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MALLEABILITY OF THE MOTOR SYSTEM: A COMPARATIVE APPROACH

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SUMMARY

The various ways in which the power output of muscles can be changed are described. As a result of exercise and growth, force production is increased by an increase in the cross-sectional area of the fibres. This is associated with changes in the rate of synthesis and degradation of muscle proteins which lead to build up of the myofibrils. These then split longitudinally when they reach a critical size. This process is repeated so that the number of myofibrils increases very considerably. Also, during growth, the displacement is increased by increasing the length of the muscles. To do this more sarcomeres are produced in series along the length of the fibres. This is induced by stretch which also encourages fibre growth in girth as well as in length. Yet another way of changing the power output of a muscle is to change the types of muscle fibres (motor units) within the muscle. Fibre type transformation has been shown to occur with cross innervation and stimulation but it does not usually occur with exercise training. It has been possible, however, to change the fibre type proportions in young animals. Also, by combining stretch with stimulation, it has been possible for instance to make the fast glycolytic fibres add on fast oxidative type sarcomeres or even slow oxidation type sarcomeres. Interestingly, fibre transformation also occurs in some species of fish during acclimation to low temperatures in that the specific myofibrillar ATPase activity is increased. This means that the reduction in power output due to decreased temperature is to some extent compensated for by an increase in the intrinsic rate of shortening. EMG studies of fish swimming at different temperatures have shown that the acclimated fish can swim faster and can derive more aerobic sustainable power as a result of this change.

Adaptability may be regarded as a basic characteristic of life. The types of living organisms that have survived the course of evolution have, by and large, been those which have been the most adaptable. Prokaryotic organisms show considerable ability to alter their metabolism in response to changes in substrate or environmental conditions. These changes are brought about by relatively rapid changes in gene expression. Cells and tissues in eukaryotic organisms, particularly higher animals, are usually regarded as less adaptable because they exist in more stable environments. However, in recent years, evidence has been accumulating which demonstrates more and more just how adaptable animal cells are. For example, increased enzyme levels

Key words: Muscle, adaptation, mechanical activity.

can be induced in many tissues in response to changed activity. The number of receptor and transport molecules in membranes can be altered by changing the ion content of the fluids and the endocrine status of the animal.

Perhaps the most adaptable cells are those in tissues which produce or are subjected to mechanical stress. For example, the cortical thickening, trabecular density and curvature of bone tissue are known to be determined to a large extent by the intermittent dynamic loads that occur during locomotion. The degree of keratinization of skin is determined by the extent of surface friction as well as by location, so skin cells too can detect mechanical stimuli. Fibroblasts also are apparently responsive to mechanical factors as connective tissue within muscles is increased in thickness in response to overload. Of all the tissues, skeletal muscle is probably the most adaptable. Unlike the other tissues that respond to mechanical stimuli, muscle fibres create the mechanical stresses as well as responding to them, so it is sometimes difficult to discern which is the cause or the effect when studying muscle adaptation. This is particularly true for growing muscle where some of the changes may be regarded as adaptation whilst others may be changes that are strictly programmed for in the DNA of the cell (see Whalen, 1985).

This paper is concerned with the adaptation for power production. Power is work done per unit time and of course work is force times displacement (length change).

$$\text{Power} = \frac{\text{Force}^{(1)} \times \text{Length change}^{(2)}}{\text{Time}^{(3)}}$$

All three of these parameters may change as a result of adaptation for increased power but they do not necessarily change simultaneously.

(1) The force a muscle can develop is proportional to its fibre cross-sectional area or more strictly to its myofibril cross-sectional area. This changes a great deal during growth and as a result of certain types of exercise training.

(2) The length of fibres increases during growth, hence the overall rate of shortening of the muscle is increased. This means for example that a greater stride length or tailbeat amplitude is possible.

(3) A faster contracting muscle is able to develop more power because it can generate force many more times a second. Therefore, another way of altering power production is to change the overall speed of contraction of the muscle.

