The current understanding of child-adult differences in muscular and neuromotor function will be reviewed while highlighting the gaps in our knowledge and raising research questions that could be addressed in the immediate or near future. Topics include muscle activation, muscle composition, strength attributes, strength-and aerobic-training, neuromotor development, where neuromuscular differences originate from, and the possible interrelationships between motor and cognitive function. The various differences will be discussed on their specific merits, but also as possible manifestations of a common underlying factor which, if true, could provide a more holistic view of child-adult functional differences.

Prepubertal children differ from adults not only in bodily dimensions, but in many anatomical, physiological, and performance characteristics. Muscle-functional differences have been some of the most studied child-adult differences in the last several decades. It is well established that children are not as strong or as fast as adults, even after correcting for dimensional differences. Lower rates of force development; higher lactate-, ventilatory- and electromyographic-thresholds, lower fatigue, better endurance, and faster recovery from exhausting exercise, constitute only a partial list of those differences. The scope of research has gradually widened and new questions can be asked. It is no longer just force, speed, or endurance that can be measured, and neuromotor function is an integral part of the research into what sets children apart from adults. This review aims to point at some of the major issues in these areas while highlighting gaps in our knowledge and relevant questions that could and ought to be addressed in the immediate or near future.

Discrete or Interrelated Differences?

Child-adult differences in muscular performance and neuromotor function may be examined independently of each other. Such an approach can provide specific knowledge, but might prevent more holistic understanding and appreciation of what sets children physiologically and functionally apart from their parents or other adults. Whether some or all of these differences can be viewed as stemming from some common underlying factor is in itself an interesting and highly relevant question and part of what ought to be answered in the foreseeable future.

Humans are typically not able to voluntarily activate all 100% of their available motor units (MUs) in a given muscle and children are even less capable in this respect than adults. The first to claim that were Asmussen and Heeboll-Nielsen, already in 1955 (1). Surprisingly, some 50 years have elapsed before attempts were made to directly show that (e.g., (21)). Invoking the Henneman size principle (13) and the known general hierarchy of MU recruitment, it stands to reason that the MUs children are incapable of recruiting are composed solely or mostly of the highest-threshold type, i.e., Type II MUs (or the higher subpopulations thereof: IIAB and IIB).

Typically, child-adult differences in muscular performance and metabolism are explained independently of each other, on grounds such as differential substrate composition, muscular immaturity, allometry, etc. While such explanations may be valid on their own, they fail to consider a possible wider perspective that might point to similarities between seemingly unrelated differences and potentially unearth common underlying factors. Good examples are child-adult differences in anaerobic- or fat-oxidation-capacities. While the former are typically attributed to deficient glycolytic enzyme and glycogen
levels, the latter are explained by higher activity of oxidative enzymes.

A valid alternative to these explanations and to regarding the child’s muscle as “metabolically immature” is to view the differences as reflecting different functional muscle-fiber compositions. In other words, greater reliance on Type I vs. Type II MU activation could similarly explain the differences. At the same time, this very same activation difference would explain children’s lower muscular force, greater endurance, and other child-adult differences. This view and children’s proven muscle-activation deficit relative to adults, has led to postulating the hypothesis that children are more limited than adults in their capacity to recruit Type II MUs (see (9) for review). While dimensional and other differences may still be important, the attractiveness of this hypothesis lies in the capacity of a single underlying factor to provide a unified theory for all the mentioned child-adult differences and many others.

Although the hypothesis enjoys a large body of supportive evidence, no conclusive evidence is yet available. Two recent studies provide the best evidence to-date in that they came closest to direct differentiation of Type II from Type I MU activation (19,25). The studies show boys and girls as having higher relative electromyographic threshold (EMGTh) levels than men and women, respectively. Edwards and Lippold (12) were the first to show a nonlinear, threshold-like phenomenon in the relationship between muscle force and its electrical (EMG) activity. Subsequently, the general understanding among EMGTh researchers has been that the EMGTh reflects the onset of accelerated recruitment of Type II MUs (or their higher-threshold, types IIAB and IIB subpopulations) (e.g., (14)). Although the EMGTh has previously only been tested in adults, there is no reason to assume the same is not true for children. Thus, it is hard to escape the conclusion that children recruit Type II MUs later and to a lesser extent than adults.

