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CONTENTS

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CONTENTS

SKELETAL MUSCLE MYOSIN HEAVY CHAIN ISOFORM EXPRESSION AND RESISTANCE ACTIVITY: A REVIEW

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ABSTRACT

Skeletal muscle tissue is characterised by its plasticity and heterogeneity. The diversity of myosin heavy chain (MHC) isoforms suggests that skeletal muscle tissue presents a continuum, rather than distinct categories, of contractile properties. Resistance activity appears to affect the expression of different MHC isoforms in skeletal muscle. These changes in MHC isoforms may be a function of muscle contraction (i.e., isoinertial, isometric or isokinetic) and/or training regimen (i.e., bodybuilding or olympic and power lifting). Typically, bodybuilding type resistance-trained athletes possess more slower MHC isoforms than athletes with chronic exposure to olympic and power lifting. In addition, changes in MHC isoform composition may take place after only a few training sessions. For example, the molecular composition of MHC isoforms in rats could significantly change after two training sessions. In humans, significant changes in MHC isoform expression have been observed to occur as early as after five workouts. However, it is not yet clear what mechanisms trigger the changes in MHC isoform content during resistance activity. It has been proposed that MHC type IIb proteins are not suitable for prolonged resistance activity. The possible mechanisms to explain losses in MHC type IIb isoform percentage may include the increased recruitment of fast-twitch glycolytic (FTb) fibres and/or increased metabolic demands of working muscle.

Key words: myosin heavy chain isoforms, resistance training

MYOSIN HEAVY CHAIN ISOFORM EXPRESSION IN MUSCLE FIBRES

Myosin heavy chain (MHC) isoform composition in the adult muscle tissue appears to be implicated in the maximum velocity of shortening, adenosine triphosphate (ATP) turnover and ATPase activity [8, 18, 42, 57, 58, 59]. In addition, the relationship between MHC isoform expression and the speed of muscle fibre contraction, and time to peak tension has been reported [42, 53]. Thus, conceivably, changes in the MHC isoform profile of muscle tissue could have performance implications. The strong correlation between the presence of MHCs and myosin ATPase activity has been used to discriminate between muscle fibre types [8, 57, 58, 59].

There are marked inter- and intra-muscle variations in the expression of MHC isoforms [42, 53]. Fast IIb, fast IIa and slow I MHC isoforms are typically found in normal adult skeletal muscle [17, 53]. Recently, an additional fast MHC isoform has been detected in rat muscle tissue [7] and perhaps also in human tissue [31, 53]. This third fast MHC isoform appears to migrate between the type IIa and IIb MHC isoforms in rats [53] and has been labelled MHC IIx [12, 18]. It has been suggested that MHC IIx is encoded by a specific messenger ribonucleic acid (mRNA) and is not a posttranslational variant of other fast MHC isoforms [18, 53]. However, as the expression of the MHC IIx gene is coordinately regulated with MHC IIa and IIb genes [18], only certain combinations of fast MHC isoforms can be coexpressed within the same muscle fibre [18].

A number of recent studies have shown that many fibres in skeletal muscle of both animals and humans may contain a mixture of different MHC isoforms [5, 6, 42, 58, 59]. MHC types I and IIa or IIa and IIb commonly coexist within a single fibre [17, 58, 59]. However, MHC isoform expression does not need to be restricted to only two MHC isoform combinations [37, 62]. For example, Klitgaard *et al.* [37] reported as many as three different MHC isoforms existing within a single fibre in a bodybuilder. Furthermore, four MHC isoforms have been seen in fibres of the extensor digitorum longus muscle of rabbit when chronically stimulated [62]. These differences in MHC character of muscle fibres are not found

histochemically, as this form of discrimination only detects the predominant MHC isoform in a single muscle fibre [17].

In summary, the existence of multiple MHC isoforms in a single muscle fibre in varying proportions suggests extensive differences in myosin ATPase activity and function between fibres. Therefore, the dynamic nature of MHC isoforms makes it difficult to categorise muscle fibres into distinct units. Furthermore, our improved understanding of the limitations associated with the histochemical discrimination of muscle fibre types has three significant ramifications [1]. First, commonly used nomenclatures to describe fibre diversity may neither reflect the spectra of associated MHCs, nor contractile properties of fibres. Second, it suggests that, in the future, MHC isoforms should be measured to detect changes in the contractile properties of skeletal muscle with changes in muscle activity. Third, caution may need to be exercised when interpreting histochemical data.

MYOSIN HEAVY CHAIN ISOFORM ADAPTATIONS TO RESISTANCE ACTIVITY

Muscle Composition in Athletes

The proportions of different fibre types tend to vary between athletic populations. In general, histochemical investigations [14, 19, 44, 66, 71] have usually reported greater strength per unit cross-sectional area in males that present a higher proportion of fast-twitch (FT) fibre types. This may be due to FT fibres having a greater cross-sectional area than slow-twitch (ST) fibres [5, 46, 54, 56] and/or fast MHC isoforms having a greater maximum force than slow MHC proteins [12, 13]. However, cross-sectional comparisons have shown that the percentage of FT fibres may vary largerly (25 to 65%) in the vastus lateralis muscle of various groups of resistance-trained athletes [64].

It has been reported that olympic and power lifters possess a higher proportion of FT fibres than bodybuilders [63]. Furthermore, the percentage of FT fibres was greater in these olympic and power lifters than sedentary control subjects [63]. In contrast, there

is evidence to suggest that bodybuilders may have a similar [4, 32, 37, 46] or even lower [10] proportion of FT fibres than sedentary controls. For example, the proportions of ST, FT oxidative-glycolytic (FTa) and FT glycolytic (FTb) fibres in the vastus lateralis muscle of eight weight-lifters were 37, 54 and 9% respectively [10]. In contrast, Essen-Gustavsson and Tesch [22] showed that the vastus lateralis muscle composition of bodybuilders was 48±8% (ST), 41±13% (FTa) and 10±8% (FTb) after three years of training. Healthy untrained males (n=8) have been reported to have 45±2, 38±2 and 17±1% of ST, FTa and FTb fibres, respectively, in the same muscle [29]. In another study, Klitgaard and colleagues [37] reported that the composition of the biceps brachii muscle fibres of elite Danish bodybuilders (n=4) was similar to sedentary controls (n=4): 51±3 vs 48±2% (ST), 31±6 vs 25±6% (FTa) and 18±5 vs 26±6% (FTb).

Thus, taken together, there appear to be different proportions of FT and ST fibres between muscles of olympic and power lifting, and bodybuilding type resistance-trained athletes. These differences may be a function of training (Table 1), sampling and/or genetic factors. Furthermore, the FT:ST fibre dichotomy is crude as it does not take into account the coexistence of various MHC isoforms within fibres.

Table 1. The comparison of typical bodybuilding and olympic and power lifting type resistance training regimens ✦.

Goal	Bodybuilding activity muscle hypertrophy	Olympic and power lifting activity maximal strength development
Load	70–75% of 1RM*	80–100% of 1RM*
Repetitions	8–12	1–6
Sets	3–5	3–5
Rest between sets (min)	1–1.5	3–5
Repetition of specific exercises (times per week)	2–3	1–2

✦ It should be noted that there is large variability about these "typical" training programmes.

* 1RM, one repetition maximum.

Changes in MHC Isoforms After Training

The data from earlier histochemical studies demonstrated that resistance training did not alter the MHC character of human muscle fibres [16, 19, 26, 34, 65]. It was believed that the predominance of certain fibre types in athletes associated with particular events was genetically determined [38]. However, there have not been many longitudinal studies investigating the effects of resistance training on the histochemical fibre type composition of human skeletal muscle (Table 2). Typically, these studies have shown significant decrements in the percentage of FTb fibres as a consequence of resistance training.

In agreement with this, recent electrophorethic studies on animals and humans indicate that chronic resistance activity can alter MHC isoform composition [2, 15, 24, 32, 33, 60, 61, 70]. The alteration in MHC isoforms with prolonged training appears to be a consequence of changes in specific MHC isoform mRNA levels, coding for each of the isoforms [15, 61].

Typical bodybuilding type resistance activity (see Table 1) may produce a shift from fast MHC isoforms towards slower MHC proteins [10, 21, 32, 36, 48, 52, 63]. In the cross-sectional study of Klitgaard *et al.* [37], bodybuilders (n=4) had a significantly greater proportion of fibres containing only MHC type IIa isoforms (36±4 vs 12±2%), and lower percentage of fibres with a coexistence of MHC types IIa and IIb protein (16±3 vs 34±2%) than sedentary controls (n=4). Interestingly, virtually no fibres contained only MHC type IIb isoforms (1±1 vs 12±1%) in the biceps brachii muscle of bodybuilders [37]. This is in agreement with the Essen-Gustavsson and Tesch [21], and Schantz and Kallman [52] histochemical investigations. Both these studies reported very few histochemically typed FTb fibres in the vastus lateralis (10±8%) and deltoid (12±2%) muscles of bodybuilders, respectively. Thus, the results of these studies suggest that an adaptation to bodybuilding type resistance activity may be an increase in the content of MHC IIa isoforms and decrease in the percentage of MHC IIb proteins in single muscle fibres.

Table 2. Histochemical fibre type distribution (mean±SD) in the vastus lateralis muscle of men and women before and after resistance training studies (FTc and FTab fibres are omitted since they were not always available). The studies are arranged alphabetically and information is given in relation to the number and training history of subjects.

STUDY	SUBJECT HISTORY	LENGTH (weeks)	TRAINING	INTEN-SITY	FREQ. (days/wk)	TIME	FIBRE TYPES (mean±SD) %					
							ST	(P<)	FTa	(P<)	FTb	(P<)
MEN												
Adams [2] (n=13)	Untrained	19	3–5×6–12RM, isoinertial	moderate	2	before after	36.0±4.0 39.0±3.0	ns	46.0±4.0 60.0±3.0	0.05	18.0±3.0 1.0±1.0	0.05
Andersen [5] (n=8)	Trained	12	4×8 RM, 0.52–0.87 rad.sec^{-1}*	low	3	before after	59.1±2.5 58.4±2.8	ns	35.4±2.1 26.7±2.4	0.05	5.5±1.8 14.9±3.9	0.05
Costill [16] (n=5)	Untrained	7	10×6 sec. 3.14 rad.sec^{-1}*	high	4	before after	44.8±5.3 41.8±4.7	ns	30.0±5.1 34.8±3.3	ns	25.2±2.3 23.4±2.4	ns
Kraemer [39] (n=9)	Untrained	12	3×10RM or 5×5RM, isoinertial	high	4	before after	55.2±11.7 55.4±11.5	ns	23.3±11.5 40.5±10.6	0.05	19.1±7.9 1.9±0.8	0.05
Ploutz [46] (n=9)	Untrained	9	3–6×12RM, isoinertial	high	2	before after	40.0±3.0 39.0±3.0	ns	36.0±2.0 33.0±2.0	ns	16.0±4.0 5.0±2.0	0.05
Staron [60] (n=13)	Untrained	8	3×6-8RM or 3×10–12RM, isoinertial	high	2	before after	40.7±7.9 40.0±9.0	ns	31.1±9.1 37.9±9.0	ns	20.7±8.4 9.5±8.9	0.05
WOMEN												
Staron [56] (n=24)	Untrained	20	3×6–8RM, isoinertial	low	2	before after	45.0±14.9 48.7±9.9	ns	32.5±11.1 39.3±7.2	ns	16.2±10.7 2.7±4.3	0.05
Staron [60] (n=8)	Untrained	8	3×6–8RM or 3×10–12RM, isoinertial	high	2	before after	38.8±8.8 44.3±7.6	ns	31.8±5.4 38.8±12.8	ns	21.4±8.1 7.9±6.8	0.05

n — number of subjects; RM — repetition maximum; * isokinetic training; ns — non-significant.

Resistance training aimed to produce muscle fibre hypertrophy may even increase the amount of MHC type I isoforms [23, 33, 49, 60]. For example, 22 weeks of leg press weight training (six sets of 15–20RM, three days a week, one minute recovery between sets) of eight young, previously untrained subjects (four men and four women) significantly increased the percentage of histochemically typed ST fibres (32 vs 47%) in the vastus lateralis muscle [49]. In another study, Jürimäe *et al.* [33] demonstrated that competitive bodybuilders (n=5) presented significantly more type I MHC isoforms than 10 month recreationally resistance-trained (n=5) subjects (31.3±2.7 vs 24.2±4.9%) in the triceps brachii muscle. Therefore, the training regimens between the two resistance training groups were similar in terms of exercises, intensities and recoveries [33]. The results of these studies suggest that prolonged bodybuilding type resistance training may even increase the content of type I MHC proteins. Clearly, this area requires further investigation before any conclusions can be drawn.

Compensatory overload has been used as an animal model for studying adaptive changes to the contractile character of muscle tissue [11, 67]. In this model, the workload of skeletal muscles is increased by either tenotomy of synergistic muscles or ablation of some of the synergistic muscles [11, 67]. Timson [67] argued that compensatory overload model in animals, when continued beyond the initial four to six weeks, was the best of current animal models to compare with chronic (> six months) resistance training in the human. As a result of compensatory overload, muscle enlargement and the nature of skeletal muscle tissue adaptation are apparently similar to bodybuilders [67]. The animal investigations of Ianuzzo *et al.* [30], Oakley and Gollnick [45] and Swoap *et al.* [61] met this time criterion, and suggested that a shift of fast MHC isoforms toward slower MHC proteins was occurring. For example, 40 days of compensatory overload significantly increased histochemically typed ST fibres in the typical slow-twitch soleus (81.7 vs 93.4%) and typical fast-twitch plantaris (10.3 vs 21.5%) muscles in rats [30]. In another study, eight weeks of surgical removal of synergists produced 330 and 82% increase in MHC type I and IIa isoforms, respectively, at the expense of MHC type IIb and IIx proteins in the plantaris muscle in rats [61]. Changes in muscle protein levels were accompanied by significant changes in corre-

sponding mRNA levels [61]. These animal data suggest that chronic resistance activity can alter the proportion of various MHC isoforms. However, the conversion of MHC isoforms towards MHC type I protein appears to be greater in other animals than humans. This may be partially explained by the fact that the remaining muscle in compensatory hypertrophy model has to assume the postural role of soleus muscle (i.e., 42).

In contrast to bodybuilding type resistance activity, Prince and associates [47] argued that typical olympic and power lifting type resistance training (see Table 1) caused a shift from histochemically typed FTa to FTb fibres. In their cross-sectional study, power lifters (n=4) presented significantly more FTb (33.3 vs 26.2%) and fewer FTa (10.5 vs 38.1%) fibres, respectively, than untrained controls (n=5) in the vastus lateralis muscle [47]. While in another study, MacDougall *et al.* [40] compared the proportion of FT fibres of elite bodybuilders (n=7) and previously untrained subjects (n=5), who underwent six months of heavy resistance training of elbow extensor muscles. Interestingly, the percentage of FT fibres was significantly greater in previously untrained subjects than elite bodybuilders (71.0±6.3 vs 66.0±10.7%) [40]. Thus, according to these studies, different resistance training formats may produce differential expression of MHC isoforms. Specifically, olympic and power lifting may shift MHC expression towards faster MHC isoforms than bodybuilding. In agreement with this, Staron and colleagues [55] reported that seven months of inactivity by an elite power lifter reduced the percentage of histochemically typed FTb fibres (55 vs 35%), but increased the percentage of FTa (16 vs 27%) and ST (31 vs 38%) fibres in the vastus lateralis muscle.

Andersen *et al.* [5] presented histochemical and electrophoretic data of muscle homogenates (m. vastus lateralis) indicating a shift towards the MHC IIb isoforms (pre- 0±0%; post- 4.9±2.9%) in national-standard soccer players (n=8) over a 12 week resistance training programme. However, the authors' demonstrated that nearly all histochemically typed FTb fibres of the soccer players displayed coexistence of both MHC IIa and IIb isoforms [5]. The training programme consisted of four sets of eight repetitions of isokinetic loading at low velocity (0.52–0.87 rad.sec^{-1}) three times a week. The rest period between sets was at least five minutes [5]. In addition, there are also animal data suggesting that resistance activity may

alter MHC character in muscle tissue towards MHC type IIb isoforms [70]. In conclusion, certain type of chronic resistance activity may produce a shift towards faster MHC isoforms. Moreover, MHC isoform composition may also be affected by different types of muscle contraction (e.g., isoinertial vs isokinetic).

These data suggest that the occurrence of MHC isoforms in skeletal muscle tissue are affected by different resistance training formats. However, there is a need to determine the exact nature and time intervals of the conversion process during different: 1) resistance training protocols (e.g., bodybuilding, and olympic and power lifting); and 2) types of muscle contractions (e.g., isoinertial, isometric and isokinetic). To date, most longitudinal studies involving histochemical analysis have reported FTb to FTa fibre type transitions after isoinertial training exercises [27, 29, 30, 34, 39, 60, 69]. Several of these investigations have used elements of both "bodybuilding" and "olympic and power lifting" (see Table 1). Specifically, these training programmes have been characterised by reasonably heavy loads (i.e., six to eight RM) and moderate to long recoveries (one to eight minutes) between sets [34, 39, 60, 69].

In summary, the expression of different MHC isoforms in single muscle fibres is not exclusively dictated by genetic factors, but may be affected by chronic resistance activity. Many training protocols (e.g., bodybuilding) appear to produce MHC isoform shifts towards the type I protein. In contrast, other regimens (e.g., olympic and power lifting) may increase the expression of faster MHCs. Though, changes in MHC isoforms may also be a function of training modality and/or detraining.

