experiencing these high tensions. The ability to decrease the activation of GTO's may lead to an increase in the ability to produce greater force, which might be necessary for optimal sports performance. The majority of GTO's are pre-set to inhibit a muscle up to 40% below what that structure can actually handle. When GTO's inhibit a muscle group from reaching its potential force output, a decrease in performance is seen, particularly in the aspect of explosive power development.

Examples of this "pre-set inhibition" in GTO's are seen in some emergency situations, such as a child being trapped under a car and a grandma lifting the car off of the child. In these extreme cases, muscle damage is often seen, suggesting that the muscular inhibitors were deactivated, or at least overridden during that situation. Such a level of force output should not be achieved in athletic competition; however, if it is possible to reset the limits of the GTO's it could leads to the belief that skill development or specific training methods, may safely alter the inhibition of the GTO's. This inhibition would ultimately lead to an increase in force production as the muscle inhibitors would allow a higher threshold to be reached before shutting down the muscle.

Renshaw Cells:

Renshaw cells are yet another way the body can control the neural output to skeletal muscles. As desired recruitment of muscle fibers and rate coding increases, the signal works through a feedback loop containing Renshaw cells. Renshaw cells play an important role in the skill learning process in regards to the activation of appropriate neurons. In the beginning stages of learning a skill, the body often functions inefficiently in the recruitment of muscle fibers, leading to a surplus of the actual needed fibers being activated. Renshaw cells work to inhibit the firing of unwanted motor units. Through proper skill learning Renshaw cells allow the appropriate motor units to be used which leads to optimal force production with minimal energy utilized. In regards to athletics, just as with the GTO's, an increase in the threshold of the Renshaw cells due to training has the ability to improve force development, especially when it is needed rapidly, as seen in the world of elite athletics. Renshaw cells associated with specific neural pathways have been shown to be dramatically reduced as a skill or movement becomes learned by the body. Reducing the activation of Renshaw cells in these specific pathways can lead to greater force production and displays the importance of skill learning, particularly in high-velocity movements.

Sarcoplasmic Reticulum:

Sarcoplasmic reticulum is a network located inside the cell surrounding muscle myofibrils and functions among other things to release and take up free calcium ions during contraction and relaxation, respectively [31,32,33]. During repeated muscle contractions, the functioning of the sarcoplasmic reticulum is significantly reduced [31,32,33]. This reduction in function occurs in both athletes and non-trained persons, and appears to be slightly better in athletes with a chronic training status. A reduction in the ability of the sarcoplasmic reticulum to release and/or take up calcium is directly related to a reduction in muscle force produced.

The functioning of sarcoplasmic reticulum during fatigue seems to be related to muscle fiber type [31,32,33]. Type I fibers do not see as rapid of a decline in function when compared to type II fibers [31,32,33]. Type II muscle fibers have an estimated double the calcium release for contraction and up to two to three times higher uptake rate than type I fibers. The higher requirements of type II fibers in regards to calcium release and uptake helps explain their greater susceptibility to fatigue [31,32,33].

In the realm of athletics, an increase in RFD could, theoretically, be caused by an enhancement of the sarcoplasmic reticulum calcium release and reuptake [31,32,33]. Studies to date have yet to conclusively answer whether or not this is trainable. One study did show a significant increase in the peak rate of calcium release after a training period involving high-intensity intermittent training [32]. This adaptation makes sense in the fact that type II fibers are being trained, which require higher rates and amounts of calcium in order to improve force output. Other studies show there is no change in the release of calcium from the sarcoplasmic reticulum [31,33]. To date, no study has shown calcium uptake is increased post-training. This lack of adaptation leads to the notion that improved calcium uptake by the sarcoplasmic reticulum is likely not the cause of fatigue if a reduced co-activation is seen within the trained muscle [31,33].

A theory for the increased calcium release seen by Matsunaga et al. could be due to multiple factors including, but not limited to, increased sarcoplasmic reticulum in the existing fibers and/or structural changes to the proteins responsible for opening and closing the calcium ion gates [32].

Cross Sectional Area:

Success in athletics is often determined by force production, which is determined by both the number of muscle fibers activated during a movement and the level of activation of each fiber. As muscle cross sectional area increases, so does the force producing capability of the muscle [2]. The hypertrophy of the powerful, type II muscles is thought to be a primary cause of this adaptation. It is improvements in hypertrophy and force production that cause coaches to focus on maximal strength, or increased muscle mass. If other factors that are needed to create force rapidly are overlooked, a coach will not be preparing his/her athletes optimally for competition.