In many cases, particularly in children (less so in women and least in men), EMGTh cannot be detected at all. Exhaustion in ramped exercise, as used in the mentioned EMGTh studies, may occur when muscle force reaches only ~50% MVC for the conditions (cadence, fatigue). Thus, it is reasonable to assume that individuals unable to attain sufficiently high MVC percentage at exhaustion may not be reaching their EMGTh point. Since children are known to be relatively weaker and less anaerobic than adults (presumably, due to their relative inability to recruit Type II MUs), they ought to be considered as less likely to attain the necessary contractile intensities to elicit EMGTh, which has been shown in those studies.

In essence, the differential EMGTh findings constitute strong evidence that children’s functional muscle composition is different than that of adults. That is, they use more Type I and fewer Type II MUs, at the upper work intensities. The current findings, however, cannot discern whether this is due to differences in MU activation or in muscle composition.

**Suggested Next Steps**

The child-adult differential muscle activation hypothesis ought to be further substantiated and proven, or else refuted. The following are some possible directions.

1. Validate child-adult EMGTh differences under different exercise modes or protocols. For example, maximal voluntary contractile force (MVC) is never attained in ramped exercise due to early exhaustion. Consequently, EMGTh may not be detected before exercise termination. To overcome that, EMGTh could be studied in relatively short, discrete bouts of isometric or dynamic contractions that could attain MVC intensity.

2. Test the EMGTh under different levels and types training (e.g., trained vs. untrained children and adults; endurance- vs. strength- or power-training) (See also under Strength Training, below). This could shed light on whether and to what extent, different types of exercise and training may affect the EMGTh and, presumably, MU activation/recruitment.

3. Advanced frequency analysis techniques, such as the Wavelet surface-EMG analysis method (29) could be used to differentiate MU recruitment patterns between children and adults.

4. Functional magnetic-resonance imaging methods, now becoming more potent and accessible, could be applied to either the brain (motor cortex) or the working muscle, or both. This might eventually provide the necessary conclusive evidence for differential MU activation.

5. Elucidate the true child-adult muscle compositional differences and their relationship to differential muscle activation (for more, see ‘Muscle Composition’ below).

**Muscle Composition**

As mentioned, possible muscle compositional differences are intimately related to child-adult muscle-functional differences because, similar to MU activation, they too determine the ultimate functional composition of the working muscle.

Due to the associated ethical constraints, pediatric muscle-biopsy data are very limited and the question of whether and how children’s muscle composition differs from that of adults is still unsettled. Nevertheless, two studies in particular appear to be robust enough to strongly suggest that, compared with adults, children’s muscle composition is skewed toward higher Type I (lower Type II) fibers. Jansson (15) compiled various muscle biopsy batches that have been obtained as part of the clinical investigation of hospitalized children and adults. Her nonlinear regression analysis showed ~60% Type I fiber composition at age 5, but only ~42% at age 40. More than half of that change having taken place between 5 and 20 years of age. Similarly, Lexell et al.
(18) showed ~70% mean Type I fiber composition at age 5, but only ~50% at age 20.

In their famous 1969 cat cross-innervation study, Buller et al. (5) showed that muscle-fiber phenotype is determined by the type of innervation it receives. Using genetic profiling techniques, Simonneau and Bouchard (28) found only ~45% of eventual muscle composition to be genetically set, while ~40% are, or could be, environmentally determined—presumably by such factors as the type, intensity, and duration of usage. Thus, the plasticity of muscle composition and its functional characteristics is well established. With this understanding, the tentative picture of decreasing Type I and increasing Type II fiber composition with maturation appears compatible with the hypothesized increased utilization potential of Type II MUs. Possibly, some MUs, genetically-destined to be Type II, first present as Type I phenotypes, or do not function at all, because of the limited impulse frequency levels their motoneurons can transmit, if at all. Only when maturation-related processes (notably neuronal myelination; further discussed later) eventually raise their transmittable frequency levels, do those MUs turn into the Type II phenotypes they were destined to become. This hypothesis will likely be hard to validate or refute in the near future.

**Suggested Next Steps**

1. Expand the pediatric muscle-composition database so as to validate or update the current picture of maturational change, currently largely based on just two studies

2. Based on a more robust muscle-composition database, elucidate the pattern of change and identify the extent to which it is environmentally affected (e.g., by type and intensity of habitual activity and physical training).

3. Aside from clinical biopsy data, the best noninvasive prospect seems to lie with the existing and expanding potential of magnetic resonance imaging (MRI). When this becomes available it will be possible to correlate muscle composition with neuronal myelination (also via MRI) from childhood to adulthood and possibly substantiate or refute the changing composition hypothesis.