Time Course of Changes in MHC Isoforms During Training

There have been only a few studies, which have investigated the time course of changes in MHC isoform expression during resistance activity [15, 32, 34, 60]. The transition in MHC composition appears to be rapid. For example, Jürimäe *et al.* [32] and Karapondo *et al.* [34] reported a significant decrease in the MHC type IIb isoform content after only four weeks training. In agreement with these results, Staron and co-workers recently presented data, which showed that resistance activity caused a significant decre-

ment in the MHC IIb isoform content after just five workouts in women (n=8) (18.9±11.9 to 11.4±7.4%) and men (n=11) (18.0±9.0 to 10.8±7.2%) [23, 60]. Furthermore, after four weeks of training (twice a week), women demonstrated a significant increase in histochemically typed ST fibres (38.8±8.7 vs 47.6±9.5%) [60] as well as in electrophoretically separated MHC I isoforms (35.5±11.9 vs 47.1±14.5%) [23]. Thus, it is clear that changes in contractile properties of muscle fibres may occur rapidly in response to resistance training.

A recent study by Caiozzo *et al.* [15] characterised time course shifts in MHC composition at both the protein and mRNA levels in the medial gastrocnemius muscle of rats after heavy-resistance training. During each training day, the rats performed four sets of 10 contractions. MHC type IIb mRNA activity was significantly decreased and MHC type IIx mRNA activity increased after two days of training [15]. Only slight protein changes occurred during the first eight days of training. However, during the training period between the eighth and 16th days significant increments in corresponding type IIx MHC isoform content occurred [15]. The key finding of this investigation was that the molecular composition of rat skeletal muscle responded rapidly to heavy-resistance training.

In summary, changes in MHC composition may take place after only a few training sessions. It is apparent that more rigorous longitudinal resistance training studies are required to determine the time courses for changes in MHC isoforms following a variety of resistance activity regimens.

Possible Mechanisms to Explain Changes in MHC Isoform Composition

Present and previous research consistently reports a significant decrease in MHC IIb isoform (and FTb fibre) content following prolonged resistance training [2, 23, 29, 30, 32, 33, 34, 46, 60, 69]. However, traditionally MHC IIb isoform (and FTb fibre) levels were thought to be critical for optimal performance during near-maximum voluntary efforts [47, 50, 55]. The possible mechanisms to explain losses in MHC type IIb isoform percentage may include

the increased recruitment of FTb fibres and/or increased demands of working muscle (Figure 1).

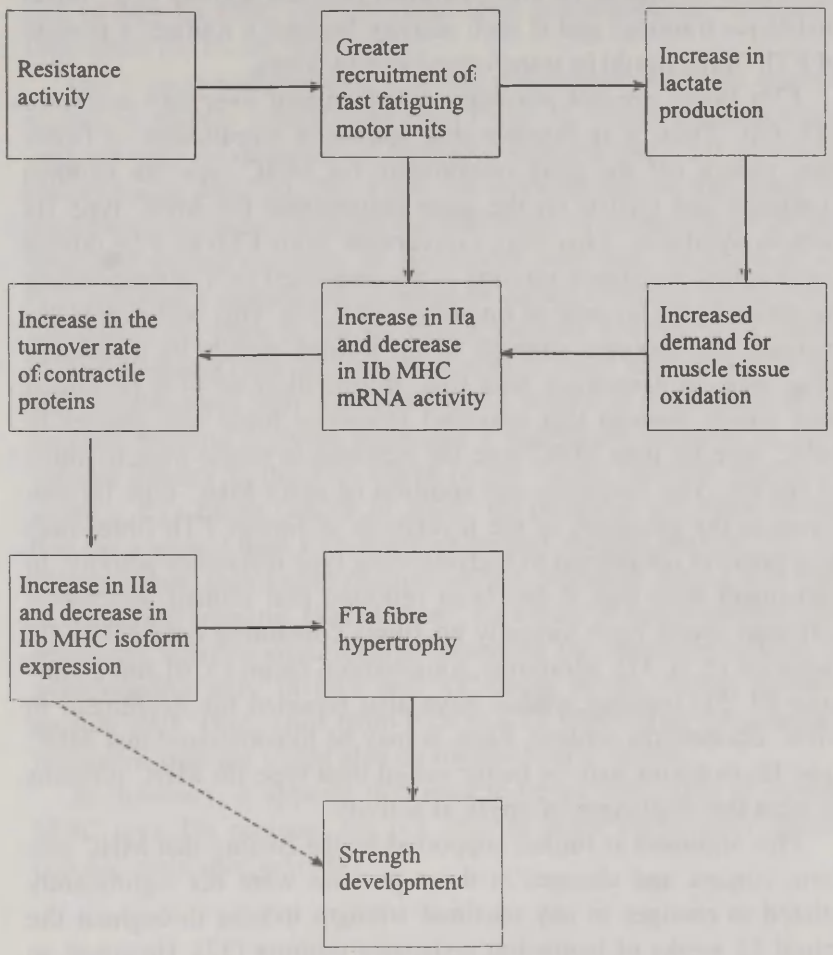

Figure 1. The possible model to explain changes in myosin heavy chain (MHC) isoform composition during the initial 12 weeks of bodybuilding type resistance activity.

A possible explanation for the decrease in MHC IIb isoform content following resistance training was first offered by Goldspink and co-workers [25]. That is, MHC IIb protein is coded by the default gene that provides a pool of fibres that can transform to FTa fibres in response to increased muscular activity. An extension to

this explanation was proposed by Adams *et al.* [2] and Jürimäe *et al.* [32], who argued that fibres containing MHC IIb isoforms were used to meet the demands of unaccustomed physical activity (e.g., novel resistance training) and if such activity became a routine, a portion of FTb fibres would be transformed to FTa fibres.

FTb fibres are not normally active during everyday activities [43, 68]. Thus, it is feasible that increased recruitment of fibres may switch off the gene responsible for MHC type IIb isoform synthesis and switch on the gene responsible for MHC type IIa protein synthesis. This fibre conversion from FTb to FTa during longitudinal resistance training is accompanied by a corresponding increases in the amount of myofibrils [41, 69]. This will eventually increase the maximal strength of the trained muscle by increasing fibre area. In agreement with this, Bottinelli *et al.* [13] presented data which showed that maximal isometric force was greater in MHC type IIa than MHC type IIb isoforms in single muscle fibres of the rat. The synthesis and addition of extra MHC type IIa isoforms to the periphery of the myofibrils of former FTb fibres may be a positive adaptation to bodybuilding type resistance activity. In agreement with this, it has been reported that trained athletes of different sports have virtually no fibres containing only MHC IIb isoforms [5, 6, 37]. Moreover, longitudinal sprint [3, 6] and endurance [9, 51] training studies have also reported the decrement in MHC IIb isoform content. Thus, it may be hypothesised that MHC type IIa isoforms may be better suited than type IIb MHC proteins to meet the challanges of physical activity.

This argument is further supported by the finding that MHC isoform content and changes in these proteins were not significantly related to changes in any maximal strength indices throughout the initial 12 weeks of isoinertial resistance training [32]. However, in another study significant ($p<0.001$) positive correlations were found between MHC type IIa isoforms and various strength indices, and negative correlations between MHC type IIb protein content and maximal isoinertial strength measures [33]. The possible explanation might be that changes in MHC isoform percentage may need longer time than 12 weeks to become a significant factor in specific strength development. Alternatively, the transition in MHC isoform content may be a prerequisite adaptation for other adaptations which underpin strength development. For example, MHC transformation

from IIb to IIa isoforms may precede FTa fibre hypertrophy, which is important factor for strength development to occur. Some researchers have reported greater hypertrophy in FTa fibres (i.e., fibres rich in type IIa MHC isoform) than other histochemically identified fibre types [5, 16, 46, 56].

The typical bodybuilding training programme involves intense repetitive muscle contractions (see Table 1), which are characterised not only by demands of muscular strength but local muscular endurance as well [20, 50]. Thus, the working muscle may need an additional oxidative capacity for buffering of intracellular lactate produced during exercise. In agreement with this, Frontera *et al.* [22] reported a significant increase in citrate synthase activity and capillaries per fibre in the m. vastus lateralis following 12 weeks of bodybuilding type resistance training in older men. While Wang and collaborators [69] reported that 18 weeks of bodybuilding resistance training regimen produced a significant increase in lipid volume density in the FTa fibres of the vastus lateralis muscle in women. FTa fibres (>50% of MHC IIa proteins) are more oxidative (as a group) than FTb fibres in human skeletal muscle [50, 56, 69], and thus, may better oxidize lactate produced during this type of resistance training. Furthermore, 18 weeks of bodybuilding type resistance activity significantly increased the absolute volume of mitochondria only in FTa and ST fibres in the vastus lateralis muscle [69]. Thus, shift from FTb to FTa fibres after longitudinal resistance training might also be metabolically driven.

In summary, it appears that muscle fibres consisting of mainly MHC type IIb proteins are not able to work sufficiently during chronic resistance activity.

CONCLUSION

The limited data available at muscle contractile protein level illustrates that muscle tissue is a very dynamic and sensitive to the influence of resistance activity. As muscle tissue plasticity is evident in early stages of strength training, training programmes for athletes should be developed very carefully. Ideally, resistance training programmes should be similar with an athlete's sporting re-

quirements. Specifically, consideration should be given for resistance training modality when training schedules are being developed. However, there is still a lack of scientific data in the area of muscle contractile protein adaptations to resistance activity. Thus, further research is needed to help athletes and coaches make decisions in relation to resistance activity.

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STABILITY OF SOMATOTYPE IN ENGLISH BOYS AGED 11 TO 15 YEARS USING SHELDON'S PHOTOSCOPIC METHOD: A LONGITUDINAL STUDY

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ABSTRACT

The purpose of this study was to investigate the somatotype stability during growth of a small group of Leeds schoolboys (n=19) through a five year period from 11 to 15 years of age, as they went through adolescent changes. A series of standard somatotype slides of the group were photoscopically analyzed using Sheldon's 7-point rating scales only. Somatotype dispersion distances (SDDs) between individual somatoplots from one year to the next were calculated and analysis of variance showed that the SDDs were non significant. H_0 was therefore accepted, that there is no significant change in somatotype over the five years of boys aged 11 through 15 years in the sample examined. The somatotype dispersion index (SDI) was calculated for each year for the group and results indicated that distribution of somatotypes about the mean are also stable from year to year. Analysis of somatoplots showed that the group moved towards mesomorphy and away from endomorphy and ectomorphy with increase in age, and somatotype distributions were mostly concentrated in the ecto-mesomorph, mesomorph or meso-ectomorph quadrants throughout the study. Analysis of individual somatoplots showed more marked changes in both component magnitude and dominance, or magnitude alone, despite the fact that group values as a whole remained more stable. Cor-

relational relationships between separate components between year one and year five also support the idea of stability (endomorphy, r_s=0.684, $p<0.01$, mesomorphy, r_s=0.476, $p<0.05$ and ectomorphy, r_s=0.734, $p<0.01$). Possible reasons for changes between individuals and stability of the group as a whole, over the five years, are discussed, as are the problems of this particular method of somatotyping, and its suitability for the purpose. Other methods are discussed which might improve rating consistency, reliability and objectivity.

Key words: somatotype, schoolboys

INTRODUCTION

The technique of somatotyping was introduced by Sheldon, Stevens and Tucker [28]. The somatotype represents an attempt to assess the genotype, although to achieve this, it is necessary to utilize measurements of the phenotype [33]. The technique for measuring somatotype was photoscopic, concentrating on body shape not size. After categorizing a series of 4,000 standard photographs of young adult American males, Sheldon *et al.* [28] concluded that all variables could be encompassed within three basic components — endomorphy, mesomorphy and ectomorphy. These were expressed by a series of three sequential numerals always recorded in the same order, each of which expresses the strength of one of the primary components on a 7-point scale, number 1 indicating lease and number 7 maximum preponderance. Endomorphy means a relative predominance of softness throughout the body and a predominance of abdomen over thorax and of trunk over the limbs; mesomorphy a predominance of bone and muscle development, evidence of broad shoulders, wide muscular neck, broad forearm and hands, slender waist and well-muscles lower limbs; ectomorphy a predominance of linearity and fragility, characterized by flatness of the chest and delicacy of the body [24]. Thus, the ratings of each component comprise the individuals somatotype which, by definition, should not change with age [19]. However, although some studies indicate some stability of the components during growth [7, 23] other research suggests somatotype alterations during growth [4, 17, 38]. Newman [20] in a

cross-sectional study of 18–35 year olds, using the Sheldonian system, found definite though limited somatotype changes associated with age; endomorphy and mesomorphy increasing, with ectomorphy decreasing. Tanner [36] commented that "...somatotype is the appearance of the individual when growth ceases, that is about twenty..." and Dupertuis and Tanner [10] pointed out that one of the permises of the constitutional anthropologist is that the components of an individual's somatotype remain constant, at least after skeletal growth is completed. Parizkova and Carter [21] examined the stability of somatotypes of 39 Czechoslovakian boys followed longitudinally from 11 until 18 years. The Heath-Carter anthropometric method was used. The authors concluded that the individual somatotypes of boys changed considerably but the somatoplots for each year were similar. All boys changed their somatotype ratings at least one and 67% changed in component dominance, but the individual differences cancelled each other in group comparisons. They found correlations of 0.79, 0.81 and 0.80 for endomorphy, mesomorphy and ectomorphy respectively between 11 and 15 years of age, all significant at the 0.01 alpha level. According to Heath and Carter [15] the overwhelming evidence is in the direction of plasticity and the inconstancy of physique values.

Petersen [24], who applied Sheldon's criteria to children, is of the opinion that changes in size, proportions and body composition during childhood and adolescence do not fundamentally alter the child's body type. Hammond [12] noted a reasonable degree of constancy of individual body type values over a 13 year period. In children between 5 and 18 years, for example, the correlations between the first and second measurements ranged from 0.65 to 0.92, but tended to be lower during puberty. This assumption of the stability of somatotype was only partially supported by Hunt and Barton [17] who correlated somatotype ratings of 71 boys at prepubertal and subadult ages and obtained correlation values of 0.45 for endomorphy, 0.50 for mesomorphy and 0.71 for the ponderal index. Hence agreement was best for the ponderal index and poorest for the visually assessed first and second components. In a second, small series Barton and Hunt [1] found correlation of 0.54 for endomorphy, 0.51 for mesomorphy and 0.62 for the ponderal index. This series used photoscopic analysis of 62 boys at age 11½ and 16–18, and the authors concluded that body build, as estimated by somatotype, was

fairly unpredictable in adolescence. Hammond [12] showed that, after allowing for general size differences, types similar to the leptosomes, pyknics and eurysomes (these correspond roughly to endo, meso and ectomorphy) in adults can be distinguished in children from aged 5–18 years, and that constancy of type was high.

Relative to somatotype variation during adolescence, the observations of Tanner [36, p. 104] are particularly appropriate:

> "...it would certainly be wrong to leave any impression that the adolescent spurt, whether late or early, causes any radical change in body build; it certainly does not. It adds only the finishing touches to a physique which is recognizable years before. Anyone who has looked at serial pictures of children followed from infancy to adulthood must be impressed chiefly by the similarity the child shows from one age to another. So great is this that there is little doubt that someone used to looking at children's photographs could predict with accuracy the adult somatotype from a picture taken at age 5 or even earlier."

Sheldon's original system [28] involved a combination of anthroscopic analysis using standardized photographs, and classification of the three traits by a 7-point scale combined with a table of the distribution of somatotype according to height-weight ratios. Objections to Sheldon's system were raised, and the 'permanence' of somatotype was questioned [15, 17]. Tanner [35, 36] found Sheldon's anthroscopic method [28] reliable and suggested that none of the proposed modifications constitute an improvement over Sheldon's original work.

The Heath-Carter modification [13, 15] eliminated adjustments for age, opened up the component rating scales at both ends, eliminated the component sum ranging from 9 to 12, and established a linear relationship between somatotype ratings and height-weight ratios. Anthropometric procedures (bone widths, limb girths and skinfold measures) were also added to the anthroscopic procedures to increase the objectivity of the ratings. Somatotype was thus redefined in terms of composition and structure of the body. Other modifications have also been proposed [8, 23]. Much of the past research with somatotype has been conducted on adults. Little work on validation of the procedure for use with children has been carried out [31] although Petersen [24] considered the Sheldonian method to be quite suitable for application to children.

The present study will re-examine the controversy as to whether somatotype is or is not constant or stable during the growth period of adolescence. The null hypothesis, H_0 is that there will be no significant change of somatotype over the years 11 through 15 in the sample examined. A significance level of 0.05 is accepted. A secondary purpose of the study is to assess the usefulness of the method used (Sheldon's anthroscopic method, using rating scales for photography) to establish somatotype.

METHOD

Measurement

A series of photographic slides were collected from a group of 19 boys (one class) from a school in Leeds, U.K. Three standardized photographs were taken for each boy, showing the subject from front, back and side views, in standard somatotype pose. The boys all wore swimming trunks so that bodily contours could be observed clearly. The photographs were taken at annual intervals, for each boy, at mean age 10.6 to 14.6 years, a total of give years. The obtained black and white slides were then viewed using a Kodak Carousel slide projector. The individual physique for each boy, for each year was determined using the criteria for somatotyping proposed by Sheldon *et al.* [28]. Regional somatotypes were rated visually by the author, using the 7-point scoring system. If descriptions for endo, meso or ectomorphy did not fit exactly the perceived appearance of the boy, then it was decided which was the least appropriate and then the other two components were both scored. Regional somatotypes were then added and the totals averaged to give the whole body somatotype number for each of the three components. Each individual's ratings across the five years were completed before continuing to the next individual, in an attempt to be consistent in scoring.