There are clearly some constraints in the design of musculoskeletal systems and these have apparently operated during evolution. As limbs have lengthened during evolution there had to be a trade-off between length of shortening and rate of shortening. If muscles in large animals were to shorten at the same intrinsic speed as those in small animals, the strain rates on the bones and tendons would have caused the animals to be pulled apart. Also, there would be a mismatch of these muscle parameters, and the resonance or harmonics of the limb system (see McMahon, 1985 for the concept of tuned springs), so there are probably mechanical design limits for the alteration of parameters (2) and (3).

Physiologists talk about two types of power; one is *sustainable power output* and

this depends on the characteristics of the energy supply mechanism of the muscle fibres as well as their contractile parameters. As aerobic pathways, particularly the oxidation of lipids, supply so much more energy than anaerobic pathways, sustainable power is essentially the same as *aerobic power*. This type of power output will be dealt with near to the end of the paper when discussing fish swimming. The other type of power is *maximal power* and this is important for short-duration activities such as sprinting, fighting and lifting heavy loads. These levels of power output involve the recruitment of most of the muscle fibres in a muscle, some of which have very poor fatigue resistance and therefore their power output cannot be maintained. Also oxygen cannot be supplied to the muscle at a sufficient rate to sustain aerobic metabolism and even if it could it is unlikely that the ATP hydrolysed by the fast anaerobic fibres could be replenished at the same rate.

In discussing adaptation for power, the subject of energy supply will not be dealt with as other authors have gone into this in considerable depth. Instead, I have chosen to consider the changes in protein metabolism and structural malleability that lead to an overall increase in muscle power during growth or exercise training. In particular, to show how rapidly muscle protein synthesis can be switched on and how the large complex protein molecules can be accumulated and assembled into sarcomeric structures. As mentioned, one of the fascinating aspects of muscle is its ability to respond to mechanical stimuli, so this paper will deal mainly with the adaptive changes to force development and stretch although the metabolic adaptation to changes in environmental temperature will also be mentioned.

ADAPTATION TO FORCE PRODUCTION

This is perhaps the most obvious type of muscle adaptation as it is quite apparent that weightlifters and sprinters develop very large muscle masses. Nevertheless, this

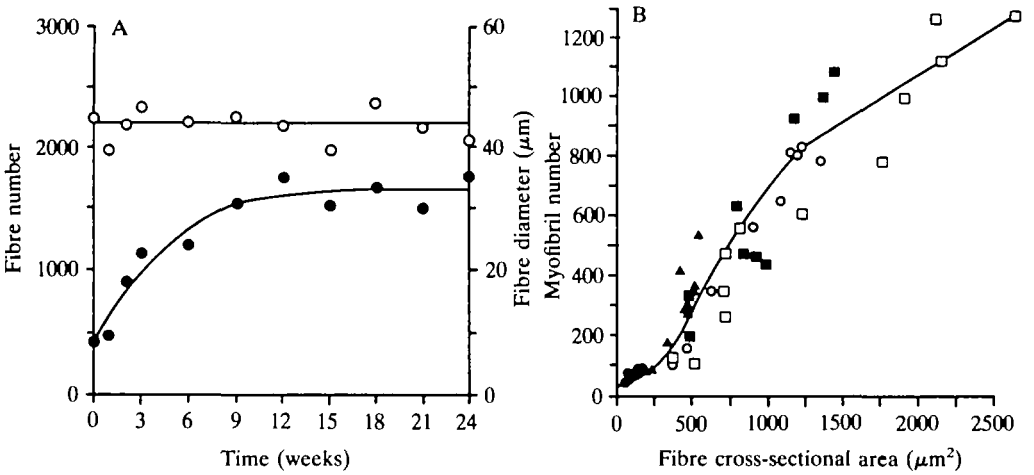

Fig. 1. (A) Fibre number (○) and fibre diameter (●) during post-natal development in the muscle biceps brachii. (B) Increase in the number of myofibrils per fibre during growth. The different symbols represent fibres taken from the biceps brachii muscle at different ages.

adaptive response is most important in increasing muscle girth during post-natal development and maintaining muscle mass in the adult. The nature of the link between the physical events involved in force development and the 'switching on' of protein synthesis is an interesting topic for investigation. As yet, very little is known about this but we do know something about the structural changes which occur during the increase in muscle girth.