**Strength Training**

Children respond to resistance training with relative strength gains comparable to those of adults (e.g., (27)). However, unlike the latter, children’s strength gains are associated with relatively little or no observed muscle hypertrophy (4,27). The reason for children’s lack of hypertrophic response is not known, but may be related to their low androgen/estrogen levels. Directly or indirectly, the rising sex-hormone levels during maturation may also be responsible for raising muscle activation levels, but there is no evidence to support or refute this hypothesis.

Hormonal involvement notwithstanding, neural adaptations are integral to the strength-training response at any age. However, their relative lack of hypertrophic response implies that children must almost exclusively rely on neural adaptations to attain their adult-comparable strength gains. Adaptations such as improved MU synchronization, increased rate (speed) of activation, or reduced antagonist-agonist coactivation likely constitute major contributors. However, children’s larger activation deficits (percentage of unactivated MUs) provide them with a greater improvement potential for attaining their adult-comparable strength gains, thus minimizing or eliminating reliance on muscle hypertrophy. Indeed, using different techniques, both Ramsay et al. (26) and Ozmun et al. (22) suggested ca.15% increases in muscle activation following resistance training, which only partly explained the ca. 25% strength increases.

It is interesting to note that children’s nonhypertrophic strength gain is the only child-adult muscle-functional difference that cannot be explained by differential muscle composition, thus providing support to the differential muscle-activation hypothesis.

Typical strength training aims at improving maximal contractile force, but other strength attributes are both functionally important and conceptually significant for understanding child-adult differences. “Explosive strength,” or high rate of force development (RFD), is often more important than maximal force. For example, in activities such as running and jumping, only a fraction of a second is available for force generation—significantly shorter than the >1 s typically needed for maximal force attainment. Greater RFD means that higher percentages of maximal force can be attained early in a given contraction. Maximal RFD (normalized to size or maximal force) is higher in adults than in children (e.g., 10). This explains why, compared with children, young adults are the better sprinters and jumpers. This is due not only to their more forceful push-offs, but no less to the fact that during the limited ground-contact time (typically 50–200 ms) they are capable of attaining considerably greater fractions of their maximal force.

To date, most pediatric strength-training research has focused on isometric, dynamic, or isokinetic strength, with little or no attention to force explosiveness. An exception is a study by Waugh et al. (30) which reinforces what has been known in adults, namely, that standard, slow resistance training produces little or no change in normalized RFD (absolute RFD may rise just as a function of the increased maximal force). Since RFD typically increases with increasing Type II fiber composition, it ought to reflect changes or differences in the functional composition of a given muscle. This may well be the reason for RFD being higher in adults, compared with children.

A study in our laboratory examined RFD from this perspective. Instead of administering a typical 8–12-week training regimen, the study compared long-term, well-trained, prepubertal boy gymnasts to their untrained peers, as well as to untrained adult men (8). The
gymnasts’ elbow-flexion force kinetics (0–100% MVC) was superior to that of the untrained boys and closely matched that of the men. However, the gymnasts’ maximal RFD, while 32% higher than that of the untrained boys, was still 13% shy of the men’s. On the other hand, the gymnasts’ size-normalized maximal force, although 20% higher than their peers’, was still 28% lower than the men’s. Note: Since the gymnasts’ advantages were observed in gymnastics-stressed muscles, but not in relatively-untrained ones, they were attributable to training rather than to athletic preselection.

The study’s findings demonstrate that: a. Suitable (explosive) training can significantly improve contractile attributes (RFD, kinetics) in prepubertal boys and not only maximal strength. It remains unresolved whether or to what extent is children’s response to explosive training different from that of adults. b. If the training effects were all due to increased muscle activation, then RFD and maximal strength should have changed similarly in relation to the untrained peers or adults. However, since the gymnasts’ RFD was only 13% lower than the men’s, but their max force was 28% lower, other neuromotor adaptations must have been heavily involved. Thus, the questions of whether, or to what extent, did increased MU activation play a role remain unanswered.

Plyometric training is a special form of strength training, which has repeatedly been shown to improve force explosiveness in adults as well as in children (e.g., 17). It involves fast muscle-tendon prestretching, which in turn reflex-activates the involved muscle to an extent typically unattainable in voluntary contractions. For a given strength gain, plyometric training was shown to induce little or no muscle hypertrophy compared with standard resistance training in adults. Plyometric training is therefore particularly favored by athletes, such as high-jumpers, who want to maximize explosive strength gains while adding little or no muscle mass (body weight).