Data Analysis

Having made somatotype ratings of the group of subjects, individual somatotypes across the five years were plotted on somatocharts. A somatochart with superimposed grid which can be used to position an individual somatotype as a somatoplot is exemplified in Figure 1. The origin of the SY co-ordinates is at 4-4-4, in the centre of the somatochart. The formulae for the X and Y co-ordinates are: —

$$X = III - I$$

$$\text{and } Y = 2II - (I + III)$$

where I = endomorphic component
II = mesomorphic component
III = ectomorphic component

(Ross & Wilson [27]; Ross, Carter & Wilson [26]).

If the somatotypes to be plotted are in whole-unit numbers, as in this study, then there is no particular plotting problem, but the formulae are particularly necessary when the somatotype to be plotted is not on the somatochart (e.g. if somatotype contains half-values). Because the somatochart is based on a mathematical relationship between the three component values, then the plotting formula can be used for positioning any somatotype rating (regardless of the method used to assess somatotype). The XY co-ordinate values are also necessary when calculating the somatotype dispersion distance (SDD). This was the second step of data analysis and represents the distance between any two somatoplots (X_1, Y_1,) and (X_2, Y_2). The SDD was calculated as follows, and is expressed in Y-axis units: —

$$SDD = \sqrt{(KX_1 - KX_2)^2 + (Y_1 - Y_2)^2}$$

Where K = 1.732 (constant factor that converts X values to Y units)
(X_1, Y_1) are co-ordinates of one somatoplot
(X_2, Y_2) are co-ordinates of the other somatoplot

(Ross & Wilson [27]; Ross *et al.* [26])

The formula is an application of the Pythagorean theorem. The mean somatotype ($\bar{S}$) for each component was obtained by finding the sum of each of the components divided by the number of subjects in the sample, each component being treated independently.

The next stage of data analysis was to calculate the somatotype dispersion index (SDI) for the group of subjects for each of the five years. The SDI is the mean of the SDDs for each somatoplot about the somatoplot for the calculated $\bar{S}$ of a distribution (Ross & Wilson [27]).

Hence, $$SDI = \frac{\sum\sqrt{(KX_1 - K\bar{X})^2 + (Y_1 - \bar{Y})^2}}{n}$$

Where K = 1.732
X_1 Y_1 are co-ordinates of the individual somatoplots
X Y are co-ordinates of the mean somatoplot for the group
n = number of subjects

Thus the SDD quantifies the distance between somatoplots and SDI describes the dispersion about a mean somatoplot (the average deviation of the somatoplot). Both quantities render parametric data. For accurate data analysis and also for plotting somatocharts, computer programs are available, written in FORTRAN (SDI and S PLOT) and can be found in Carter (1975), where further parametric and non parametric analyses of somatotype data are described.

One-way related analysis of variance was computed upon the SDD data across the five years of the study.

Finally, the somatotypes were disassembled and individual components treated independently for the first and last years of the measurement, and Spearman's rank-order correlation coefficients calculated between these two years for each of the three component ratings.

RESULTS

Individual Somatotype Components

These components for all subjects across the five years are shown in Table 1. The greatest change in endomorphy between ages 11 and

Table 1. Somatotype ratings for nineteen subjects over five years, mean somatotype ratings ($\bar{S}$), mean somatotype co-ordinates (X, Y) and somatotype dispersion indices (SDI)

Subject Code (n = 19)	Age 11 1975	Age 12 1976	Age 13 1977	Age 14 1978	Age 15 1979	S	(X, Y)
01	1–4–4	1–5–5	1–6–5	1–6–4	1–6–5	1–5.4–4.6	(3.6,5.2)
03	2–5–4	1–6–4	2–5–4	1–5–3	2–4–4	1.6–5–3.8	(2.2,4.6)
10	2–4–4	2–4–4	2–5–4	2–5–4	2–5–4	2–4.6–4	(2,3.2)
11	3–6–2	2–7–2	3–6–4	4–6–2	3–7–2	3–6.4–2.4	(–0.6,7.4)
12	2–3–6	2–4–5	2–4–5	2–5–5	1–5–4	1.8–4.2–5	(3.2,1.6)
15	3–5–3	2–5–3	2–5–3	1–6–3	1–6–4	1.8–5.4–3.2	(1.4,5.8)
17	1–2–6	1–3–6	1–3–6	1–4–6	1–5–5	1–3.4–5.8	(4.8,0)
18	6–3–1	6–4–1	6–5–1	4–6–1	3–6–1	5–4.8–1	(–4,3.6)
20	3–5–2	3–5–3	4–6–1	4–5–1	5–5–1	3.8–5.2–1.6	(–2.2,5)
21	1–3–4	1–3–4	1–6–4	1–6–5	1–6–3	1–4.8–4	(3,4.6)
26	5–3–2	4–4–2	4–3–2	4–4–2	2–5–2	3.8–3.8–2	(–1.8,1.8)
27	2–6–4	2–6–4	1–7–4	2–7–4	2–6–4	1.8–6.4–4	(2.8,7)
30	6–2–1	6–2–1	6–2–1	5–4–1	5–5–1	5.6–3–1	(–4.6,–0.6)
31	2–5–5	2–6–5	1–6–4	1–6–4	1–6–3	1.4–5.8–4.2	(2.8,5)
32	2–3–5	2–3–5	2–4–5	2–4–5	2–5–5	2–3.8–5	(3,0.6)
34	2–3–6	1–4–5	1–4–5	1–4–5	1–4–5	1.2–3.8–5.2	(4,1.2)
35	2–4–5	2–4–5	1–4–6	2–5–5	2–6–5	1.8–4.6–5.2	(3.4,2.2)
36	2–5–5	1–5–5	1–5–4	1–6–4	1–6–3	1.2–5.4–4.2	(3,5.4)
38	2–3–5	2–3–5	2–4–4	3–5–4	2–5–4	2.2–4–5.4	(3.2,0.4)
$\bar{S}$	2.6–3.9–3.9	2.3–4.4–3.8	2.3–4.7–3.8	2.2–5.2–3.6	2–5.5–3.4		
(X, Y)	(1.3,1.3)	(1.5,3.7)	(1.5,3.3)	(1.4,4.6)	(1.4,5.6)		
SDI	5.44	4.83	5.08	5.07	4.5		

15 were subjects 18 and 26 who decreased by 3 units, the majority of the remaining differences were zero (50% of the sample) or minus one unit (25% of sample). For mesomorphy, 30% increased by one unit, 30% by 2 units and 20% by 3 units. The ectomorphy component showed the least change, the differences being plus or minus one unit for 35% of the sample, no change for 45% and 15% losing 2 points. Thus ectomorphy was the most stable component, 80%

changing by a maximum of one unit, followed by endomorphy, with 75% showing one unit maximum of change, and with mesomorphy only 50% changing by a maximum of one unit. The strength of the relationship between the three components between the first and last years of the study (1975 and 1979) for the sample were found to be as follows — for endomorphy r_s — 0.684 ($p<0.01$) mesomorphy, r_s — 0.476 ($p<0.05$) and ectomorphy, r_s — 0.734 ($p<0.01$). Group mean somatotype component ratings over the five years are displayed in Figure 1. For both endo and ectomorphy there are very

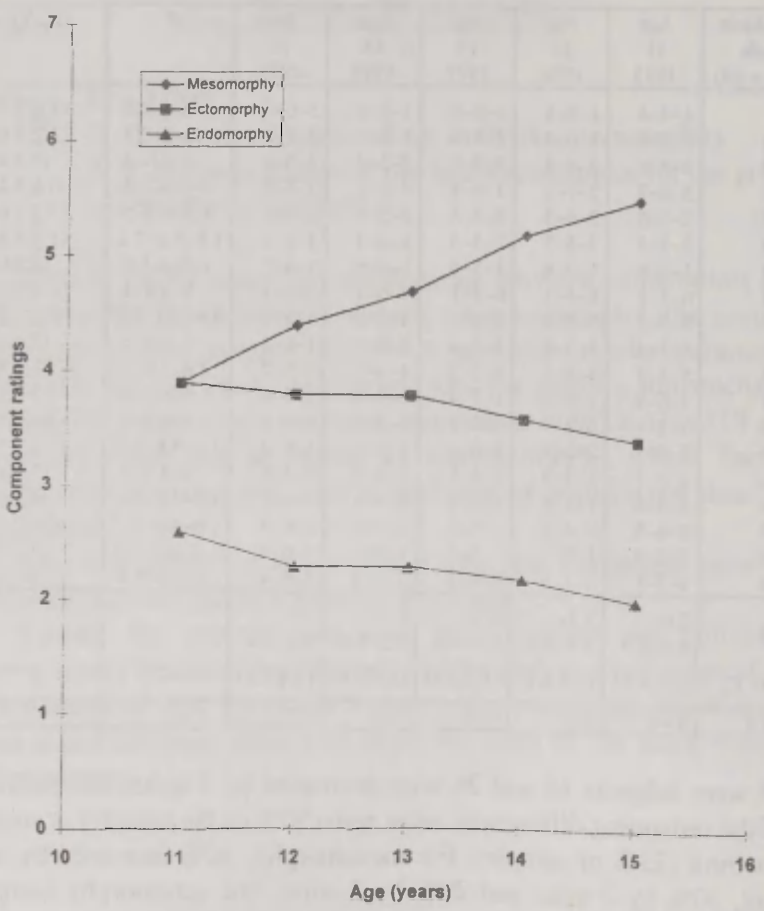

Figure 1. Somatotype component rating of boys followed for five years (means)

small differences between the means, especially for adjacent ages, the overall trend being a slight decrease in the mean from ages 11 through 15. There is a consistent increase in mesomorphy over this period both between adjacent years and as a whole. Thus the group of boys appeared to become more mesomorphic and less endo and ectomorphic at age 15 than at age 11.

Somatotype as a whole

Individual somatoplots of all members of the group, from first to last year are shown in Figure 2. The distribution appears to be concentrated on the right-hand side of the mesomorph (vertical) axis, in the ecto-mesomorph, mesomorph or meso-ectomorph quadrants.

Mean somatotypes across the five years for the group are also shown in Figure 2 as the triangles. This corroborates the information shown in Figure 1.

Table 2. Somatotype dispersion distances (SDD) between individual somatoplots from year to year

Subject Code (n=19)	Ages 11–12	Ages 12–13	Ages 13–14	Ages 14–15	Σ SDD
01	1.99	2	1.99	1.99	7.97
03	3.46	3.46	2	4	12.92
10	0	2	0	0	2
11	3.46	5.29	5.29	3.46	17.5
12	3.46	0	2	2	7.46
15	1.99	0	3.46	1.9	7.44
17	2	0	2	3.46	7.46
18	2	2	5.29	1.99	11.28
20	1.99	5.99	2	1.99	11.97
21	0	6	1.99	2.73	10.72
26	3.46	2	2	5.29	12.75
27	0	3.46	1.99	1	6.45
30	0	0	5.29	2	7.29
31	2	2	0	1.99	5.99
32	0	2	0	2	4
34	4	0	0	0	4
35	0	3.46	3.99	2	9.45
36	1.99	1.99	2	1	6.98
38	0	3.46	1.99	1.99	7.44
X	1.67	2.37	2.28	2.15	8.48
Y	1.46	1.95	1.72	1.27	3.69

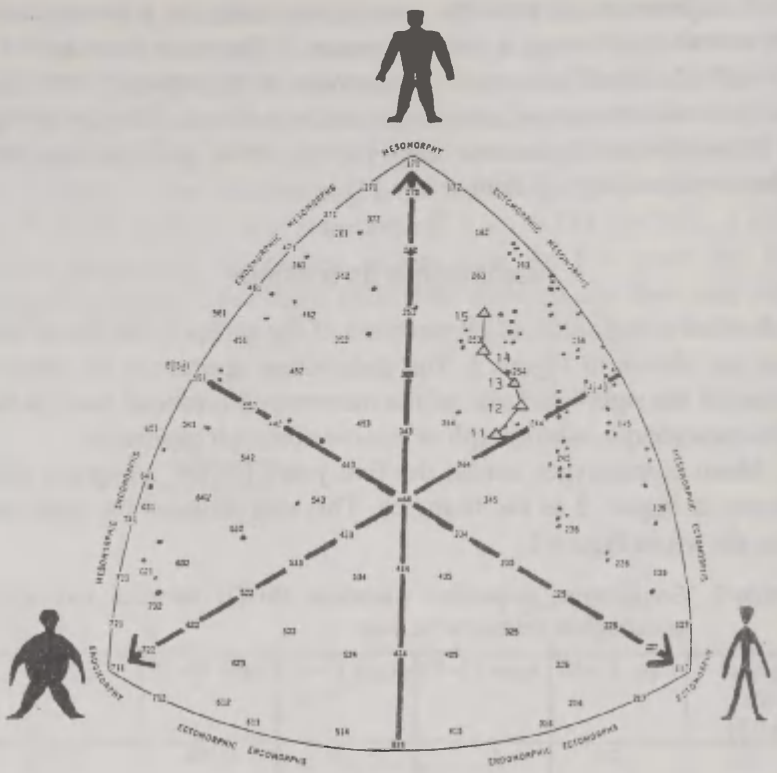

Figure 2. Individual somatoplots over the five years. Mean somatotypes are shown as triangles.

The migratory distance (the sum of SDDs between the successive somatoplots from the first to the fifth year range) from a low of 2 to a high of 17.5 with a mean of 8.48. Because the migratory distance is a scalar and not a vector quantity, the individual somatocharts must be examined to determine if changes cancel each other, or if somatotype dominance is changed. The greatest mean difference moved by the sample was between ages 12 to 13 ($\bar{X}$ = 2.37, SD = 1.95), and the least distance between ages 11 to 12 ($\bar{X}$ = 1.67, SD = 1.46). Thus Figure 2 and analysis of SDDs indicate that the mean somatotypes year to year are relatively stable, and the SDī values in Table 1 indicate that distributions of the somatotypes about the mean are also stable.

DISCUSSION

These results indicate that the null hypothesis must be accepted and that there is no significant change of somatotype for the group from age 11 through to age 15. This means that there was considerable stability of somatotype during the adolescent period. Probably the most outstanding characteristic of the analysis is the contrast between the group statistics and the individual patterns. The means for the whole group for separate components, and in their somatochart distributions, show relatively little change; however, examination of individual patterns over the five years indicates that some individuals show considerable change. The individual changes are in random direction in relation to each other, thereby cancelling out in group analysis. Inter age correlations between the first and fifth year for single components support the findings of Hunt and Barton [17], although they used ponderal index values for the ectomorphy score, but do not support the findings of Parizkova and Carter [21], although the latter found the coefficients dropped to 0.52, 0.71 and 0.66 respectively after eight years. Thus there seems to be fair agreement between different studies that somatotypes of boys during adolescent may change, although the group as a whole may show more stability, for reasons previously discussed and evidenced by the SDD and SDI values. This finding agrees with Parizkova and Carter [21] who also found stability of mean somatotypes from year to year and stability of somatotype distributions about the mean. While there is little indication of group change from year to year, some individuals change somatotype markedly during adolescence. Claessens *et al.* [7] also found considerable stability of somatotypes during adolescence, using Sheldon's anthroscopic method, and concluded that total body shape can be fairly well predicted although mean values of individual components fluctuate somewhat, particularly the increase in mesomorphy, consistent with the development of muscle mass during adolescent growth in boys, as was found in this study. Mean component values for the group across the five years were 2.3–4.7–3.7 which if fairly close to the findings of Slaughter and Lohman [31] who quote 3.8–4.2–3.5 for a group of 45 boys aged 7–12 years, and Morton (1967) who found means of 3.4–4.2–3.2 for a group of boys aged 8–12. However, this sample was small (n = 19) and not randomly selected, as was shown by the

skewed distribution of somatoplots on the somatocharts, so was not necessarily representative of a population of boys of such an age.

Hammond [12] offered another explanation for constancy of physique found in his determination of physical type in children. He considered that it was due not so much to continuing to develop along characteristic type lines, but because growth is mainly general and therefore fails to upset the type pattern once found.

The individual somatoplots illustrate the variability and magnitude of change of the individuals. However, some generalities can be observed from principally Figure 1, that the group became more mesomorphic and less endo and ectomorphic over the period 11 years to 15 years, similar to the trend found for comparable age groups by Parizkova and Carter [21] and Toteva [38]. Practically all skeletal and muscular dimensions take part in the adolescent growth spurt at around ages 12–15 in boys when they acquire the wide shoulders and muscular neck of the man. At this time limb fat in boys thins out in parallel with the adolescent height spurt [36]. If mesomorphy is a measure of underlying amounts of muscle tissue, then the findings of mesomorphic increase is consistent with the development of muscle mass and structural width changes during adolescence. However, Claessens *et al.* [7] suggested that, particularly for mesomorphy as measured by the anthroscopic technique, and the second component measured by the Heath-Carter anthropometric method, these two methods do not measure the same underlying factors, thus the two methods cannot be considered to be equivalent. Carter and Heath [3] on the other hand, compared ratings using Sheldon's photoscopic ratings and Heath-Carter photoscopic ratings in a sample of young adults and found that, for males, there were no differences between mean somatotypes or between component means. They did find differences for women and suggested this might be because of the lack of rating criteria for females in the Sheldon method. Further, Slaughter and Lohman [32] found little association between lean body mass and mesomorphy or the second component. Thus the increase in mesomorphy found during adolescence may or may not represent increase in muscle mass, depending on what it is actually measuring. Another possible reason for the lack of stability of the mesomorphic component found in this study could be inconsistency in rating by the author as several researchers have found mesomorphy to

be the most difficult component to rate and ectomorphy the easiest [7, 17, 35].