Briefly, it has been shown that the number of muscle fibres does not change during post-natal growth or as a result of exercise training at reasonable intensity levels. However, the mean cross-sectional area of the existing fibres increases considerably and this is associated with a large increase in the myofibrillar content of the fibres. The myofibrillar mass becomes subdivided as it increases in volume, hence the number of myofibrils increases (Fig. 1). This allows the sarcoplasmic reticulum and transverse tubular systems to invade the mass and to surround individual myofibrils. During growth the number of myofibrils within a muscle fibre increases from just a few at the myotube stage to over a thousand in a mature muscle fibre. This proliferation results from the longitudinal splitting of existing myofibrils. There is a built-in mismatch between the actin and myosin lattice so that the actin filaments are slightly displaced as they run from the Z disc (square lattice) to the A band (hexagonal lattice) (Fig. 2). This displacement or oblique pull of the actin filaments causes a mechanical stress to occur in the centre of each Z disc which results in splitting of the myofibril (Goldspink, 1970, 1971).

This splitting tends to be more complete in fast-contracting fibres and therefore the myofibrils in these fibres are small and punctate (fibrillenstruktur). In slow-contracting fibres the splitting is often incomplete and therefore the myofibrils appear branched in longitudinal section (Shear & Goldspink, 1971).

An increase in myofibril cross-sectional area and total number of myofibrils occurs during growth and during certain types of exercise training. The maximum force production of a muscle is related to the myofibril cross-sectional area so that the physiological significance of this type of malleability is apparent. However, we still need to ask what biochemical changes are occurring in an overloaded muscle which cause it to respond by producing more myofibrillar proteins so that more myofibrils can be assembled. There are two main ways in which proteins can be accumulated during growth or exercise training. One way is to increase the rate at which proteins are synthesized. The other is to decrease the rate at which they are broken down. Even in adult muscle proteins are constantly being synthesized and broken down and the turnover, or half-life, of the contractile proteins is of the order of 7–15 days. The soluble sarcoplasmic proteins have even shorter half-lives. A process in which more than half of the contractile proteins are broken down and replaced every 7 days or so would seem to be rather wasteful. However, it does enable the muscle to replace

Fig. 2. (A) Examples of splitting myofibrils in exercised, rapidly growing muscle. Magnification, approximately ×30 000 and 15 000. (B) illustrates the way in which the myofibrils are believed to increase during growth and exercise training. The oblique pull of the more peripheral thin filaments is believed to cause the Z discs to rip and hence each myofibril divides into two or more daughter myofibrils. Data from Goldspink, 1971.