The much-limited hypertrophy means that plyometric training effects are largely neural in nature. This, in turn, makes plyometric training a potential window into the nature of Type II MU involvement, not provided by standard strength training. The augmented reflexive response to the plyometric prestretching presumably activates more MUs than can be accessed voluntarily. The outstanding question then is whether plyometric training merely induces ‘standard’ neuromotor factors such as activation rate and muscular coordination, or perhaps also lowers the activation threshold of Type II MUs that previously were voluntarily inaccessible.

Plyometric-training research in children has so far focused on quantifying performance gains such as in jumping height or distance and in sprinting speed (e.g., 17), but data on neuromotor responses have been completely missing. Since there is some evidence for increased muscle activation following resistance training in children (22,26), and since plyometrics presumably access a greater proportion of the MU pool than do standard contractions, it stands to reason that increased activation (i.e., Type II MU recruitment) would be significantly more prominent in plyometric- than in resistance-training. This has not yet been shown.

A practical question waiting to be addressed is whether children’s differential responses to strength training, compared with adults, should mean different approach and guidelines for optimizing the training and coaching of young athletes and children in general?

**Suggested Next Steps**

1. Compare plyometric- vs. resistance-training effects on physical performance (laboratory dynamometers) and neuromotor functions in both children and adults.
2. Estimate possible training-induced changes in Type II MU recruitment via changes in activation level and EMG frequency profiles in both children and adults.
3. Compare possible (inverse) relationships between activation and hypertrophic changes in response to each type of training, in both children and adults.

**Aerobic Training**

Aerobic training does not seem to belong in the discussion of children’s muscle-functional and neuromotor attributes. However, it might be precisely the peculiarity of these attributes that could provide an explanation to one of pediatric exercise science’s most significant enigmas.

Although body-weight-normalized maximal aerobic power of pre- and early-pubertal children is on par with that of young adults, its response to training is significantly more limited and stands in stark contrast to children’s comparable gains in response to strength training. The reason for this is not known, and no satisfactory explanation for that has yet been put forth.

As mentioned earlier, children attain lower levels of maximal voluntary muscle activation compared with adults (e.g., 21). The hypothesized implication, that children have lesser access to the higher-threshold Type II MUs, may be of fundamental significance in aerobic training. Since the lower-threshold, Type I MUs are more frequently used and more aerobically adept than their Type II counterparts, their potential aerobic-training effects may be more limited than those associated with the less-recruited and therefore less trained Type II MUs. Thus, children’s greater reliance on Type I MU use in habitual activities and training may mean lesser training effect. On the other hand, at the same relative training intensities, adults presumably recruit more of the habitually-less-active and aerobically-weak, Type II MUs, and might consequently be able to reap greater aerobic gains.

In light of the above, an intriguing question arises. If plyometric, or any other forms of training, can indeed induce increased Type II MU recruitment in children, could that improve children’s response to aerobic training, as well?
Suggested Next Steps

1. Aerobically train children and compare aerobic improvements in those who previously had no training vs. those who had resistance- or plyometric-training.

2. Compare aerobic training effects in children with higher voluntary activation levels, or lower EMG_{Th} values, with children possessing lower activation / higher EMG_{Th} values.

Neuromotor Development

When muscles undergo maturational changes in performance or composition, one or more developmental processes must be involved. Very little, however, is known about the specifics of such processes in relation to neuromotor development and whether and how they might be related to other known maturational changes.

Boucard study (28) which showed muscle-composition differences originate from, or mediated through the relative abundance of several synaptic-vesicle proteins (SV2A, SV2B, SV2C) in the neuromotor end-plates, where SV2A is most prevalent at birth and early life and imposes slow-twitch (Type I) characteristics (6). It is not clear how those proteins are regulated, but this might very well be centrally determined by the nature of the in-coming neural impulses. It is relevant to recall the aforementioned Simoneau and Bouchard study (28) which showed muscle-composition variance to be as much as ~40%, environmentally-determined. In other words, the manner in which muscle fibers are used, and its associated firing frequencies, may well influence their phenotypic expression.