Conclusion:

In the realm of athletics, an ability to produce high forces is an important quality. However, based on the limited amount of time allotted to perform movements seen in competition, the rate at which force is produced truly becomes the predictor of performance. Rate of force development is determined based on the force production and coordination of the musculature completing the desired movement, with many sub-categories forming these two factors within the nervous and muscular systems. Specific training has the ability to improve RFD qualities when appropriate methods are implemented.

References

1. Aagaard, P., Simonsen, E., Andersen, J., Magnusson, S., Dyhre-Poulsen, P. (2002). Increased rate of force development and neural drive of human skeletal muscle following resistance training. *Journal of Applied Physiology, 93*(4), 1318-1326. doi:10.1152/japplphysiol.00283.2002.
2. Andersen, L., Andersen, J., Zebis, M., Aagaard, P. (2009). Early and late rate of force development: differential adaptive responses to resistance training? *Scandinavian Journal of Medicne & Science in Sports, 20*(1), 162-169. doi:10.1111/j.1600-0838.2009.00933.x.
3. Cormie, P., McGuigan, M., Newton, R. (2011). Developing maximal neuromuscular power: part 1 – biological basis of maximal power production. *Sports Medicine, 41*(1), 17-38. doi:10.2165/11537690-000000000-00000.
4. Oliveira, F., Oliveira, A., Rizatto, G., Denadai, S. (2013). Resistance training for explosive and maximal strength; effects on early and late rate of force development. *Journal of Sports Science and Medicine, 12*(3), 402-408.
5. Tillin, N., Folland, J. (2014). Maximal and explosive strength training elicit distinct neuromuscular adaptations, specific to the training stimulus. *European Journal of Applied Physiology, 114*(2) 365-374. doi:10.1007/s00421-013-2781-x.
6. Tillin, N., Pain, M., Folland, J. (2012) Short-term training for explosive strength causes neural and mechanical adaptations. *Experimental Physiology, 97*(5), 630-641. doi:10.1113/expphysiol.2011.063040.
7. Young, W. (2006). Transfer of strength and power training to sports performance. *International Journal of Sports Physiology and Performance, 1*(2), 74-83.
8. Griffin, L., Cafarelli, E. (2005). Resistance Training: Cortical, Spinal, and Motor Unit Adaptations. *Canadian Journal of Applied Physiology, 30*(3), 328-340. doi:10.1139/h05-125.
9. Oliveira, F., Rizatto, G., Denadai, B. (2013). Are early and late rate of force development differently influenced by fast-velocity resistance training? *Clinical Physiology and Functional Imaging, 33*(4) 282-287. doi:10.1111/cpf.12025.
10. Moritani, T. Time course adaptations during strength and power training. *Mechanisms of Adaptation* (266-278).
11. Geertsen, S., Lundbye-Jensen, J., Nielsen, J. (2008). Increased central facilitation of antagonist reciprocal inhibition at the onset of dorsiflexion following explosive strength training. *Journal of Applied Physiology, 105*(3), 915-922. doi:10.1152/japplphysiol.01155.2007.
12. Carolan, B., Cagarelli, E. (1992). Adaptations in coactivation after isometric resistance training. *Journal of Applied Physiology, 73*(3), 911-917.
13. Aagaard, P., Simonsen, E., Andersen, J., Magnusson, S., Bojsen-Møller, F., Dyhre-Poulsen P. (2000). Antagonist muscle coactivation during isokinetic knee extension. *Scandinavian Journal of Medicne & Science in Sports, 10*(2), 58-67. doi:10.1034/j.1600-0838.2000.010002058.x.
14. Bazzucchi, I., Riccio, M., Felici, F. (2008). Tennis players show a lower coactivation of the elbow antagonist muscles during isokinetic exercises. *Journal of Electromyography and Kinesiology, 18*(5), 752-759. doi:10.1016/j.jelekin.2007.03.004
15. Remaud, A., Cornu, C., Guével, A. (2009). Agonist muscle activity and antagonist muscle co-activity levels during standardized isotonic and isokinetic knee extensions. *Journal of Electromyography and Kinesiology, 19*(3), 449-458. doi:10.1016/j.jelekin.2007.11.001.
16. Baker, D., Newton, R. (2005). Acute effect on power output of alternating an agonist and antagonist muscle exercise during complex training. *Journal of Strength and Conditioning Research, 19*(1), 202-205. doi:10.1519/1533-4287(2005)19<202:AEOPOO>2.0.CO;2.
17. Karst, G., Hasan, Z. (1987). Antagonist muscle activity during human forearm movements under varying kinematic and loading conditions. *Experimental Brain Research, 67*(2), 391-401. doi:10.1007/BF00248559.