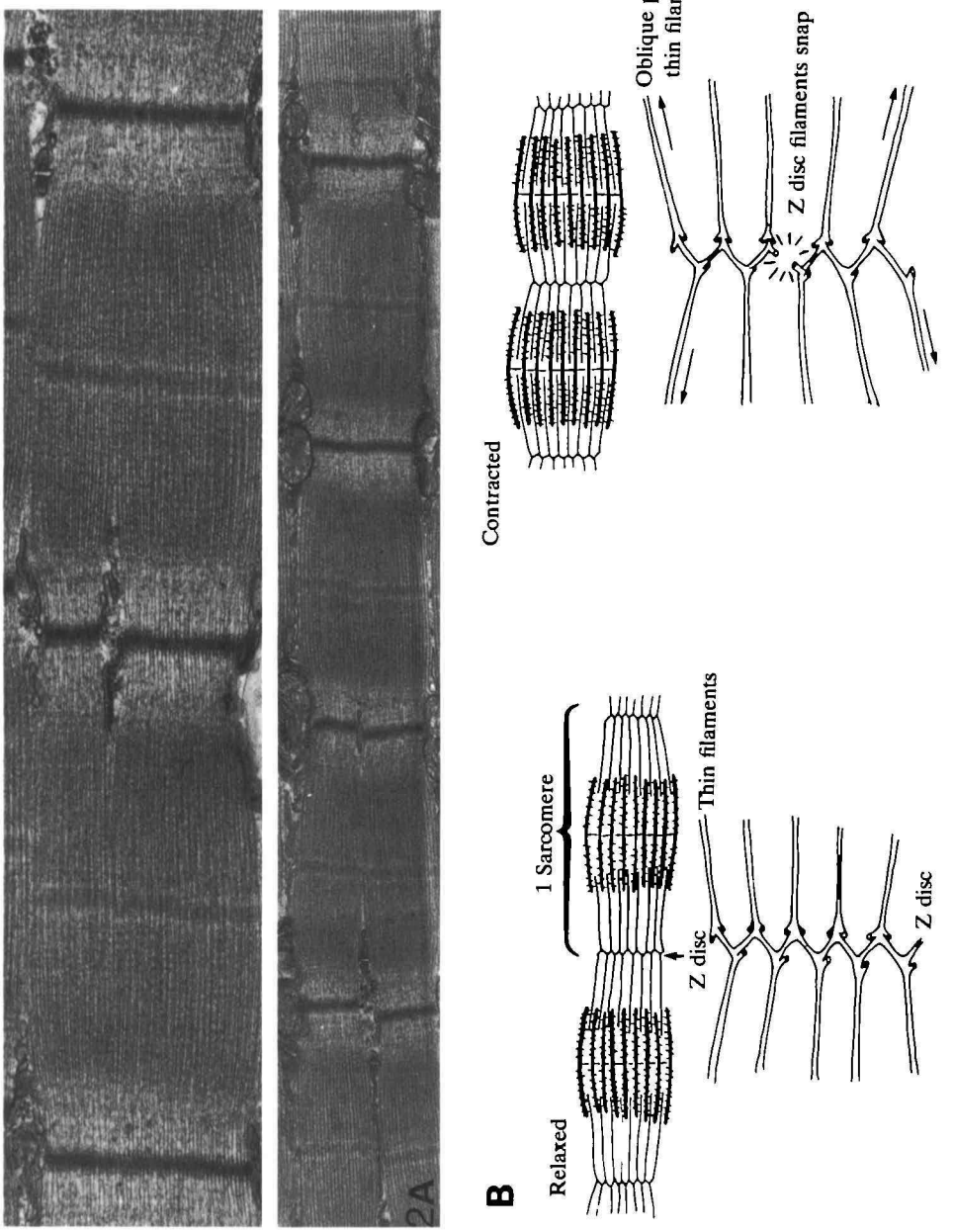
2A
B
Relaxed
1 Sarcomere
Z disc
Thin filaments
Z disc
Contracted
Oblique pull of thin filaments
Z disc filaments snap

Fig. 2

damaged proteins and it confers on the muscle a certain adaptability for changing the type of protein at certain stages of development and under certain physiological conditions.

Changes in the balance between protein synthesis and degradation resulting from exercise training were investigated using a method in which young rats had to jump for their food (Watt, Kelly, Goldspink & Goldspink, 1982). This caused considerable hypertrophy of the hindlimb muscles (approximately 30% increase in muscle fibre cross-sectional area in the soleus and extensor longus muscles). The animals were exercised for 1 month before measuring the changes in synthesis and degradation rates. In the extensor digitorum longus muscle, which is made up of predominantly fast fibres, the accumulation of muscle proteins occurred mainly as a result of an increased synthesis rate although the degradation rate was slightly decreased. Interestingly, the soleus muscle, which is a slow contracting postural muscle, exhibited a different strategy. In this case it was the degradation rate which significantly changed whilst the synthesis rate was only slightly elevated. It is known that the energy metabolism of these two types of muscle differs. Therefore, it is interesting to see that the way they handle protein also apparently differs. It is also interesting to note that these different types of muscle fibres respond somewhat differently to inactivity and starvation. The slow fibres tend to atrophy considerably in response to zero gravity, as occurs during space flight, and yet they are very resistant to the effects of starvation and to certain diseases such as muscular dystrophy. These differences may therefore be due to differences in their protein metabolism.

All the types of muscle fibres are capable of undergoing hypertrophy but they do not usually hypertrophy to the same extent. The fast contracting fibres are recruited only infrequently (for rapid power movements or high intensity isometric contractions). When they are recruited and 'overloaded', they tend to undergo hypertrophy very readily (Fig. 3). Therefore, the cross-sectional area of the fast fibres is increased even though the total number stays the same. This means that there are more fast-type sarcomeres in series and in parallel and hence the muscle is not only capable of producing more total force but it is capable of developing that force more rapidly. Selective hypertrophy of the fast fibres can thus be regarded as adaptation for increased power production under situations when all or most of the fibres are being recruited.