Endomorphy as measured by Sheldon's method and the first component of the Heath-Carter method has been found to reflect body fatness [32], hence the slight decrease over the five years of this study would be consistent with normal changes of adolescence [36].

For the above reasons, explanations of changes in somatotype during adolescence being linked to growth and change in body composition should be treated with caution, particularly as the Sheldonian method was used in this study and this has been found to be less closely related to body composition than the Heath-Carter method [32]. Thus the conceptual differences between different methods of somatotyping has to be considered in the interpretations of the findings of this study. The Sheldonian somatotype is, be definition, unchanging throughout adolescence or at any other time as it is revealed in its completed form when the subject has reached young adulthood [28]. In contract, the Heath-Carter technique is unconcerned with the underlying continuity of body build and is simply a system for combining various measurements. Hence it might be useful to consider whether the Sheldonian system is an appropriate one to use to investigate growth changes in physique during adolescence, or whether the Heath-Carter anthropometric method would be better, as it seems to be more closely linked with underlying structure. In this study, of course, there was no option as it was retrospectively based on photographs.

Methodological problems were involved with this study, where only Sheldon's rating scales were used and height and weight data were not available, so the ponderal index (PI) could not be calculated. Carter, (1975) suggests this can improve the reliability of photoscopic ratings. The PI can be consulted to determine which somatotypes are possible for the calculated PI, and usually several of the alternative somatotypes can be disregarded [25].

The reliability of the technique has to be considered. Tanner, [35] stated that trained observers agree on their ratings on the 7-point scale in 90% of instances. He also suggested that minimal experience should consist of a few weeks training in the laboratory, performing over one hundred practice ratings on known reference material. This involved inspection of standardized photo-

graphs and comparison of key files. Although long training is required to become an expert, Damon and Bleibtreu [9] suggest that with only a few hours instruction or even by reading Sheldon's text, an untrained observer "can establish component dominance, but he cannot rate consistently to within a point on a 7-point scale.". Sheldon *et al.* [28] found a high correlation ($r = 0.92$) between rates for the three components and Hunt and Barton, [17] reported correlations between 0.66 and 0.89 for the first and second components. Intra-rater reliability has been found to be higher than inter-rater reliability [14].

Use of such rating scales for the photoscopic method implies a degree of subjectivity. As Parnell, [22] points out "if somatotypers agree, it will mean that they have learnt to sing in harmony but their song does not thereby become a science, it remains an art."

Subjectivity can be lessened and objectivity increased by inclusion of anthropometric measures, as suggested by Carter, (1975). Hunt and Barton, [17] outline the need to arrange photographs in rank order according to their degree of manifestation of one component at a time.

A further aspect of this study, using the rating scales, involved the use of language which may be applicable to adults but is difficult to apply to children — terms such as 'rugged', 'well-muscled', and 'deep-chested' do not seem particularly appropriate, increasing the difficulty of rating the mesomorphy component.

Very few illustrations of childrens somatotypes have appeared in the literature. There are several childrens photographs in Parnell [23] and 560 in Petersen's Atlas [24]. Petersen's ratings are supposed to be based on Sheldonian criteria, although Sheldon has never published any criteria for children (Carter, 1975), making the atlas difficult to use as a rating reference.

Another factor linked with rating error in this study was the technique of identifying the least likely designation and scoring for the other two, which occurred on a number of occasions. This technique could lead to artifically high average scores for components, and product totals of more than 12 and less than 9, which is not permissible in the Sheldonian rating system (Carter, 1975). Initial rounding of average component scores is a source of systematic error. Use of a computer programme for raw data handling would aid this problem.

Despite such problems associated with photoscopic rating, Carter (1975) states that a photograph is essential for somatotyping children and Parnell [23] points out that the photograph cannot be discarded but that anthropometric estimates would "sharpen" the definition. Dysplasias are also easily recognised in the photograph. If photographs are used for any photogrammetric analysis, then the correct post is essential. Dupertuis and Tanner [10] stated that the largest error in an otherwise reliable technique is due to inaccurate posing of subjects. Other advantages of photoscopic technique are that outlines do not move as they are measured [34] and photographs are quick to take and provide many measurements, also give a permanent record of the subjects actual appearance [22].

Unlike the criterion of constancy for the Sheldonian systems, the more sensitive the physique rating system is to changing shape and composition, the greater is it's applicability to fitness and performance studies [2]. Somatoplots lend themselves to non parametric comparative techniques for problems relating physique with sex, age, growth, maturity, disease, obesity, temperament, behaviour or performance phenomena or personality (Hebbelinck, Duquet & Ross [16]; Philips & Hornak [25]; Tanner [36]; Malina & Rarick [19]; Tancred & Tancred [33]; Lowrey [18]; Falls, Humphrey, Sills & Mitchein [11]; Caskey & Felker [6]; Carter, Stepnicka & Clarys [5] Damon *et al.* [9]; Barton & Hunt [1]).

CONCLUSION

There have been many questions regarding somatotype technique, methodology and findings, particularly about permanence or somatotype, reliability of ratings and subjective factors in anthroscopy. These issues have been considered above.

The overall finding of the study indicate that somatotype of boys during adolescence remains fairly constant when group differences and somatotype as a whole are examined. However, when individual differences are examined, the picture is less clear, and differences in somatotypes in some individuals change much more than others.

It is important to distinguish between the Heath-Carter method which, although using the same terminology as the Sheldonian method (used in part in this study) differs in concept and method a principal one being that somatotypes are rated as present, phenotypic expressions, and permanence is not assumed. Both approaches have been in evidence in studies discussed here. As methods are not entirely interchangeable, this should be noted in analysis of data from the literature.

Whether or not stability in somatotype is found during growth appears to be partly dependent on which methods is used to assess somatotype.

Problems of reliability and subjectivity have been discussed and suggestions made for improving rating consistency. Inclusion of anthropometric data would increase objectivity of ratings.

The use of somatotype dispersion distance and the somatotype dispersion index in analysis of data has proved of some use in quantifying change in individuals and groups.

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THE RELATIONSHIPS BETWEEN PHYSICAL FITNESS AND PHYSICAL ACTIVITY IN CHILDREN

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ABSTRACT

The aim of this study was to investigate the relationships between health related fitness and physical activity in 10, 12, 13-year-old boys and girls. The validity of the Godin and Shephard [11] questionnaire was also examined. The subjects were 174 10, 12 and 13-year-old children. The physical fitness was assessed by EUROFIT test battery and physical activity by physical activity recall modified by Godin and Shephard [11] by which children reported how much time they spent in total and separately on low, moderate or vigorous physical activities. For validity testing of the questionnaire the energy expenditure measured with help of Caltrac accelerometer and questionnaire were compared. Our results indicate that the questionnaire is a valid self-report for children ($r = 0.730–0.939$). In 10 and 13-year-old boys the results of the 7 fitness tests were significantly ($p<0.05$) related with total physical activity. The stepwise multiple regression analysis indicated that total and vigorous physical activity accounted for 15–59% of variance of the endurance shuttle run. The relationships with other components of fitness (speed, strength etc.) were not so clear.

Key words: physical fitness, physical activity, children

INTRODUCTION

The use of meta-analysis was leading to the conclusion that there is an inverse relationship between physical activity and coronary heart disease (CHD) in over two-thirds of the studies in adults [19]. Epidemiological studies suggest that risk factors for CHD can be identified in children and that most of the risk factors track into adult life [9]. Physical inactivity is a risk factor for CHD in adults and physical activity behaviors may track from childhood into adulthood [6]. However, analysing the descriptive epidemiology of physical activity in adolescents, Pate *et al.* [18] concluded that a substantial number of males, and the majority of females, are not meeting the guideline for moderate to vigorous physical activity.

The relationship between aerobic fitness and health is well established in adults but not in children [4, 29]. In the review of Morrow and Freedson [15] it was concluded that there are mostly low to moderate relationships between physical activity and aerobic fitness in this population. Only few studies [24] have quantified the influence of physical activity in other components of health-related physical fitness (muscular endurance, muscular strength, body composition and flexibility).

There are two major categories of methods for establishing physical activity [7]: 1. methods by which the physiological responses to activity, e. g. oxygen uptake, heart rate, etc., are obtained, 2. observational methods by which the physiological response is estimated, via direct or indirect recording of movements (diary method, questionnaires). There are several problems with all of those methods, and no one measure appropriates for all purposes. In epidemiological studies the questionnaires are very popular. Consequently, several questionnaires have been developed and extensively analysed [13]. There is no "gold standard" by which physical activity questionnaires, used in population based research, can be validated. The traditional approach to validate activity questionnaires has involved comparing the questionnaires to objective measures of physical activity, such as oxygen consumption, activity monitors etc. One of the most popular is the simple quantitative questionnaire by Godin and Shephard [11] whose reproducibility is relatively high for example in boys and girls of grades 7 to 9 [10]. However, using questionnaire the children tend to overestimate the time spent on activity [26].

It appears that the relationships between children's health-related fitness and physical activity of different intensities are poorly studied. The aim of this study was to investigate the relationships between health related fitness and physical activity in 10, 12, 13-year-old boys and girls. A second purpose was to assess the validity of the Godin and Shephard [11] questionnaire.

METHODS

Subjects

The subjects of this study were 174 10, 12 and 13-year-old Estonian elementary school children (94 boys and 80 girls). All subjects were healthy and participated in the regular school physical education lessons twice a week. The parents of the children gave permission for testing. The children were instructed not to modify their usual daily physical activity patterns during testing days.

Testing procedures

Body height was measured using Martin's metal anthropometer to the nearest 0.1 cm, body mass was measured to the nearest 0.05 kg using medical scales, and the body mass index (BMI) was calculated (body mass in kg/height in m^2). Pubertal stages were determined according to the criteria of Tanner [33]. Physical fitness was assessed using EUROFIT testing protocols [8] by 9 tests, including the Flamingo balance, sit-and-reach, 10×5 m shuttle-run, handgrip strength, standing broad jump, bent-arm-hang, plate tapping, sit-ups during 30 s, and 20 m endurance shuttle-run. Fitness testing was realized at schools in the morning and separate stations were set up for each measurement.

Physical activity was assessed by physical activity recall modified by Godin and Shephard [11]. In the present study during one week (7 days), the children reported in the written form with the help of their parents in the evenings of every day how much time (in minutes) they spent on total physical activities (TPA) separately classified as low (LPA, 3 MET-s), moderate (MPA, 5 MET-s) or

vigorous (VPA, 9 MET-s). Classification of activities in each activity category was completed using the Compendium of Physical Activities designed by Ainsworth *et al.* [1]. Examples of activities most frequently used by the children (sports, home and leisure activities etc.) were selected and provided in the written form. Special attention was given to moderate to vigorous physical activities such as games and other sport activities.

Validity of the Godin and Shephard [11] questionnaire

Twenty four children were divided into the following groups: 10, 12 and 13-year-olds. In each age group there were 4 boys and 4 girls. The CALTRAC accelerometer (HEMOKINETICS, INC., MADISON, WI) was used to assess energy expenditure in kilocalorie values. The CALTRAC monitor was placed on the child in the morning (7–8 a. m.) and returned before the bed time (10–11 p.m.). The children used CALTRAC on two schooldays (Thirsday, Friday) and two weekend days (Saturday, Sunday) and the mean result of four days was used. Physical activity was assessed on the same days as CALTRAC was used by physical activity recall modified by Godin and Shephard [11].

Statistical analysis

The SAS statistical package was used for data analysis [27]. Descriptive statistics included means (M) and standard deviations (±SD). One-way ANOVA was used to test the differences between groups and zero-order correlations were calculated between physical activity and physical fitness scores. Multiple regression analysis was performed with total, low, moderate and vigorous physical activities as independent variables and physical fitness tests as dependent variables in each age and sex group. The validity of the Godin and Shephard [11] questionnaire was examined using a Pearson product moment correlation. The level of $p \leq 0.05$ was considered significant.

RESULTS

Physical characteristics

Tables 1, 2 describe the physical characteristics of the boys and girls groups. There was a significant increase in body height and mass in all age groups, but the BMI increased significantly ($p<0.001$) in girls between 12 and 13 years of age (Table 3). All 10-year-olds were in the Tanner stage 1, three 12-year-old boys and 5 girls were in stage 2 and six 13-year-old boys and 15 girls were in stage 2 or 3.

Table 1. Mean (M) and standard deviation (SD) of physical growth characteristics, physical fitness tests and physical activity scores in boys

	10-year-old (n=28)		12-year-old (n=35)		13-year-old (n=31)	
	M	±SD	M	±SD	M	±SD
Growth characteristics						
Body height (cm)	140.3	7.0	150.6	5.2	154.9	6.9
Body mass (kg)	34.8	5.8	39.0	4.0	43.2	5.3
BMI (kg/m^2)	17.6	1.9	17.1	1.3	17.9	1.6
Physical fitness tests						
Balance (x)	13.6	6.0	13.0	5.3	12.9	5.1
Sit-and-reach (cm)	17.1	5.1	18.6	7.2	16.4	6.7
10×5 m shuttle-run (s)	22.7	2.1	21.7	1.7	22.1	1.8
Handgrip strength (kg)	14.6	5.5	20.0	5.4	25.5	7.8
Standing broad jump (cm)	163.0	15.2	175.4	17.9	184.3	16.0
Bent-arm-hang (s)	27.3	16.2	27.0	18.2	30.3	16.3
Plate tapping (s)	15.1	2.4	13.9	1.3	12.9	1.8
Sit-ups (x)	22.0	4.1	24.2	5.2	25.9	4.3
Endurance shuttle-run (min)	5.2	1.4	5.6	1.2	5.7	1.3
Physical activity scores						
TPA (kcal/24h)	671.0	324.7	734.9	404.3	708.0	445.9
LPA (kcal/24h)	242.6	101.8	315.9	141.3	315.1	128.0
MPA (kcal/24h)	207.6	158.4	192.6	128.5	131.3	126.6
VPA (kcal/24h)	220.8	162.0	226.4	212.0	261.7	283.0

TPA — total physical activity
LPA — low physical activity
MPA — moderate physical activity
VPA — vigorous physical activity

Table 2. Mean (M) and standard deviation (SD) of physical growth characteristics, physical fitness tests and physical activity scores in girls

	10-year-old (n=25)		12-year-old (n=28)		13-year-old (n=27)	
	M	±SD	M	±SD	M	±SD
Growth characteristics						
Body height (cm)	139.5	6.0	153.3	7.8	160.5	6.2
Body mass (kg)	33.7	7.0	42.8	8.5	49.7	10.9
BMI (kg/m^2)	17.2	2.4	17.8	2.4	19.1	2.9
Physical fitness tests						
Balance (x)	10.9	5.3	9.6	4.0	12.0	4.0
Sit-and-reach (cm)	20.6	4.9	23.0	5.0	22.6	5.4
10×5 m shuttle-run (s)	23.5	1.6	22.4	1.9	23.0	1.6
Handgrip strength (kg)	10.4	3.9	16.3	5.5	20.6	7.2
Standing broad jump (cm)	145.0	18.3	161.5	13.2	167.2	13.3
Bent-arm-hang (s)	14.1	10.6	16.7	10.9	6.9	7.3
Plate tapping (s)	15.9	2.5	12.8	2.7	13.2	1.8
Sit-ups (x)	20.2	4.9	21.7	5.8	21.2	3.2
Endurance shuttle-run (min)	4.4	1.3	5.2	1.1	4.8	1.3
Physical activity scores						
TPA (kcal/24h)	461.4	225.1	430.7	152.5	338.1	132.8
LPA (kcal/24h)	133.7	80.8	223.6	90.5	146.5	78.8
MPA (kcal/24h)	154.8	154.5	75.9	66.2	80.0	76.3
VPA (kcal/24h)	172.8	97.0	130.4	76.2	111.5	38.3

TPA — total physical activity
LPA — low physical activity
MPA — moderate physical activity
VPA — vigorous physical activity

Physical fitness

The mean results are presented in Tables 1, 2. The results of the boys were significantly (Table 3) better than those of girls of the same age (except balance and sit-and-reach).

Physical activity

The results of the Godin and Shephard [11] questionnaire, which are demonstrated in Tables 1, 2, 3, indicate that the TPA of the boys is significantly ($p<0.05$–0.001) higher than that of the same

aged girls. Only in the 13-year-old boys the LPA ($p<0.02$) and in 12-year-olds the MPA ($p<0.001$)is higher than in the girls. The VPA in all age groups of boys is significantly ($p<0.01$–0.001) higher than in girls.