Some known developmental changes in nervous system attributes provide other likely perspectives of the same issue. Koh and Eyre (16) have shown corticospinal conduction velocity to increase from 33 weeks of gestational age to adulthood, with adult-level velocities already attained around 11–12 years of age. More than 10 years later, both Paus et al. (23) and Barnea-Goraly et al. (3) demonstrated maturation- or age-related structural changes and increased white-matter prevalence in the brain. Since ‘white matter’ is widely regarded as reflecting neuronal myelination, those observations nicely complement the increasing conduction velocities shown earlier by Koh and Eyre (16). These findings go hand-in-hand also with the higher conduction velocities typical of the α motor-neurons that innervate Type II MUs which presumably become more accessible during that same time period. Furthermore, low myelination levels effectively filter out higher-frequency impulses and thus may be rendering genetically-destined Type II MUs incapable of functioning as such. A major difference between the myelination and conduction-velocity observations was that while rising conduction velocity plateaued at 11–12 years of age, the increase in white matter appeared to proceed steadily at ~10% per year between the ages of 6 and 20 (3). Preliminary data from our laboratory suggest that functional changes consistent with increased Type II MU activation tend to plateau or greatly diminish at puberty. As the myelination data refer only to the CNS, a tentative explanation is that motor cortex and motor-neuron maturation is generally complete at puberty and that the continued myelination is consequential only for continued mental/cognitive development. This, of course, remains to be elucidated.

The ever-growing specialization in all areas of research has facilitated great scientific advances. However, the flip side of the same coin is a growing interdisciplinary disconnect. This has curtailed the discovery of important relationships between seemingly unrelated findings from different areas of research. Researchers in areas such as pediatric exercise physiology, neurol-ogy, neuromuscular physiology, psychology, and motor development are a relevant case in point. They possess extensive knowledge in their respective fields, but rarely make concerted efforts to share knowledge and bridge respective gaps therein. Interdisciplinary cooperation could greatly enhance understanding of the multifaceted aspects of neuromotor and muscle-functional development.

Suggested Next Steps

1. Seek interdisciplinary cooperation (across motor development, neurology, exercise physiology, neuromuscular physiology, and psychology).

2. Collect more data on variables such as myelination and conduction velocities to better characterize their maturational patterns and interrelationships (e.g., why conduction velocity plateaus ca. puberty, while the myelination process is still ongoing for several more years?)

3. Correlate changes in central vs. peripheral myelina-tion rates to the corresponding changes in conduction velocity, muscle contractility, and EMG activity/ frequency patterns.

Where do Motor-Functional Differences Originate From?

Children have been shown to activate a smaller fraction of their MU pool in maximal voluntarily contractions, compared with adults (e.g., 21). However, the origin of this differential activation has not yet been located. Spinal or peripheral inhibition of specifically higher-threshold, Type II MUs has been proposed, but evidence such as
that provided by the afore-mentioned myelination studies (3,23) lends credence to the CNS as the primary or exclusive origin of this difference. Moreover, a threshold phenomenon in EEG activity during graded exercise (EEGTh, not unlike the EMGTh) has been shown in adults (2). That single finding still requires confirmation, but if true, it strongly suggests that the point at which Type II MUs begin to be extensively recruited is determined already in the CNS rather than peripherally. This, in turn, makes it highly likely that child-adult EMGTh-like differences would also be found in the EEGTh.

When a given muscle is made to maximally and explosively contract, command stimuli are generated in the motor cortex. Considering the known age-related increases in myelination level and conduction velocity, and if the child-adult activation differences do indeed originate in the CNS, then one or more of the following scenarios appear possible: a. The generated stimuli are of lower frequency in children and therefore cannot activate a portion of the higher-threshold (‘typeII’) motoneurons; b. Due to factors such as myelination level, a portion of the motoneuronal pool, destined to eventually activate Type II MUs, cannot relay higher-frequency impulses and temporally activates its associated MUs as type I, rather than type II. c. Motoneurons, destined to eventually innervate higher-threshold, Type II MUs, are not fully developed during childhood (e.g., have activation thresholds that are too high to transmit any impulses).

Suggested Next Steps

1. As suggested above, seek interdisciplinary cooperation (across motor development, neurology, exercise physiology, neuromuscular physiology, and psychology).
2. Existing EEG analysis techniques are sufficient to facilitate the following:
   a. Confirm the existence of the EEGTh.
   b. Compare children’s EEGTh with that of adults and find out whether the differences, if any, are comparable to those recently shown for the EMGTh.
   c. Measure EEGTh and EMGTh simultaneously and find out the extent of their congruence in children and in adults (a high correlation and particularly coincidence, would strongly suggest direct EEGTh-to-EMGTh causality).
3. Functional MRI may be used to distinguish motor cortex activity between children and adults.