The slow fibres may also increase in size as a response to very frequent recruitment, but to a lesser extent than the fast fibres. In repetitive low-intensity exercise, the fast fibres may hardly ever be recruited. Under these conditions they may atrophy at the same time as the slow fibres are undergoing some slight hypertrophy (for example long duration treadmill running or long distance cycling, Fig. 3). Thus, there is a selective response depending on the type of training.

The other way muscle fibres respond to repetitive type training is to produce more mitochondria and oxidative enzymes. Accompanying this there is also an increase in the number of capillaries per fibre (see Hoppeler & Lindstedt, 1985). In our experience both the slow-contracting and the fast-contracting fibres may respond by increasing their oxidative enzyme levels (Goldspink & Waterson, 1971). This type of mutability therefore confers increased fatigue resistance on the muscle and hence an increased aerobic power output.

The question is often asked as to whether hyperplasia occurs as well as hypertrophy in response to strenuous exercise training. In our experiments using normal types of exercise we have not found any change in the total number of fibres or the ratio of fast- to slow-contracting fibres (Goldspink & Ward, 1979). We did however observe splitting muscle fibres in surgically overloaded muscle, that is to say, removal of a large synergistic muscle (Vaughan & Goldspink, 1979). It is therefore possible that

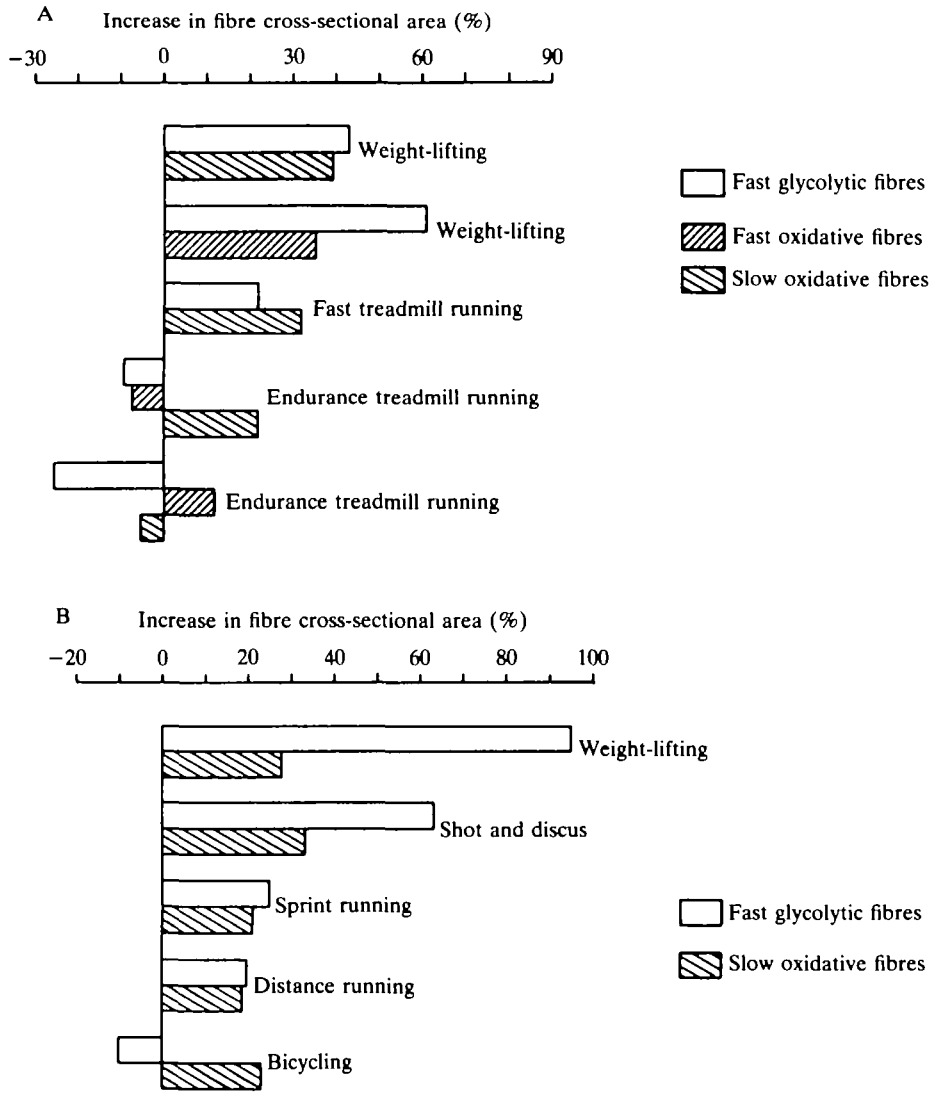

Fig. 3. (A) The percentage increase in the cross-sectional area of different muscle fibre types resulting from exercise training in experiments involving rodents. Data derived from the author's laboratory. (B) The percentage increase in the cross-sectional area of different muscle fibre types associated with different types of athletic training in man. Data derived from quadruceps biopsies and values for athletes are expressed as an increase or decrease of those for sedentary controls (zero) and are taken mainly from the work of Gollnick and Saltin.

splitting may lead to hyperplasia, under for example, conditions of repeated, incremental exercise. As far as fibre conversion is concerned, that is, the ability of one type to change into another (for example, to change from a fast glycolytic type to a slow oxidative type), this is still an unresolved question. Fibre conversions can be achieved by chronic electrical stimulation and it is possible that chronic exercise regimes which involve many hours of training per day may have this effect. Just recently we have managed to bring about interfibre conversion in some muscles of young rats by subjecting them to very different types of early activity (Watt, Goldspink & Ward, 1984). This may reflect the increased plasticity in the young animal. It may also indicate that early activity may determine what type of muscle fibres develop in particular anatomical muscles. Certainly, it is known that some change in the ratios of the different fibre types does occur during the growth of muscles and it is usually the predominant fibre type that increases, possibly indicating that some re-innervation takes place (Goldspink & Ward, 1979).

LENGTH CHANGES AND RESPONSE TO STRETCH

Work in the author's laboratory has demonstrated that stretch is a very important promoter of muscle growth. Earlier work with the Tardieus and Tabarys and with Pamela Williams showed that if adult muscle fibres are stretched by immobilizing the muscle in its lengthened position, then the fibres add on 20 or 30 % more sarcomeres in series. Studies with radioisotope precursors have shown that the new sarcomeres are added onto the ends of the existing myofibrils (Fig. 4). Conversely, if the muscles are immobilized in the shortened position, they lose 20–30 % of their sarcomeres in series so the fibre becomes shorter. In other words the sarcomere number is adapted to the new functional length of the muscle (Williams & Goldspink, 1971; Tabary *et al.* 1972). The changes involving the addition or removal of sarcomeres are rapid and completely reversible (Williams & Goldspink, 1973).

There are also other changes associated with the change in sarcomere number. In particular, the muscles held in the shortened position develop a much higher compliance and become inextensible and this has been shown to be the result of a remodelling of the connective tissue. Indeed, recent data (Williams & Goldspink, 1984) suggest that the connective tissue changes before the fibres alter length by sarcomere loss (Fig. 5). This is surprising because connective tissue was thought to be a very stable component with a long turnover time. However, these results suggest that the connective tissue is remodelled within a period of less than 1 week and that the muscle fibres fit into the new framework by losing sarcomeres (presumably at the ends) until the optimum sarcomere length is restored. In the converse situation, the question as to how the 'muscle fibres know' what number of sarcomeres they should

Fig. 4. (A) The change in the number of sarcomeres along the muscle during growth and as a result of immobilization in the lengthened and shortened postions. (B) Data from radioisotope precursor experiments which show the incorporation of radioactive amino acids during the lengthening process in growing muscle and in muscle recovering from immobilization in the shortened position. These and other data show that the new sarcomeres are added onto the ends of the muscle fibres.