Table 3. Levels of significance between groups for physical growth characteristics, physical fitness tests and physical activity scores

	1–2	3–4	5–6	1–3	1–5	3–5	2–4	2–6	4–6
Body height	NS	NS	<0.01	<0.001	<0.001	<0.01	<0.001	<0.001	<0.001
Body weight	NS	<0.05	<0.01	<0.01	<0.001	<0.001	<0.001	<0.001	<0.02
BMI	NS	NS	NS	NS	NS	NS	NS	<0.01	<0.001
Balance	<0.01	<0.01	NS	NS	NS	NS	NS	NS	<0.05
Sit-and-reach	<0.02	<0.01	<0.001	NS	NS	NS	NS	NS	NS
10×5 m shuttle-run	NS	NS	<0.05	<0.05	NS	NS	<0.05	NS	NS
Handgrip strength	<0.01	<0.01	<0.01	<0.02	<0.001	<0.01	<0.02	<0.001	<0.02
Standing broad jump	<0.001	<0.001	<0.001	<0.01	<0.001	<0.05	<0.001	<0.001	NS
Bent arm hang	<0.01	<0.01	<0.001	NS	NS	NS	NS	<0.01	<0.001
Plate tapping	NS	<0.05	NS	<0.02	<0.001	<0.01	<0.001	<0.001	NS
Sit-ups	NS	NS	<0.001	<0.05	<0.01	NS	NS	NS	NS
Endurance shuttle-run	<0.05	NS	<0.02	NS	NS	NS	<0.02	NS	NS
TPA	<0.05	<0.001	<0.001	NS	NS	NS	NS	<0.05	<0.05
LPA	NS	NS	<0.02	NS	NS	NS	<0.01	NS	<0.001
MPA	NS	<0.001	NS	NS	NS	NS	NS	<0.05	<0.05
VPA	<0.01	<0.001	<0.001	NS	NS	NS	NS	<0.01	NS

1 — 10 year old boys
2 — 10 year old girls
3 — 12 year old boys
4 — 12 year old girls
5 — 13 year old boys
6 — 13 year old girls
NS — not significant

Relationships between physical fitness and physical activity

The zero-order correlation coefficients between physical fitness tests results and physical activity scores are presented in Tables 4–6. In 10- and 13-year-old boys the results of the 7 tests out of 9 depend of the TPA. There are less significant relationships in girls

groups. The relationships between LPA and components of physical fitness were significant in the 10-year-old boys, in contrary, in 13-year-olds the influence of VPA was higher (Tables 4 and 6). There are only a very few significant relationships between physical fitness and physical activity parameters in girls.

Table 4. Zero-order correlation coefficients between physical activity scores and physical fitness tests in 10-year-olds (girls in brackets)

	TPA	LPA	MPA	VPA
Balance	–.316 (.184)	–.218 (.233)	–.155 (–.001)	–.117 (.235)
Sit-and-reach	.329 (.314)	.248 (.264)	.230 (.106)	.221 (.321)
10×5 m shuttle-run	–.559^{x}(–.499^{x})	–.594^{x} (.300)	–.412^{x} (.178)	–.518^{x} (–.220)
Handgrip strength	.563^{x}(–.012)	.464^{x}(.028)	.309(–.300)	.024 (.172)
Standing broad jump	.654^{x}(.344^{x})	.536^{x}(.029)	.486^{x}(.186)	.501^{x}(.480^{x})
Bent-arm-hang	.632^{x}(.467^{x})	.578^{x}(.007)	.436^{x}(.266)	.032 (.606^{x})
Plate tapping	–.573^{x}(.002)	–.530^{x}(.216)	–.675^{x}(.073)	–.207(–.291)
Sit-ups	.669^{x}(.282)	.542^{x}(–.011)	.479^{x}(.001)	.317^{x}(.742^{x})
Endurance shuttle-run	.670^{x}(.496^{x})	.676^{x}(–.095)	.490^{x}(.297)	.245 (.756^{x})

x — $p<0.05$

Table 5. Zero-order correlation coefficients between physical activity scores and physical fitness tests in 12-year-olds (girls in brackets)

	TPA	LPA	MPA	VPA
Balance	–.018 (–.171)	–.123 (–.307)	.006 (.040)	–.070 (.008)
Sit-and-reach	–.245 (.295)	–.092 (.211)	–.324^{x} (.158)	–.282 (.132)
10×5 m shuttle-run	–.180 (–.468^{x})	–.316^{x}(–.422^{x})	–.260 (–.046)	–.164 (–.327^{x})
Handgrip strength	.028 (.011)	.192 (.102)	.248 (.188)	.334^{x} (–.183)
Standing broad jump	.028 (.286)	.200 (.134)	–.030 (.254)	.004 (.129)
Bent-arm-hang	.301^{x} (.365^{x})	.360^{x} (.173)	.280 (.268)	.361^{x} (.295)
Plate tapping	–.299^{x} (–.348^{x})	–.311^{x}(–.398^{x})	–.211 (.140)	–.231 (–.274)
Sit-ups	.289^{x} (.217)	.230 (.217)	–.118 (.066)	.233 (.072)
Endurance shuttle-run	.367^{x} (.481^{x})	.256 (.390^{x})	.120 (.116)	.247 (.390^{x})

x — $p<0.05$

The stepwise multiple regression analysis (Table 7) indicates that the TPA predicted 54–59% of variance of endurance shuttle-run, standing broad-jump and bent-arm-hang in 10-year-old boys. In the same aged girls, relationships (25% of common variance) between TPA and endurance shuttle-run (Table 7) were found. The LPA accounted for 46% of variance of endurance shuttle-run and 25% of variance of the 10×5 m shuttle-run results respectively, in boys

and girls. The VPA accounted for 27% of the variance in 10×5 m shuttle-run in boys and 57% of variance in endurance shuttle-run. In 12-year-olds, the physical activity scores moderately, but significantly (23% of variance) influenced to the results of the endurance shuttle-run or 10×5 m shuttle-run (Table 8).

Table 6. Zero-order correlation coefficients between physical activity scores and physical fitness tests in 13-year-olds (girls in brackets)

	TPA	LPA	MPA	VPA
Balance	.223 (.083)	.006 (.393^x)	.136 (–.240)	.287 (–.127)
Sit-and-reach	.106 (–.214)	–.161 (–.206)	.064 (–.205)	.210 (.021)
10×5 m shuttle-run	–.601^x (–.044)	–.371^x (.101)	–.436^x (–.088)	–.581^x (–.214)
Handgrip strength	.302^x (.067)	.205 (.084)	.269 (.060)	.261 (–.038)
Standing broad jump	.691^x (.233)	.314^x (.072)	.457^x (.246)	.739^x (.254)
Bent-arm-hang	.668^x (.059)	.395^x (–.169)	.581^x (.144)	.610^x (.315)
Plate tapping	–.469^x (–.270)	–.190 (–.215)	–.231 (–.058)	–.547^x(–.398^x)
Sit-ups	.610^x (.446^x)	.345^x (.454^x)	.429^x (.346^x)	.609^x (.042)
Endurance shuttle-run	.694^x (.172)	.291 (.188)	.489^x (.100)	.739^x (.044)

x — $p<0.05$

Table 7. Results of multiple regression analysis in 10-year-olds

STEP	F	R^2	p
DEPENDENT VARIABLE: TPA			
10-year-old boys			
1. Standing broad jump, bent arm hang, endurance shuttle-run	12.46	0.56	0.0000
2. Standing broad jump, bent arm hang	16.79	0.54	0.0000
3. Handgrip strength, standing broad jump, bent arm hang	13.99	0.59	0.0000
10-year-old girls			
1. Endurance shuttle-run	7.51	0.25	0.01
DEPENDENT VARIABLE: LPA			
10-year-old boys			
1. Endurance shuttle-run	21.83	0.46	0.0000
10-year-old girls			
1. 10×5 m shuttle-run	7.66	0.25	0.01
DEPENDENT VARIABLE: VPA			
10-year-old boys			
1. 10×5 m shuttle-run	9.54	0.27	0.005
10-year-old girls			
1. Endurance shuttle-run	30.70	0.57	0.0000

Table 8. Results of multiple regression analysis in 12-year-olds

STEP	F	R^2	p
DEPENDENT VARIABLE: TPA			
12-year-old girls			
1. Endurance shuttle-run	7.87	0.23	0.01
DEPENDENT VARIABLE: MPA			
12-year-old boys			
1. Endurance shuttle-run	5.16	0.14	0.05
12-year-old girls			
1. 10×5 m shuttle-run	5.65	0.18	0.05
DEPENDENT VARIABLE: VPA			
12-year-old girls			
1. Endurance shuttle-run	4.67	0.15	0.05

A significant relationship (48–62% of common variance) between VPA and endurance shuttle-run or in combination with 10×5 m shuttle-run and standing broad jump were found in 13-year-old boys and girls (Table 9).

Table 9. Results of multiple regression analysis in 13-year-olds

STEP	F	R^2	p
DEPENDENT VARIABLE: TPA			
13-year-old boys			
1. Endurance shuttle-run	27.00	0.48	0.0000
2. 10×5 m shuttle-run endurance shuttle-run	18.65	0.54	0.0000
DEPENDENT VARIABLE: VPA			
13-year-old boys			
1. Endurance shuttle-run	35.08	0.55	0.0000
2. Standing broad jump, endurance shuttle-run	25.94	0.62	0.0000

Validity of the Godin and Shephard [11] questionnaire

The relationships between the Godin and Shephard [11] questionnaire and Caltrac estimated energy expenditure were high in all age groups, respectively r=0.939, r=0.730, r=0.910 and r=0.916 in 10, 12, 13 year-olds and in the total group (n=24).

DISCUSSION

The amount of physical activity beneficial to children's health and fitness is not well established [30, 31]. Cureton [5] has suggested to use the Blair *et al.* [4] minimum exercise energy expenditure recommended for adults (3 kcal/kg/day) which represents approximately 100 kcal of exercise energy expenditure for a 34 kg child. However, this recommendation seems to be too low and in our study even the VPA was higher. Suter and Hawes [32] reported that the activity energy expenditure in children 10 to 15 years of age was approximately from 500 kcal/day (girls) to 750 kcal/day (boys). Using this criterion the TPA of our subjects was approximately of same amount.

In our study the TPA in boys was significantly higher than in the same aged girls in all age groups (Tables 1–3). On the other hand, with increasing the age the TPA did not change, but in girls even decreased at the beginning of puberty (13-year-olds). These results are in agreement with major findings of the National Children and Youth Fitness Study [21] which reported that elder children have fewer activities than younger children.

Most investigations have supported the use of the Caltrac accelerometer as a valid and reliable measure of physical activity in children [23]. Thus, it is justified to use this accelerometer for validation of the Godin and Shephard [11] questionnaire. The correlations found in the present study (r=0.730 to 0.939) were higher than the results of other studies examining the validity of children's physical activity in self-reports [22]. We agree with Sallis *et al.* [25] that the Godin and Shephard [11] questionnaire is a much promising report for children.

The relationship between aerobic fitness and health poorly is established in youth [4, 29]. Several investigators reported no relationship between maximal O_2 consumption and physical activity [12]. Atomi *et al.* [2] concluded that the volume (intensity and duration) of daily physical activity above heart rate corresponding to 60% maximal O_2 consumption in preadolescent children might contribute to increase aerobic power. The results of the previous study indicate that the amount of TPA predicted significantly the results of the endurance shuttle-run (except in 13-year-old girls). However, frequently the more intensive activities are necessary

too. Our results are similar with Pate and Ross [16] reporting that children who perform higher on the mile walk/run test tend to participate more in community-based physical activity. Although the findings of this study are partly in agreement with the main conclusion of the review of Morrow and Freedson [15], that if the relationships between the physical activity and maximal O_2 consumption are significant, then the relationships greater than 0.40 are generally found in children younger than 14.

The multiple regression analysis indicates that the amount of TPA predicted mostly the variance (23–59%) of the endurance shuttle-run. On the contrary, Pate *et al.* [17] reported that the global ratings of the child's physical activity accounted for approximately only 11% of the variation in 1.6 km run time. In 10-year-old girls and 13-year-old boys the amount of VPA considerably (55–57%) and in 12-year-old girls moderately influenced the endurance shuttle-run test results. The amount of LPA characterizes the aerobic fitness in 10 and 12-year-old boys 14% and 46% respectively. Thus, the results of the present investigation demonstrate that the amount of TPA and VPA are significant predictors of aerobic fitness in 10–13-year-old children.

In general, moderate to high correlations between TPA and most of the EUROFIT test results (except balance and sit-and-reach) were found. The multiple regression analysis indicates that inclusion TPA and VPA in the model accounted for 27–59% of the variance in several physical fitness tests where the body mass was moved or projected. Simons-Morton *et al.* [29] reported that there is no evidence to suggest that children's generally high levels of physical fitness are due to frequent participation in moderate to vigorous physical activity. Cross-sectional studies indicate better levels of physical fitness in more active boys [20] and girls [14]. However, longitudinal studies indicate that there are no significant differences in several physical fitness tests in more and less physically active boys and girls [3], but in the Dutch study [34] time spent in sports participation is more highly correlated with fitness items than total time in physical activities. Indicators of physical activity are significantly but moderately related to strength of 12-year-old children [28]. It is possible that more skilled children tend to be more physically active [35]. Then future research needs to assess the relationships between physical fitness (especially

speed, strength, flexibility, agility) and the amount of physical activity of different intensities.

In summary, our study results demonstrate that the level of aerobic fitness is highly dependent on the amount of TPA and VPA in 10, 12 and 13-year-old children. The relationships with the other motor abilities (speed, strength etc.) are not so clear. The Godin and Shephard [11] questionnaire is a valid measure of physical activity in children.

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FATIGUE AND POTENTIATION AFTER ISOMETRIC KNEE EXTENSION AT LOW TO SUBMAXIMAL TARGET FORCE

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ABSTRACT

It was hypothesised that physical exercises may induce both fatigue and potentiation of muscle contraction, and interaction of these factors depends on the specificity of performed exercises. The aim of the study was to determine: 1) the interaction between maximal voluntary contraction (MVC) and twitch tension; 2) twitch tension and the intensity of performed exercise; 3) the ability of the muscle to use up potentiation according to performed exercise; and 4) the dependence of time course of twitch recovery on exercise intensity.

Our results showed that: 1) MVC and single twitch tension change independently both during exercise and recovery. MVC decreased approximately to 80% of initial after all four exercises while twitch tension exhibited specificity on intensity; 2) twitch tension increases intensity of the exercise; 3) low intensity does not induce twitch potentiation despite remarkable decrease in MVC. The ability of twitch tension to potentiate is being used progressively with increases in exercise intensity; 4) the time course of twitch recovery depends on the intensity of performed exercise. Submaximal exercise tended to delay the recovery of tension in comparison with low and maximal intensities.

Key words: fatigue, post-tetanic potentiation, m.quadriceps femoris

INTRODUCTION

It has been shown that the contractility of the muscle after exercise depends on interaction of post-tetanic potentiation and fatigue [13]. Both of these phenomena are the reflection of the events which have distinct underlying mechanisms. Post-tetanic potentiation phenomena is associated with phosphorylation of myosin regulatory light chains (LC) by calmodulin-dependent myosin LC kinase [9, 14, 15]. Fatigue, referred to be the reduction of maximal force generating capacity , may be of the central [2] and/or peripheral origin and related to changes in afferent input [8], shift in metabolic state [12], deficit of energy sources [1], changes in calcium release [17], morphological alterations [10].

It was hypothesised that physical exercises may induce both fatigue and post-tetanic potentiation of muscle contraction and interaction of these factors depends on the specificity of performed exercises. In addition, the same set of fatigue and post-tetanic potentiation may differently effect distinct contractile characteristics. The aim of this study was to determine: 1) the interaction between maximal voluntary contraction (MVC) and twitch tension; 2) twitch tension and the intensity of performed exercise; 3) the ability of the muscle to use up potentiation according to performed exercise; and 4) the dependence of time course of twitch recovery on exercise intensity.

MATERIAL AND METHODS

Subjects. Six males healthy volunteers aged 25–32 years participated in this study. The subjects were physically active but none of them took part in any formal exercise or sport. The experimental sessions were separated by at least two days intervals.

Force measurement. The subject was seated upright in the experimental chair with a vertical back support provided and remained seated until the end of each experiment. A strap secured the hips and thigh to minimise uncontrolled movements. The leg was clamped in the force measuring device with the knee semi-flexed. A plastic cuff, placed around the leg proximal to malleoli,

was tightly attached to a linear variable differential transducer. The output of the transducer, proportional to knee extension force was amplified and digitised at a sampling rate of 1kHz by a 12-bit analogue-to-digital converter installed in IBM-compatible AT 386 personal computer. The digitised signal was stored on a hard disk for subsequent analysis. At the same time, the output from force transducer was displayed on the ampermeter in front of the subject.

Electrical stimulation. A high voltage stimulator (MG 440, Medicor, Budapest, Hungary) was used. Electrical stimuli as 1-ms duration pulse of 150 V were delivered to quadriceps muscle through surface electrodes soaked in water. One stimulation electrode was placed and fixed just above the pattela, while the other covered the large portion of the muscle belly in the proximal third part of the thigh. The subjects were introduced to electrical stimulation during their first visit to the laboratory.

Experimental protocol. Experimental protocol was designed to evaluate the effect of: 1) sustained isometric voluntary contraction at different force level; and 2) 10-s maximal voluntary contraction (10-s MVC) on maximal voluntary contraction force and single twitch tension. The time course of the experiment session was divided into periods of rest, performance and recovery (Figure 1). There were no differences in procedures between left and right legs during the periods of rest and recovery. In both legs, the period of performance consisted of sustained isometric exercise. While in the right leg, 10-s MVC was additionally performed following post-exercise twitch and after the period of recovery.

Rest. After 5 min rest in the experimental chair, MVC of the leg was measured following evoked single twitch. These values were referred to be initial. MVC was recorded as the best of two maximal voluntary knee extensions lasting approximately 4–5 s each. A rest pause of 3 min was allowed between the attempts. After the testing procedures, the subjects rested for 5 min before starting the isometric exercise.