Relationship Between the Motor and Cognitive Functions

To discuss child-adult muscle-functional differences is to talk about fundamental aspects of neuromotor development. Even among typically-developing children, early gross-motor development has been associated with better cognitive/executive function of school-age children (24) and adults (20). Since typical gross-motor testing involves large muscle coordination tasks such as walking, jumping, balancing, ball handling, or even the age at first standing, no assessment of isolated muscle attributes such as maximal force or RFD is ever done. Thus, the developmental relationships between neuromotor and cognitive attributes have never been established.

A particularly interesting aspect of possible interdependence between motor function and cognition is the cerebellum-prefrontal-cortex axis. The two have long been viewed as fulfilling completely different and unrelated functions. Although the evolutionary parallelism between the development of the prefrontal cortex and that of the neocerebellum was demonstrated already in the early 1940s (11), the relationship between the two structures has escaped attention until much more recently. In her review of the relationships between these two brain parts, Adele Diamond states that “Motor development and cognitive development may be much more interrelated than has been previously appreciated. Indeed, they may be fundamentally intertwined”. She concludes that “…the cerebellum may not only subserve motor function, but may play a role in cognition as well. Conversely, prefrontal cortex, through its connections with cortical and subcortical centers important for movement control, may play a role in motor function, not simply in cognition” (7).

The interrelationship between normal motor and normal mental/cognitive development is now well recognized in child upbringing (e.g., 20,24). This relationship is clearly not claimed to extend to the extremes of these two capacities. We do not necessarily see top athletes at the top of the mental/cognitive scale, or vice versa. Still, the question arises as to whether general or specific cognitive benefits might be gained from heightened levels of physical/motor training. Would early, high-level cerebellar development translate into early or lasting cognitive advantages? Gymnastic training and competition is a particularly interesting case in point. Well-trained young gymnasts already possess motor-skill and coordination level on par with adult gymnasts and much superior to that attained by untrained children or adults. Since the cerebellum is responsible for motor coordination, the young gymnasts’ cerebellum must be highly developed. Do these gymnasts have any identifiable mental/cognitive advantages, or even just differences, compared with their nonathletic peers, due to their presumably highly-developed cerebellum? If so, are gymnastics and comparable other types of physical activity and training, superior to others in bestowing cognitive/mental benefits? Quite likely, a distinction must be made between the basic cerebellum that is present in vertebrates and in all mammals (some of which are motorically far superior to humans) and the neo-cerebellum that has developed in parallel with the prefrontal cortex (11). Motor skill and coordination might be the sole domain of the ‘old’ cerebellum with no direct bearing on cognitive, prefrontal-cortex functions. The neo-cerebellum, on the other hand, might be a
 coordinator of higher, nonmotor functions, one of which might be cognitive function.

At this point it seems most plausible that the functional motor-cognitive relationship is mainly relevant at the basic levels, which are developmentally significant in infancy and early childhood, while higher motor and cognitive capacities are likely much more, or completely independent. Not inconceivable, however, is that high-level motor/cerebellar capacity might have limited positive effects on very specific aspects of cognitive function. Currently, this exciting topic is largely “terra incognita” and there is much theoretical and practical to be learned here.

**Suggested Next Steps**

1. As suggested earlier, seek interdisciplinary cooperation (across motor development, neurology, exercise physiology, neuromuscular physiology, and psychology).

2. Find out what cognitive differences, if any, could be discerned in adolescents and young adults who, as children, grew up with widely varying levels of physical activity. A subquestion of the above is whether such differences are extended in high-level athletes compared with nonathletes. A particular challenge is to tease out the cognitive attributes of the specific sport, which might affect athlete preselection, from those that might be improved by the activity per se.

3. Similarly, if such differences indeed exist, are they specific to certain types of physical activity rather than others?

**Conclusions**

Much is yet to be learned about the time course of the various muscle-functional and neuromotor changes children and adolescents undergo during maturation. The available evidence, however, strongly invites correlation with the time course of the rising sex-hormones levels. In both sexes these hormones are known to be muscle-hypertrophic, aside from being responsible for so many other physiological and psychological changes. The question of whether these hormonal changes can be linked with the known or suspected changes in muscle activation and composition, or the responses to strength and aerobic training, will likely be pivotal in understanding neuromotor development and where the various child-adult differences originate from.

Finally, how motor development and type of physical activity may be related to cognitive development and function is an intriguing question with potential implications on how physical education is viewed in the educational system.

**References**