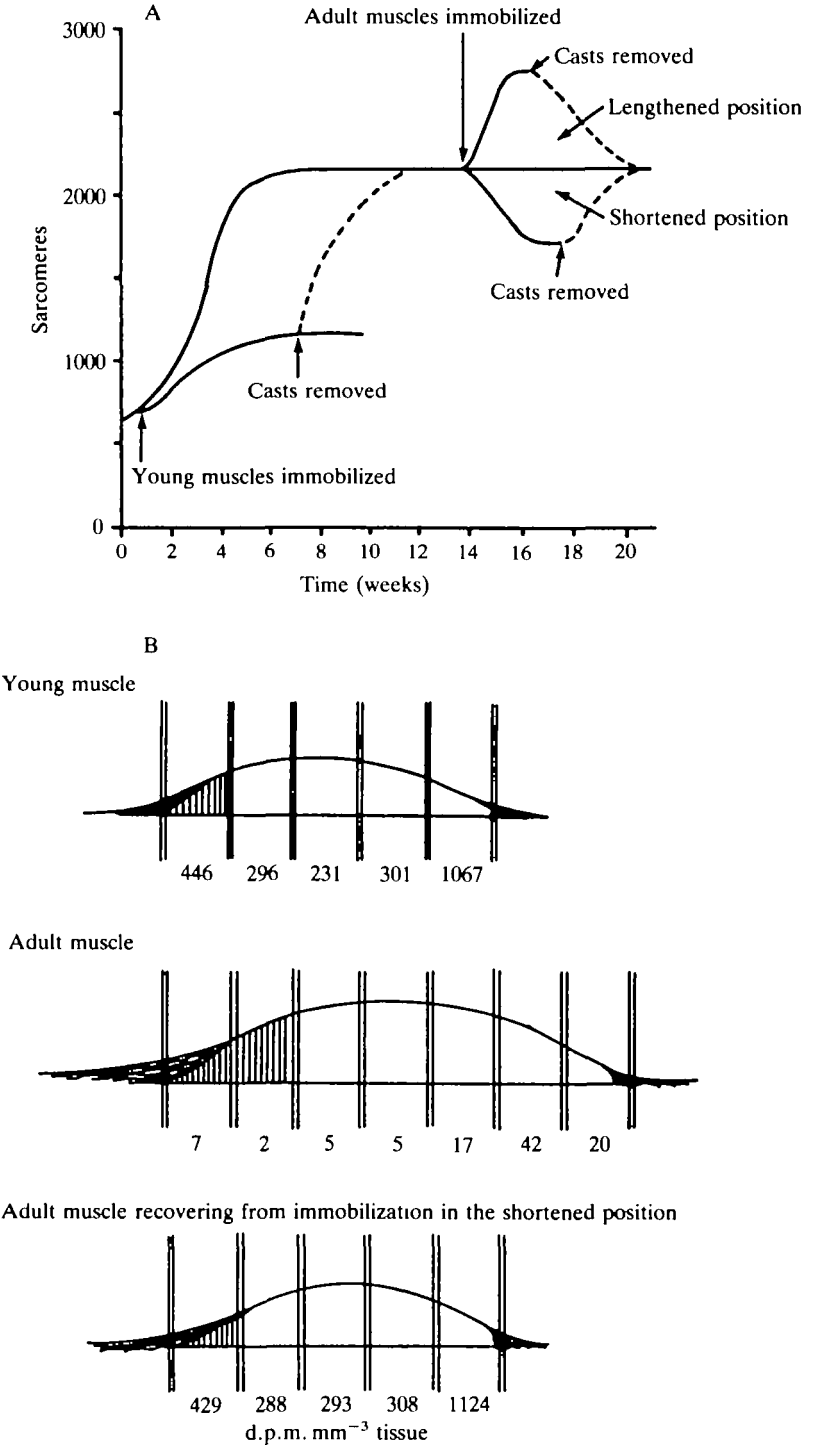
A
Adult muscles immobilized
Casts removed
Lengthened position
Shortened position
Casts removed
Casts removed
Young muscles immobilized
Sarcomeres
3000
2000
1000
0
0 2 4 6 8 10 12 14 16 18 20
Time (weeks)
B
Young muscle
446 296 231 301 1067
Adult muscle
7 2 5 5 17 42 20
Adult muscle recovering from immobilization in the shortened position
429 288 293 308 1124
d.p.m. mm^{-3} tissue