Performance. The period of performance was characterised by different target force and duration of the isometric exercise: 10% MVC for 8 min, 20% MVC for 4 min, 40% MVC for 1 min, 60% MVC for 30 s and 10-s MVC. According to the target force, the

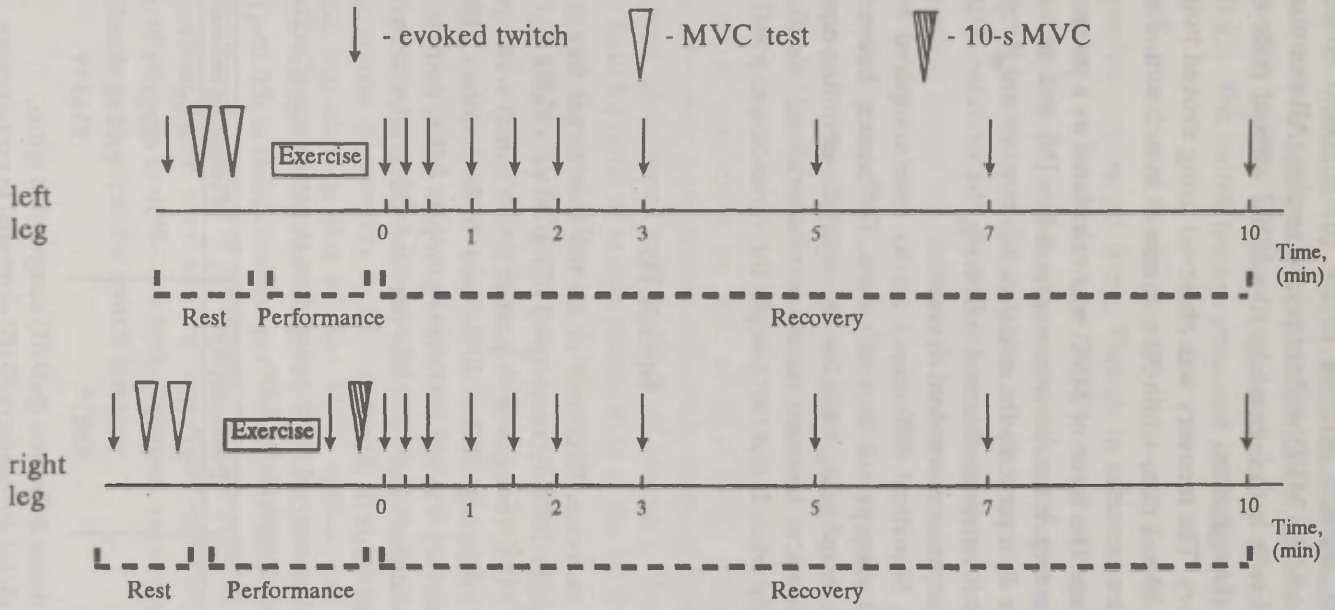

Figure 1. Experimental set up.

experiment sessions referred to be 10%-, 20%-. 40%-, 60%-exercise and 10-s MVC without prior-exercise. All exercises were performed with both legs while 10-s MVC without prior-exercise only with the right one.

Recovery. The recovery was observed using evoked twitches at 0 s, 15 s, 30 s, 1 min, 1 min 30 s, 2 min, 3 min, 5 min, 7 min and 10 min after exercise.

Calculation. The force of MVC was calculated as a percentage of initial. The twitch tension was expressed in [N] and as a ratio of P/P_o where P_o represents the initial twitch tension and P represents those twitch tensions obtained following the exercise. Data were presented as mean ± standard deviation.

Statistics. Statistical differences across time compared to initial was tested using paired Student's t-test. Differences between exercises were tested using t-test for two samples assuming equal variance. Differences between means were considered significant for $p<0.05$ only when F-test two sample for variance was $p>=0.05$.

RESULTS

Effect of exercise. Exercises of all the four target forces caused approximately 20% decrease ($p<0.05$) in MVC (Table 1). The recovery of MVC (compared to post-exercise value) was significant ($p<0.05$) following 20%-, 40%- and 60%-exercises. However, 10 min recovery was not sufficient to regain MVC completely and there were differences ($p<0.05$) between force after recovery and initial force value in all cases.

Table 1. Maximal voluntary contraction as percentage of initial. Mean and standard deviation.

	Post-exercise	Following recovery
10%	81±10*	90±6*
20%	82±6*	92±4*♦
40%	79±5*	92±3*♦
60%	83±7*	93±4*♦

* — significant difference ($p<0.05$) compared to initial,

♦ — significant difference ($p<0.05$) compared to post-exercise

Figure 2 illustrates the effect of all four exercises and 10-s MVC on twitch tension. 10-s MVC without prior-exercise increased the twitch tension, expressed as a ratio of P/P_o, (2.24±0.45). The twitch tension remained potentiated ($p<0.05$) for 10 min.

The time course of twitch recovery following exercises showed the dependence on target force. Though in every case, the twitch tension tended to be potentiated ($P/P_o>=1$) in contrast to MVC. The potentiation of twitch tension tended to be greater with increasing target force of the exercise. Twitch tension after 10%-exercise showed only slight differences compared to that of initial. 20%-exercise caused the increase in twitch tension ($p<0.05$) from the 1-st to 7-th minute of recovery. 40%- and 60%-excercises caused twitch potentiation ($p<0.05$) over the period of recovery. 60%-excercise induced peak immediately after performance to 2.19±0.50. While following 40%-excercise twitch tension peak of 1.61±0.49 delayed until the third min. the twitch tension after 60%-exercise was close to that of 10-s MVC without prior-exercise. However, potentiation induced by mean of former tended to be better pronounced until the end of recovery. However, the greatest twitch extend of twitch potentiation at the 10-th minute of recovery was observed following 40%-exercise.

Effect of 10 s-MVC. All four exercises equally affected the twitch force in both right and left legs. Additional 10-s MVC, immediately after exercise and evoked twitch, caused significant differences between twitch tension of the right and left legs at the corresponding time. 10-s MVC increased twitch tension and affected the time course of recovery in 10%-exercise. The peak-twitch potentiation was close to that of 10-s MVC without prior exercise and even tended to exceed it. In 20%-excercise, significant increase ($p<0.05$) in twitch tension persisted up to the 2-nd minute of recovery, though, extend of potentiation tended to be lower than in 10%-exercise. There were not any significant differences caused by 10-s MVC both in 40%- and 60%-exercises, however, persisted the tendency to suppress the twitch force.

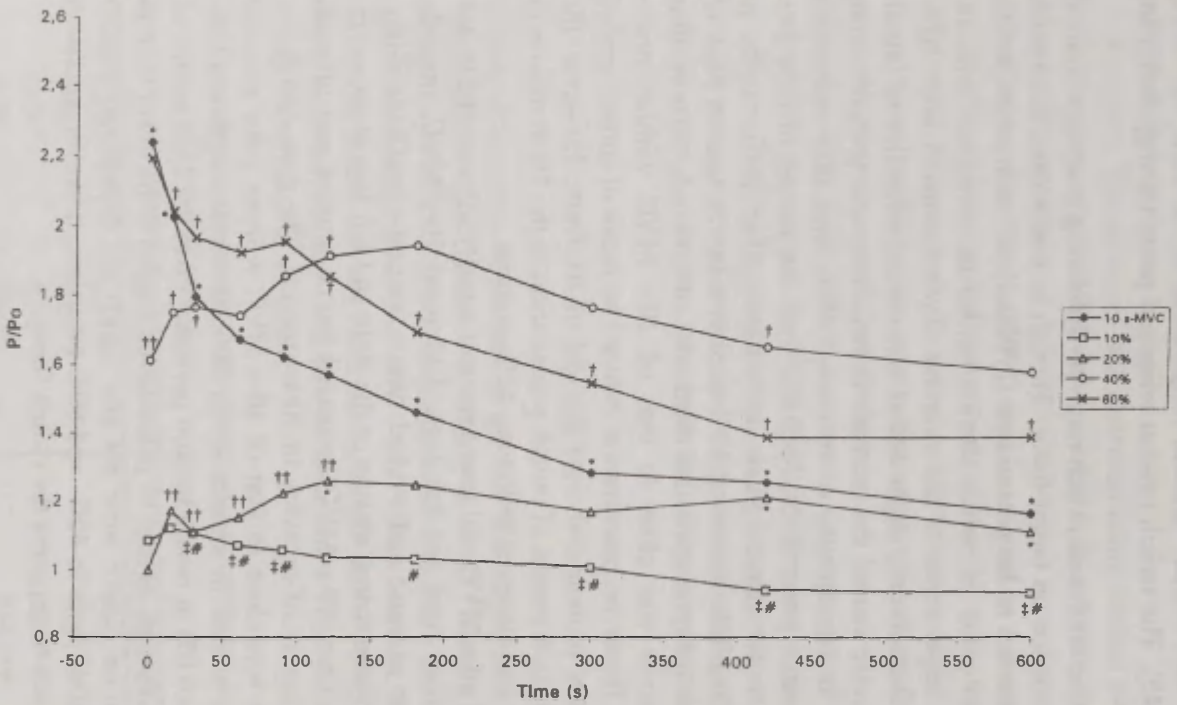

Figure 2. Twitch tension after all four exercises and 10-s MVC, left leg. †† — significant difference ($p<0.05$) compared to 10-s MVC, * — 10%-exercise, † — 20%-exercise, — 40%-exercise, # — 60%-exercise.

Table 2. Twitch tension (N) of both legs at rest (initial) and immediately after exercise (post-exercise). Mean ± standard deviation.

Exercise	Initial		Post-exercise	
	right leg	left leg	right leg	left leg
10%	52±25	52±20	51±19	58±25
20%	67±21	62±22	64±17	60±18
40%	54±18	47±19	70±25	79±39
60%	50±16	49±19	105±34	119±54

DISCUSSION

Why were different exercise intensities chosen?

Different intensity and duration of the exercise was chosen with the aim to evoke similar level of fatigue detected by decrease in MVC. Fatigue was significant but not profound. Though decrease in MVC was similar following all four exercises (Table 1), however, main fatigue-induced mechanisms differed undoubtedly. This assumption is based on study of Enoka *et al.* [6] which revealed that origin and degree of muscular fatigue depends on the intensity, duration and regimes of performed exercise as well as on the duration of the rest between series and total amount of physical work. Thus, it was considered that the decline in MVC was caused by central fatigue in 10%-exercise. While fatigue dominated due to metabolic changes in 20- and 40%-exercises and in 60%-exercise fatigue was caused by both alteration in central drive and metabolic state. In addition, 10-s MVC was performed as a stimulus to induce maximum post-tetanic potentiation [16].

What do single twitch force and MVC indicate?

Our results showed that performing different exercises changes in MVC were similar while single twitch exhibited specificity on the intensity (Figure 2). This suggests the dependence of theses characteristics of muscle contractility on mechanisms with different localisation. MVC is determined by the integrated influence of both central and muscular force generating mechanisms while sin-

gle twitch only by peripheral ones. In the periphery, single twitch tension depends on the speed of calcium ions [7], while MVC is determinated by the total amount of released calcium and contractility of myofibrils [18]. Therefore, posttetanic potentiation increases single twitch tension by means of increased affinity of myofibrils to calcium ions [11] without any effect on MVC. Thus, mechanisms determining used characteristics might be differently affected while performing the exercises on different intensities.

Why does the increase in the exercise intensity not induce the fatigue of twitch tension?

Our results confirmed the hypothesis that augmentation of exercise intensity increases twitch tension when changes in MVC are similar. Increase in exercise intensity is associated with mobilisation of motor units enervating fast twitch fibres [4], for whom post-tetanic potentiation is known to be specific [3]. The appearance of this phenomena reveals these contractile characteristics which depend on the affinity of myofibrils to calcium ions, and the amount of potentiation is directly related to the level of activation of muscle mass [11]. As mentioned above, single twitch is one of such characteristics. Although the main mechanism of post-tetanic potentiation has been expected to be phosphorylation of myosin LC which depends on calcium concentration in myoplasm [14]. The results of recent experiments on humans and rats showed that there is no direct relationship between twitch potentiation and degree of phosphorylation [9, 15].

What is the interaction of post-tetanic potentiation and fatigue?

According to our hypothesis, the time course of single twitch tension following exercise depends on the interaction of two opposite processes: post-tetanic potentiation and fatigue. MVC is an indicator of fatigue while single twitch tension exhibits interaction of fatigue and post-tetanic potentiation (however not all reasons that had caused fatigue of MVC may affect twitch tension). Decrease in MVC during exercise indicates the appearance of fatigue [5] but when it is

concomitant with augmentation of twitch tension we consider the predominant effect of potentiation on fatigue in the periphery of the force generating machinery. This is the one way to observe the interaction of the effects of fatigue and post-tetanic potentiation. However, it is actually impossible to evaluate the influence of these two phenomena only in this way. For example, according to our results, it remains not clear whether the higher level of potentiation or the lower level of fatigue caused the tendency of twitch tension following 60%-exercise to exceed that after 40%-exercise. The another way is to use 10-s MVC immediately after post-exercise twitch. It reveals twitch tension was predominantly affected by fatigue in case of 20%- and 40%-exercises (following 10-s MVC it was approximately twice lower than after 10-s MVC without prior exercise) in comparison with 60%-exercise where no additional potentiation was already observed. It seems that neither fatigue nor post-tetanic potentiation significantly affected twitch tension after 10%-exercise because it remained very close to pre-exercise level immediately following performance (no reduction which would indicate fatigue) and exhibited maximal potentiation after 10-s MVC (which suggests being not potentiated due to the exercise) (Figure 3). The third way to evaluate the influence of post-tetanic potentiation and fatigue is the observation of time course of twitch recovery (see below).

Why does the time course of twitch recovery depend on exercise intensity?

The results showed that the twitch time course depends on the intensity of performed exercise in recovery (Figure 2). It is interesting to note that the twitch recovery delayed after 40%- and 60%-exercises in comparison with 10-s MVC without prior-exercise. Similar results were observed by Vandervoort *et al.* [16]. According to our hypothesis the time course of twitch recovery depends on the interaction of fatigue and potentiation. The twitch recovery may be delayed when fatigue disappears more quickly than potentiation. However, this can not explain the divergence of twitch recovery from the second minute between 10-s MVC and especially in 40%-exercise. Houston *et al.* [9] and Vandervoort *et al.* [16] showed that 10-s and 60-s MVC induced similar myosin LC phosphorylation and it was

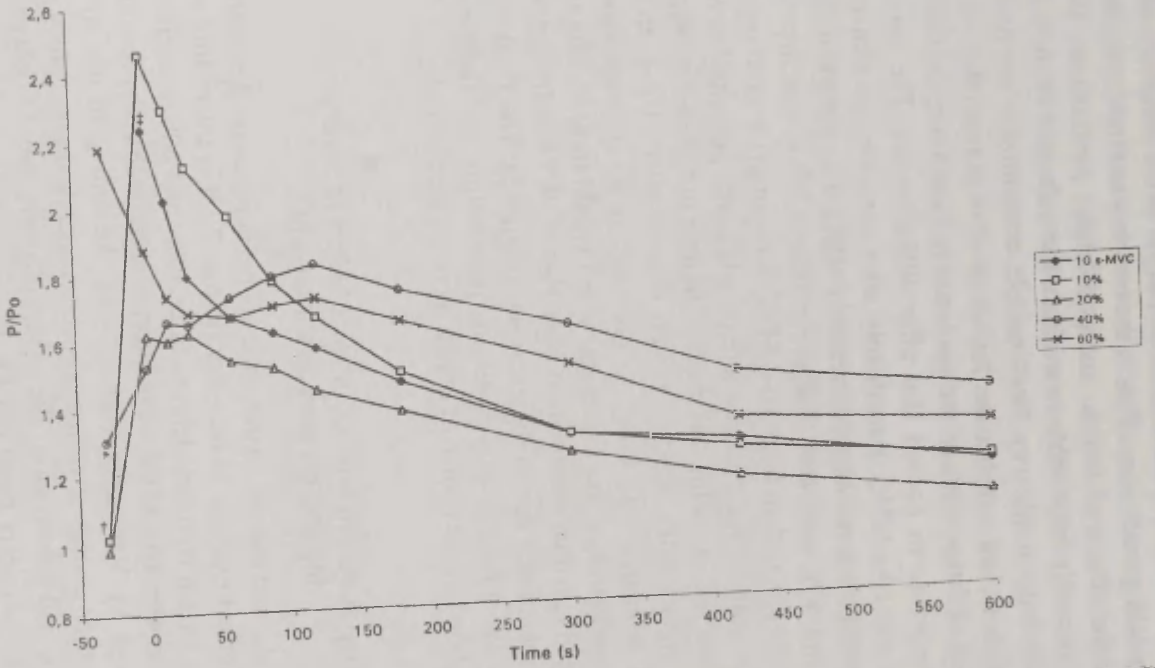

Figure 3. Twitch tension after all four exercises and 10-s MVC, right leg. * — significant difference ($p<0.05$) compared to 10%-exercise, † — 20%-exercise, — 40%-exercise.

similar between trials following 10 minutes of recovery. Thus, our results support the doubt about the single mechanism underlying post-tetanic potentiation phenomena. 20%-, 40%- and 60%-exercises are associated with accumulation of metabolites which reduce not only force generating capacity but also decrease relaxation rate [7, 17]. Besides the increase in twitch tension caused by phosphorylation, the prolongation of relaxation time may bring about additional increase, especially when recovery of released calcium amount has already occurred. However, we do not have any direct evidence to support our speculation.

Thus, the results confirmed the hypothesis that physical exercises induce both fatigue and post-tetanic potentiation of muscle contraction and the interaction of these factors depends on the specificity of performed exercises. In addition, the same set of fatigue and potentiation may differently effect distinct contractile characteristics.