18. Maeo, S., Yoshitake, Y., Takai, Y., Fukunaga, T., Kanehisa, H. (2013). Effect of short-term maximal voluntary co-contration training on neuromuscular function. *International Journal of Sports Medicine, 35*(2), 125-134. doi:10.1055/s-0033-1349137.
19. Sale, D. (1988). Neural adaptation to resistance training. *Medicine and Science in Sports and Exercise, 20*(5Suppl), S135-145.
20. Rutheford, O., Jones, D. (1986). The role of learning and coordination in strength training. *European Journal of Applied Physiology, 55*(1) 100-105.
21. Dal Maso, F., Longcamp, M., Amarantini, D. (2012). Training-related decrease in antagonist muscles activation is associated with increased motor cortex activation: evidence of central mechanisms for control of antagonist muscles. *Experimental Brain Research, 220*(3-4), 287-295. doi:10.1007/s00221-012-3137-1.
22. Arai, A., Ishikawa, M., Ito, A. (2013). Agonist-antagonist muscle activation during drop jumps. *European Journal of Sports Science, 13*(5), 490-498. doi:0.1080/17461391.2013.764930.
23. Draganich, L., Jaeger, R., Kralj, A. (1989). Coactivation of the hamstrings and quadriceps during extension of the knee. *The Journal of Bone and Joint Surgery, 71*(7), 1075-1081. doi:10.1016/0021-9290(90)90232-R.
24. Gabriel, D., Kamen, G., Frost, G. (2006). Neural adaptations to resistive exercise: mechanisms and recommendations for training practices. *Sports Medicine, 36*(2), 133-149. doi:10.2165/00007256-200636020-00004.
25. Manning, C., McDonald, P., Murnaghan, C., Bawa, C. (2013). Reciprocal inhibition versus unloading response during stretch reflex in humans. *Experimental Brain Research, 226*(1), 33-43. doi:10.1007/s00221-013-3408-5.
26. Rao, G., Amarantini, D., Berton, E. (2009). Influence of additional load on the moments of the agonist and antagonist muscle groups at the knee joint during closed chain exercise. *Journal of Electromyography and Kinesiology, 19*(3), 459-466. doi:10.1016/j.jelekin.2007.12.001.
27. Remaud, A., Guével, A, Cornu, C. (2007). Antagonistic muscle coactivation and muscle inhibition: effects on external torque regulation and resistance training-induced adaptations. *Clinical Neurophysiology, 37*(1), 1-14. doi:10.1016/j.neucli.2007.01.002.
28. Wierzbicka, M., Wiegner, A. (1992). Effects of weak antagonist on fast elbow flexion movements in man. *Experimental Brain Research, 91*(3), 509-519. doi:10.1007/BF00227847.
29. Carlson, R. (2006). Changes in muscle coordination with training. *Journal of Applied Physiology, 101*(5), 1506-1513. doi:10.1152/japplphysiol.00544.2006.
30. Scholz, J., Klein, M.C., Behrens, T.E.J., Johansen-Berg, H. (2009). *Training induces changes in white matter architecture. Nature Neuroscience, 12*(11), 1370-1371. doi:10.1038/nn.2412.
31. Li, J., Wang, X., Fraser, S., Carey, M., Wrigley, T., McKenna, M. (2002). Effects of fatigue and training on sarcoplasmic reticulum Ca2+ regulation in human skeletal muscle. *Journal of Applied Physiology, 92*(3), 912-922. doi:10.1152/japplphysiol.00643.2000.
32. Matsunaga, S., Yamada, T., Mishima, T., Sakamoto, M., Sugiyama, M., Wada, M. (2007). Effects of high-intensity training and acute exercise on in vitro function of rat sarcoplasmic reticulum. *European Journal of Applied Physiology, 99*(6), 641-649. doi: 10.1007/s00421-006-0381-8.
33. Ortenblad, N., Lunde, P., Levin, K., Andersen, J., Pedersen, P. (2000). Enhanced sarcoplasmic reticulum Ca^{2+} release following intermittent sprint training. *The American Journal of Physiology - Regulatory, Integrative and Comparative Physiology, 279*(1), 152-160.