CONCLUSIONS

1. MVC and single twitch tension change independently both during exercise and recovery.
2. Twitch tension increases with increased intensity of the exercise.
3. Low intensity does not induce post-tetanic potentiation despite remarkable decrease in MVC. The ability of twitch tension to potentiate is being used progressively with increase in exercise intensity.
4. The time course of twitch recovery depends on the intensity of performed exercise. Submaximal exercise is tended to delay the recovery of tension in comparison with low and maximal intensities.

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CHANGES IN MUSCLE FORCE AND EMG AT CONSTANT FORCE-TIME PRODUCT OF ISOMETRIC EXERCISE PERFORMED AT DIFFERENT TARGET FORCE

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ABSTRACT

The effect of intermittent isometric exercise was examined on maximal voluntary contraction, single twitch, paired twitch of 100 Hz, 20 and 50 Hz evoked force, and EMG amplitude of m. quadriceps femoris. Six persons performed three sessions characterised by the constant force-time product and different target force of the exercise with at least 7 days interval between the sessions. The results showed that: 1) the constant force-time product equally affected MVC but the specificity of exercise intensity was observed for 20 Hz force and for appearance of low-frequency fatigue; 2) evoked forces rely to the state of the muscle that depends on interaction of potentiation and fatigue; and 3) potentiation of Pt and Pd was associated with the drop of EMG amplitude at the onset of target force generation while the increase in EMG was accompanied by the deficiency of high-frequency evoked forces. The results suggest that the same muscle force may be maintained due to a different activation from central nervous system (CNS) in accordance with the muscle state.

Key words: muscle, EMG, isometric intermittent exercise, fatigue, potentiation.

INTRODUCTION

It has been shown that various involuntary forces of muscles may be non uniformly effected by the same exercise [9, 11]. Short bout of high intensity isometric exercise is known to potentiate muscle force at low frequencies especially single twitch [1] without significant effect on the force at high frequencies or maximal voluntary contraction (MVC). While prolongation of the exercise and/or repetition of few bouts leads to decrease in both single twitch force and forces of high frequency as well as MVC indicating appearance of fatigue [13]. Thus, changes in contractile characteristics depend on muscle state referred to interaction of fatigue and potentiation of the muscle [1, 9, 10]. The influence of both of them is related to the duration and intensity of exercise [22, 26]. CNS is able to compensate the decreased muscle force by recruitment of new motor units and/or by an increase in firing rate of already recruited motor units [5, 23]. Another type of motoneurons and muscle fibres' interaction described as "muscle wisdom" phenomena is associated with concomitant decrease in fibres' relaxation rate and motoneuron firing rate [9]. Fine co-ordination between motoneurons and muscle is necessary to maintain target force because of changing muscle state during exercise. The purpose of our study was to investigate: 1) the effect of constant force-time product of the exercise performed at different intensities on the m. quadriceps femoris force; 2) the effect of interaction of potentiation and fatigue on muscle force; and 3) changes in root mean square of EMG amplitude while maintaining target force.

MATERIAL AND METHODS

Subjects. Experiment was carried out on six healthy volunteers (males) aged 23–35 (mean 29) years. The subjects were physically active but none of them took part in any formal exercise or sport. All subjects participated in the *session III* of investigation while five subjects participated in *sessions I* and *II*. The sessions were separated by at least weekly intervals.

Force measurement. The subject was seated upright in the experimental chair with a vertical back support provided and remained until the end of each experiment. A strap secured the hips and thigh to minimise uncontrolled movements. The right leg was clamped in the force measuring device with the knee semi-flexed. A plastic cuff, placed around the leg proximal to malleoli, was tightly attached to a linear variable differential transducer. The output of the transducer, proportional to knee extention force was amplified and digitised at a sampling rate of 1 kHz by a 12-bit analogue-to-digital converter installed in IBM-compatible AT 386 personal computer. The digitised signal was stored on a hard disk for subsequent analysis. At the same time the output from force transducer was displayed on the ampermeter in front of the subject.

Electrical stimulation. A high voltage stimulator (MG 440, Medicor, Budapest, Hungary) was used. Electrical stimuli to quadriceps muscle were delivered through surface electrodes soaked in water. One stimulation electrode was placed and fixed just above the patella, while the other covered large portion of the muscle belly in the proximal third part of the thigh. Electrical stimulation was delivered in trains of square wave pulses of 1-ms duration using voltage of 150 V. The subjects were introduced to electrical stimulation during their first visit to the laboratory.

EMG recording. The surface electrodes were placed over the vastus lateralis muscle near to the middle of the right hip between stimulating electrodes. The skin was shaved before and an electrode cream was used to reduce resistance. The signal was amplified and digitised at a sampling rate 2500 Hz by a analogue-to-digital converter installed in IBM-compatible AT 386 personal computer. The signal was stored on a hard disk for subsequent analysis.

Experimental protocol. After 5 min rest in the experimental chair MVC concomitant with EMG was measured following a muscle force generating capacity test (MFGCT). These values were referred to initial. The MFGCT comprised muscle contractions in response to electrical stimuli. Subsequent stimulation pattern was used, i.e. single twitch (Pt), paired twitch of 100Hz (Pd) and 1 s trains of 20 and 50 Hz. A pause of 2 or 3 s was necessary to change frequency after each train. After 2 min rest MFGCT was performed followed by MVC and EMG

measurement. MVC was recorded as the best of two maximal voluntary knee extensions lasting approximately 4–5 s each. A rest pause of 3 min was allowed between the attempts. The sampling of EMG covered the plato of MVC and lasted 3 s. The subject rested for 5 min after test procedures before starting the intermittent isometric exercise. The exercise consisted of repeated voluntary contractions. Six contractions with 1 min rest between contractions comprised a series. Three series interspaced with 20 min rest comprised the experiment session. The target force of repeated voluntary contraction as well as the duration were different in every session: *session I* — 20% of MVC *vs* 60 s, *session II* — 40% of MVC *vs* 30 s and *session III* — 60% of MVC *vs* 20 s. This allowed to maintain the same force-time product of the exercise in all three sessions. EMG of target force was recorded at the beginning and at the end of every contraction. The sampling time was 5 s with exception of *session III*, where the sampling time was 3 s. MFGCT was carried out directly after the last voluntary contraction of the series. Approximately 1 min later MVC concomitant EMG recording was followed. The experiment sessions were carried out at random order.

Calculation. Muscle force output was represented by a peak force as a percentage of initial. The root mean square of EMG amplitude was used as a characteristic of EMG while generating MVC (EMG_{MVC}) and maintaining target force (EMG_{TF}). Both of them were expressed as a percentage of initial.

Statistics. Paired Student's t-test was used for data analysis.

RESULTS

MFGCT. Table 1 presents force in response to electrical stimuli. Force at 20 and 50 Hz decreased gradually from series to series during all sessions (Figure 1.A). The exception was 20 Hz force performing *session II* when no additional drop was observed following the third series. 20 and 50 Hz forces were significantly reduced ($p<0.05$) after all sessions. Performing *session III*, 20 Hz force was depressed significantly ($p<0.05$) stronger than that of 50 Hz — 45% *vs* 61% respectively (Figure 2). Only 20 Hz force dependence on the intensity of the exercise was found. Following

performance of *session III* it was significantly ($p<0.05$) lower than that after *session II* — 45% *vs* 68%. In contrast, Pt and Pd forces tended to be potentiated after the first series especially performing *sessions II* and *III* though the wide dispersion of the data was typical for these forces (Figure 1.B).

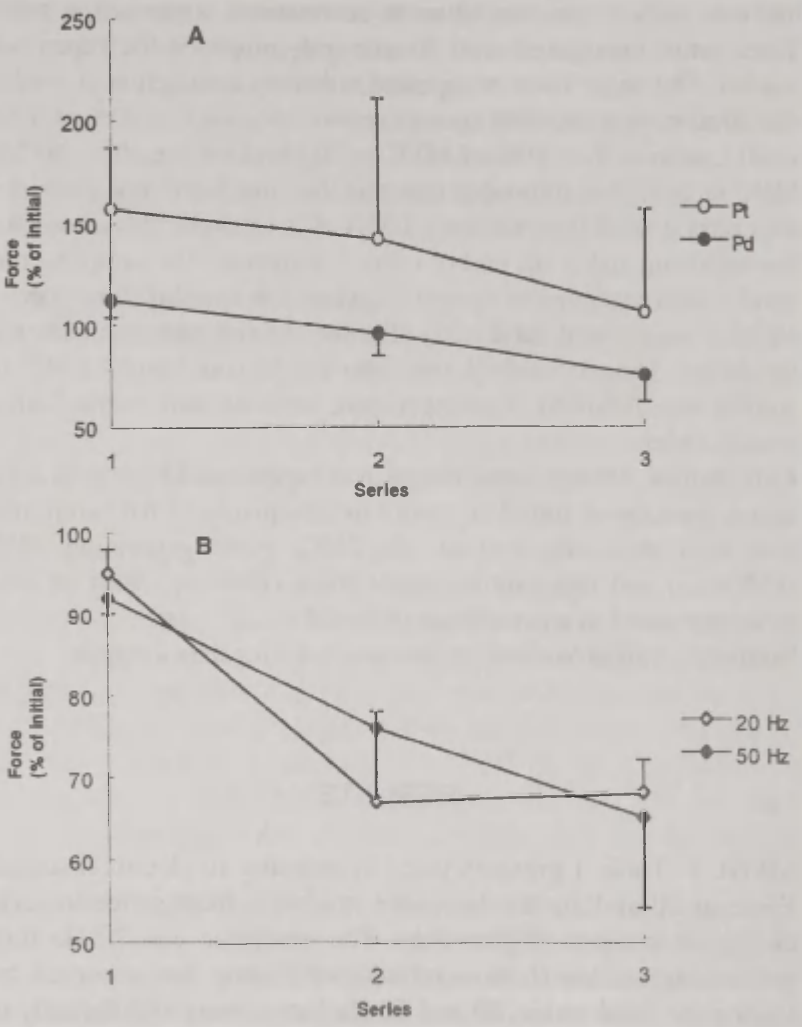

Figure 1. Force peaks of evoked contractions during *session II* (as percentage of initial). A — 20 Hz and 50 Hz, B — single twitch (Pt) and paired twitch of 100 Hz (Pd). Mean and standard error.

Table 1. Force of evoked muscle contractions (as percentage of initial). Mean and standard error (SE). Pt — single twitch, Pd — paired twitch of 100 Hz, * — significant ($p<0.05$) difference from initial value.

Index, series	*Session I*		*Session II*		*Session III*	
Pt	mean	SE	mean	SE	mean	SE
1	113	23	157	30	161	33
2	84	19	142	69	108	23
3	68	24	106	50	95	25
Pd	mean	SE	mean	SE	mean	SE
1	99	14	112	8	114	13
2	82	11	96	11	88	15
3	72	15	75	13	67	13
20 Hz	mean	SE	mean	SE	mean	SE
1	86	6	95	3	85	8
2	76	9	67*	11	59*	9
3	61*	8	68*	4	45*	8
50 Hz	mean	SE	mean	SE	mean	SE
1	91	4	92*	2	90	7
2	74*	6	76	9	71*	6
3	69*	9	65*	11	61*	9

MVC and EMG_{MVC}. The MVC was significantly ($p<0.05$) depressed following all sessions without any dependence on target force having been maintained (Table 2). However, MVC tended to be less affected than 20 Hz and 50 Hz evoked forces (Figure 3). 20 Hz force following 2-nd and 3-rd series at *session III* as well as after 3-rd series in *session II* was significantly ($p<0.05$) lower in comparison with MVC at the same time: 59% *vs* 84%, 45% *vs* 84% and 68% *vs* 86% respectively.

The amplitude of EMG concomitant with MVC (EMG_{MVC}) tended to be reduced following performance of every series at all sessions.

EMG_{TF}. The level of EMG_{TF} at the start was close to the respective level of target force maintained, i.e. 20±2%, 36±6% and 52±9%. The EMG_{TF} increased from the beginning to the end of the voluntary contraction (Figure 4). The rest among contractions caused the decrease in EMG_{TF} while performing *session I*, EMG_{TF} dropped until 16±3% and 15±2%, at the start of 2-nd and 3-rd voluntary contractions. While at the 3-rd and 6-th muscle contraction,

EMG_{TF} dropped until 47±8% and 47±6%, respectively when performing *session III*. It was significantly ($p<0.05$) lower than at the start of performance in all cases mentioned above. There was general increase of EMG_{TF} during the first series maintaining all the levels of the target force. The tendency of increase in EMG_{TF} during the second series remained through all the sessions especially at the end of the voluntary contraction. The enhancement of EMG_{TF} was kept up in the 3-rd series performing *sessions I* and *II*. The last contraction in the *session III* was characterised by the drop of EMG_{TF} to the pre-exercise level. The rest pause between voluntary contractions caused 10% and more drop of EMG_{TF} as compared to the end of the former contraction while the 20-min recovery after series produced only approximately 5% of decrease. No significant differences between EMG_{TF} at the end of the first series and at the start of the second one were found.

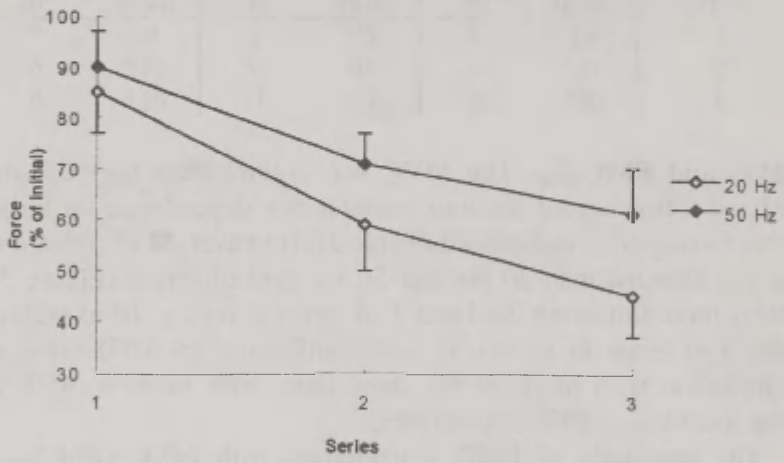

Figure 2. Force of evoked contractions while performing *session III* (as percentage of initial). Mean and standard error.

Table 2. Maximal voluntary contraction (MVC) and concomitant root mean square of EMG (EMG_{MVC}) as percentage of initial. Mean and standard error (SE).

Index, series	*Session I*		*Session II*		*Session III*	
MVC	mean	SE	mean	SE	mean	SE
1	94	3	96	2	92	2
2	89	4	94	2	84*	2
3	86*	2	86*	2	84*	2
EMG_{MVC}	mean	SE	mean	SE	mean	SE
1	86*	2	79*	5	75*	4
2	74	8	80	6	70*	5
3	74	13	77	9	72*	7

* — significant ($p<0.05$) difference from initial value.

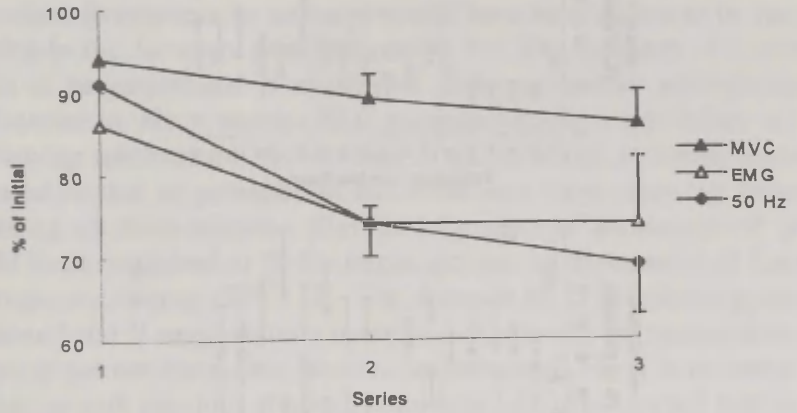

Figure 3. 50 Hz evoked and maximal voluntary contraction (MVC) force with concomitant root mean square of EMG while performing *session I* (as percentage of initial). Mean and standard error.

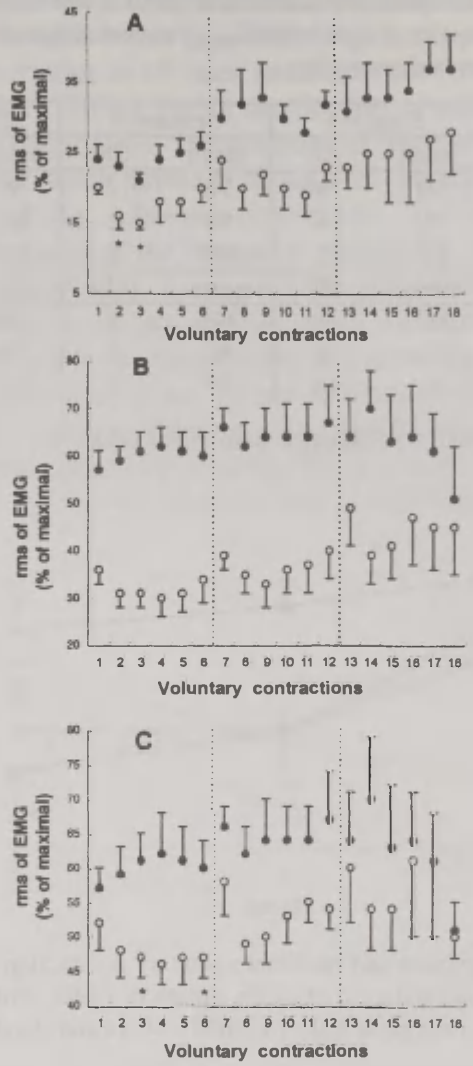

Figure 4. Root mean square of EMG while maintaining target force (EMG_{TF}) as percentage of initial value. Mean and standard error. A — performing *session I*, B — *session II* and C — *session III*. Opened symbol reflects EMG_{TF} at the onset of contraction, filled one — at the finish of the same contraction. * — significant difference ($p<0.05$) of EMG_{TF} at the onset of contraction as compared with initial. Dotted line separates the series of voluntary contractions.

DISCUSSION

Electrical stimulation. According to the manner of the changes two groups of electrically invoked muscle forces might be selected: 1) Pt and Pd and 2) 20 Hz and 50 Hz. A reason for the resemblance in their changes might be caused by the similar sensitivity of Pt and Pd as well as 20 and 50 Hz forces to the muscle state associated factors. Such factors as 1) the increase in cross bridges attachment — detachment rate due to the phosphorylation of myosin regulatory light chains (RLC); 2) the increase in the troponin C sensitivity to Ca^{2+}; and 3) activation of adenosine triphosphate (ATP) hydrolysis [4, 9, 10] are relevant for the force generated during Pt and Pd contraction. Meanwhile, the decrease in ATP hydrolysis rate, the increase in [Pi] as well as decrease in pH and the decreased amount of released Ca^{2+} from sarcoplasmic reticulum in response to action potential have been shown to be limiting factor, for tetani peak induced [8, 10]. The tendency of Pt and Pd to be potentiated is associated with post-tetanic potentiation phenomena where myosin RLC phosphorylation is one of the underlying mechanisms. An increase in the influence of fatigue compared to that of potentiation has been seen from series to series during all three sessions. Significantly stronger depression of 20 Hz force compared to 50 Hz might indicate an appearance of low-frequency fatigue (LFF) [7]. The absence of LFF following *sessions I* and *II* suggests the importance of intensity of isometric exercise but not force-time product for inducing LFF. It is in confirmation with previous studies [16] where LFF was exerted following intermittent MVC. However, it has been shown that prolongation of exercise duration is able to induce LFF even when maintained isometric force is lower than 40% of MVC [25]. It is known that slow twitch fibres (those known to be in deep portion of a muscle) are being reqruited when maintaining low target force, while fibres located near the skin surface are especially exposed to transcutaneous stimulation. It seems likely that low intensity exercise did not effect fibres exposed to stimulation and exercise affected fibres were not recruited during stimulation. It may account for 20 Hz force reduction in *session III* in comparison with *sessions I* and *II*. Thus, fatigue following different exercise intensity might be located in different portions of the muscle.

MVC and EMG_{MVC}. Mobilisation of new motor units (MU) instead of fatigued MU during second and third series might affect the contractile properties of fibres exposed to transcutaneous stimulation. There is no ground to reject the reason that muscle was fatigued relatively deeper as activating mechanisms of CNS during such kind of exercise. The decrease in EMG_{MVC} maintaining all target forces might be the reflection of impairment of activation from CNS, i.e. motoneurons firing rate or/and recruitment [2, 3]. The ability of motoneurons to maintain the firing rate and to be recruited depend also on various afferent mechanisms as well [18, 21]. Afferent input may have changed during exercise. However, we are not able to evaluate these changes because EMG depends on numerous factors [23]. This complicates the interpretation of our data.

EMG_{TF}. EMG_{TF} rises in time course of target force generation because of mobilisation of new MU with the aim to compensate fatigue and maintain desired target force [20, 24, 28]. The dynamics of EMG_{TF} depends not only on recruitment of new MU but also on motoneurons firing rate which may decrease during exercise because of adaptation [17]. Similar effect was also observed when contractions were performed at maximum intensity [2]. It has been known for a long time that the activity of motoneurons is co-ordinated with the muscle state [3, 14, 23]. Thus, the efficiency of motor control is achieved. The CNS has to resolve a complicated problem as there exists a wide variety of muscle states. Muscle was exposed to several different states during our experiment depending on target force and on stage of the exercise. The decrease in EMG_{TF} noticed after one minute rest in comparison with the start level may be attributed to post-tetanic potentiation (the increase in Pt and Pd following first series accounts for it) knowing that it remains decreased after the exercise for a few minutes [12]. Potentiation may be critical for force generation when some mechanisms caused fatigue are diminished. It suggests the ability to maintain same submaximal force in response to lower motoneurons firing rate. On the other hand, the long pause of recovery (15 min) almost eliminated the influence of potentiation resulting in the narrower range of the drop in EMG_{TF} after the series than after the muscle contraction. Besides, EMG_{TF} is not able to reflect the level of potentiation of the whole muscle because: 1) EMG predominantly characterises electrical activity of

muscle fibres located near the surface (as usual, here are found fibres of the fast twitch type which are known to be recruited in accordance with the "size" principle); 2) the decrease in EMG_{TF} may be concomitant with an increase in electrical activity in m. vastus medialis or m. rectus femoris because they may have been activated non-uniformly during the same exercise [18, 27]; 3) both the increase in activity of mechanoreceptors and decrease in activation from muscle spindles while maintaining the force may depress the firing rate of motoneurons more than it is necessary for the "muscle wisdom" or for the potentiation of the muscle [21]; and 4) the activity of motoneurons may be suppressed due to the adaptation or weak excitation from CNS compensating it by mobilisation of new motoneurons [17]. The influence of the factors mentioned above may vary during the recovery.

In summary, the results of the study showed that: 1) the constant force-time product equally affected MVC but the specificity on exercise intensity was observed for 20 Hz force; 2) the force evoked rely to the state of muscle that depends on interaction of potentiation and fatigue (low-frequency forces are predominantly sensitive to potentiation while high-frequency forcesare sensitive to fatigue); 3) the increase in EMG_{TF} was accompanied by the deficiency of high-frequency evoked forces while potentiation of Pt and Pd was associated with the drop of EMG_{TF} at the onset of target force generation. It suggests that the same muscle force may be maintained due to a different activation from CNS in accordance with the muscle state.

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RELATION BETWEEN BIOLOGICAL MATURATION AND MOTOR ABILITIES IN PUBESCENT GIRLS

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ABSTRACT

The study was performed in order to check the dependence of the formation of motor capacities on biological maturation. In 77 girls (age 11 to 14 years) sexual maturation stages were evaluated according to Tanner's method. Skeletal age was assessed by x-ray picture of the left wrist. Motor capacities were assessed with the aid of Cooper's test, Harvard step-test, shuttle run 4×9 m, 20m dash, sit ups 30s, squats in 30s, standing long jump, and trunk forward flexion. Increases of results of standing long jump and forward flexion test were related to the transition from the sexual maturation stage II to stage III. Reaching stage IV associated with improved sprint velocity, as well as with reduced endurance. In stage II, girls with delayed skeletal maturation exhibited lower results in standing long jump and trunk forward flexion. The obtained results confirm the significance of sexual maturation in the development of motor capacities in girls.

Key words: biological maturation, motor abilities, pubescent girls

INTRODUCTION

Dependence of the development of motor abilities on biological maturation is widely assumed [2, 9]. Correlations between motor performance capacities and indices of skeletal and sexual maturation have been found in boys of age 13 to 16 years [4, 6]. However, in girls the corresponding correlation is low [3, 6]. Anyway, in early adolescence early maturing girls are stronger than their late maturing peers [3, 8, 9]. Significant correlations have been established between the results of 40-yard dash, standing long jump, throw for distance, agility (sidestepping) and ball catching, with skeletal but not with chronological age in primary-grade school children girls [10].

This study was aimed to check the dependence of the formation of motor capacities on biological maturation evaluated by stages of sexual maturation. For additional information attention was paid to skeletal age.

MATERIAL AND METHODS

Informed consent was obtained from 77 healthy girls of 10 to 15 years. Sexual maturation stages were evaluated according to Tanner's method [11]. Stages of development of pubic and axillary hair and breast were evaluated. If there were discrepancy between three evaluation, the final decision was made by the breast stage. Four maturation groups corresponding to stages I,II, III and IV were distinguished. Stage V was detected only in one girl. Results obtained in this girl were not considered in statistical analysis. When age groups were compared three 10-year-old girls and three 15-year-old girls were not considered. Skeletal age was assessed by x-ray picture of the left wrist and hand. The Greulich-Pyle method [7] was applied in the original manner without bone specific analysis. Therefore, the obtained information on skeletal maturation has to be considered as an addition but not the main. In order to evaluate the significance of deviations of skeletal age from chronological age, each group of sexual maturation was divided into three subgroups: (a) delayed skeletal maturation (bone

age one year or more behind chronological age), (b) bone age equal to chronological age, (c) advanced skeletal maturation (bone age one year or more ahead of chronological age).

Cooper's 12-min running test, Harvard step-test, shuttle-run 4×9 m, 20m dash, sit ups 30s, squats in 30s, standing long jump and trunk forward flexion were used for assessment of motor capacities. The tests were selected taking into consideration the results of a questionnaire with fitness experts of European countries [5] and the databank "SPODAT" [1]. The Harvard step-test was added to the battery recommended in "SPODAT".

One way ANOVA with t-test for independent unequal samples and ANOVA Simple Factorial were used for comparison of group values. Relationship of results of motor test with chronological age or with sexual maturing was tested also by the Pearson product-moment correlational analysis. 2-tail significance was evaluated designating the 0.05 probability level as significant.

RESULTS

Sexual maturation associated with a gradual increase in height and body mass up to the stage III (Table 1). The differences between groups of sexual maturation stages III and IV were statistically insignificant ($P>0.05$). Significant difference ($P<0.05$) in sprint velocity (results of 20 m dash) were found only if the groups as sexual maturation stages II and IV were compared. However, in results of shuttle run the results improved significantly ($P<0.05$) up to the stage III. Results of standing long jump and trunk forward flexion improved significantly ($P<0.05$) only receiving the stage III. Differences depending on sexual maturation stages were not found in number of sit ups, distance of 12-min run and Harvard step-test index. Sexual maturing (evaluated by the maturation stages) was in significant correlation with chronological age ($r=0.77$), height ($r=0.72$), body mass ($r=0.71$), results of 20m dash ($r=-0.22$), 4×9 m shuttle run ($r=-0.22$), standing long jump ($r=0.41$) and trunk forward flexion ($r=0.36$).

When the material was divided into age groups (Table 2), significant differences ($P<0.05$) were found between subsequent

groups in height (from 11 to 14 years), body mass (from 11 to 13 years), results of 20m dash (between 13 and 14 year olds), shuttle run (between 13 and 14 year olds), standing long jump (between 11- and 12- and 14-year olds) and trunk forward flexion (between 12 and 13 year olds). Age correlated with height (r=0.67), body mass (r=0.59), results of 20m dash (r=–0.27), shuttle run (r=–0.31), standing long jumps (r=0.45) and trunk forward flexion (r=0.28).

Table 1. Age, height, body mass, and motor abilities in pubertal girls depending on sexual maturation stage (mean±SD)

Test	Sexual maturation stage			
	I n=6	II n=44	III n=20	IV n=6
Age (years)	10.8±0.4*	11.5±0.8*	12.6±0.8*	14.2±0.8
Height (cm)	142.8±5.8*	149.0±6.1*	162.2±6.2	165.0±6.0
Body mass (kg)	31.0±4.3*	38.1±5.5*	49.6±5.2	48.7±4.6
20m dash (s)	4.5±0.4	4.2±0.3	4.2±0.3	4.0±0.3
4×9 m shuttle run (s)	13.3±1.1*	12.2±1.0*	11.9±1.1	12.2±1.5
Standing long jump (cm)	145±21	156±16*	170±23	172±21
Squats in 30s (no)	26±4	27±3	29±3	27±3
Sit-ups in 30s (no)	18±3	21±4	19±4	18±3
Trunk forward flexion (cm)	7.0±3.6	9.3±4.8*	14.2±3.5	11.0±4.0
Cooper 12-min run (m)	2088±223	2238±297	2274±284	2157±349
Harvard step-test index	82±9	83±10	83±10	81±8

* significant difference ($P<0.05$) between subsequent groups

Table 2. Height, body mass, and motor abilities in pubertal girls depending on chronological age (mean±SD)

	Chronological age (years)			
	11 n=29	12 n=25	13 n=11	14 n=6
Height (cm)	147.2±6.1*	153.0±8.3*	161.7±6.0*	167.2±3.9
Body mass (kg)	36.9±6.0*	41.6±7.6*	50.1±5.9	47.2±3.0
20m dash (s)	4.3±0.4	4.2±0.3	4.3±0.3*	3.8±0.2
4×9 m shuttle run (s)	12.5±0.9	12.0±1.1	12.4±1.3*	11.2±0.3
Standing long jump (cm)	152±17*	162±17	167±26	178±20
Squats in 30s (no)	21±4	20±5	19±4	18±4
Trunk forward flexion (cm)	9.5±4.9	10.0±4.8*	14.0±4.6	13.2±3.3
Cooper 12-min run (m)	2208±327	2280±230	2169±380	2366±220
Harvard step-test index	83±9	84±10	89±14	80±6

* statistically significant difference between subsequent age groups

One-way ANOVA indicated significant differences in distribution of individual data between either sexual maturation or age groups in age height, body mass, results of 20m dash (only between age groups), shuttle run, standing long jump and trunk forward flexion. ANOVA Simple Factorial demonstrated that differences between sexual maturation groups disappeared in trunk forward flexion but not in standing long jump and shuttle run when age was used as cofactor. Using height or weight or cofactor all differences between distribution of results of motor tests disappeared (Table 3). Differences between age groups disappeared if either sexual maturation, height or body mass were cofactors (Table 3). The exception was only trunk forward flexion.

Table 3. ANOVA Simple Factorial Analysis of distribution of individual data (F values) on results of motor tests between groups distinguished either by sexual maturation stages (the upper part of the Table) or by age (the lower part of the Table)

	20 m dash	4×9 m shuttle run	Standing long jump	Trunk forward flexion
Groups of sexual maturation stages				
One-way ANOVA	1.8	2.6*	5.0*	7.3*
ANOVA Simple Factorial				
Cofactor age	2.2	6.8*	5.5*	0.2
Cofactor height	1.9	0.5	0.6	0.1
Cofactor body mass	0.8	2.0	0.9	0.1
Groups of chronological age				
One-way ANOVA	3.1*	3.5*	3.5*	2.8*
ANOVA Simple Factorial				
Cofactor sexual maturation	0.2	0.1	0.9	5.6*
Cofactor height	0.1	0.2	1.0	2.8*
Cofactor body mass	0.1	0.2	0.1	2.7

* F value, indicating statistically significant difference in distribution of results between groups

When girls of delayed or advanced skeletal maturation were distinguished, no differences were statistically significant ($P>0.05$), except lower results in the flexibility test and standing long jump in girls of stage II with delayed skeletal maturation compared to girls with equal skeletal and chronological age

(forward flexion 9.3±2.0 and 14.3±1.5 cm and long jump 160±2 and 170±3 cm, respectively). A similar but insignificant difference was found in the results of standing long jump in girls of stage III (162±10 cm in girls with delayed and 178±7 cm in girls with normal skeletal maturation). However in stage IV a tendency to the opposite difference was found (163±6 cm in girls with advanced maturation, compared to 178±8 cm in girls of normal skeletal maturation).

DISCUSSION

The obtained results confirm the dependence of the development of motor capacities on biological maturation in girls. Advanced sexual maturation associated with an increase in running speed (20 m dash and shuttle run), explosive power (standing long jump), and flexibility (trunk forward flexion). These findings are comparable with the results that (1) early maturating girls are stronger than their late maturating peers in early adolescence [3, 8], (2) improvement in flexibility from age 12 to 13 years in early and average maturing but not in late maturing girls [9]. Results of Espanschade [6], obtained in 1940, indicate on best results in standing long jump 1.5 years before the menarche. However by her results a new improvement in standing long jump occurs 0.5 years after the menarche, which appears in most girls in sexual maturation stage III [9]. Obviously, the improvement of results of standing long jump in stage III, observed by us, is related to the secondary improvement of results in the study of Espenshade [6].

When age groups were considered, most of significant differences in results of motor test were found between 13- and 14-year old girls, thus a year later than between sexual maturation group taking into the account the mean age of the groups. This was related to different distribution of girls of advanced sexual maturation between age groups. Thus the sexual maturation seems to be more essential determinant of motor development than chronological age in pubertal girls. This suggestion was confirmed by results of ANOVA Simple Factorial: when age was used as cofactor the differences in distribution of individual data in results of shuttle

run and standing long jump between sexual maturation groups did not disappear. However, when the significance of sexual maturation was considered, the differences between age groups by shuttle run and standing long jump became insignificant. The dominating significance of age seems to exists in regard of trunk forward flexion.

In a previous study significant correlation of various motor capacities was found with skeletal but not with chronological age in primary-grade school children [10]. Our attempt to distinguish the girls of delayed and advanced skeletal maturation did not provide evidence confirming the dependence of motor development on skeletal maturation. However, in our study the number of girls with delayed or advanced skeletal maturation was only 5 to 12 in groups of various sexual maturation. Moreover, in most cases the difference between skeletal and chronological age was only one year. Therefore, our results cannot be considered conclusive in regard to the significance of skeletal maturation.

In conclusion, achieving of the sexual maturation stage III associates with an improvement of explosive strength of leg muscles, result of shuttle run, and trunk forward flexion. In stage IV sprint velocity reaches the highest level.

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