Fig. 4

add on along their length, has been investigated. An obvious candidate for detecting the length change is the sensory nervous system *via* reflex pathways. However, it was shown that denervated muscle could still adapt to a length change by adding or removing sarcomeres (Goldspink *et al.* 1974). The way the sarcomeres adjust to the optimum sarcomere length after being pulled out to a greater length is not known but it may involve the short-range forces (spurious crossbridge activity in the resting muscle). This would tend to make the myofibrils shorten and to leave a gap at the end where new sarcomeres can be added. In any event the detecting mechanism for the length change seems to be in each individual fibre.

In addition to promoting longitudinal growth in the form of addition of sarcomeres, stretch also promotes the increase in the cross-sectional area of the fibres (Fig. 6). In some recent experiments in our laboratory, we have combined electrical stimulation with stretch (immobilization in the shortened position) using rabbit muscles. By involving these two parameters we have been able to obtain increases in muscle weight of approximately 30 % within a period of only 7 days (Fig. 7). As seen from Fig. 8, stimulation (5 Hz continuous) only produced an increase in fibre girth of the slow oxidative fibres but this type of stimulation is expected to be less effective in promoting hypertrophy of fast glycolytic fibres than short trains of high-frequency stimulation. (The results of these latter experiments are at present being analysed.) In the rabbit anterior tibialis stretch plus stimulation produced hypertrophy of all three fibre types and this, combined with the addition of almost 2000 sarcomeres in series, is why such a rapid increase in muscle mass occurs. As we know the number of additional sarcomeres we can calculate approximately the number of molecules of myosin produced in this 7-day period. The number of new myosin molecules is approximately 2000 (sarcomeres) × 1000 (myofibrils) × 500 (filaments per myofibril)

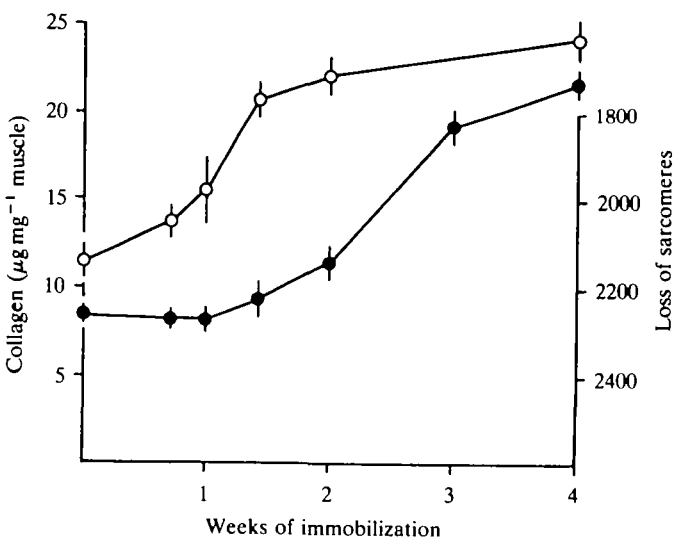

Fig. 5. The build-up of endomysium and perimysium connective tissue (open circles) and the loss of sarcomeres (closed circles) during adaptation to a shorter functional length. Note that the connective tissue framework is remodelled before sarcomere loss occurs. Data from Williams & Goldspink, 1984.

× 300 (myosin molecules per filament). This represents a synthetic rate of approximately 30 000 000 molecules per fibre per minute. If these are related to the number of nuclei in the fibre (approximately 1000) then each nucleus is directing the synthesis of approximately 30 000 myosin molecules per minute. These rates are, however, probably no higher than the synthesis rates associated with turnover if one takes the half-life of muscle proteins as being 7 days.

In some experiments in which we have attempted to simulate zero gravity conditions using the 'suspended hindquarter model' (rats are fitted with a jacket which is attached to a moveable arm and only their forelimbs are allowed to touch the floor of the cage), we found that soleus muscle atrophies by approximately 38 % in 5 days. This atrophy could however be completely arrested by immobilizing the muscle in its stretched position. Therefore, there seems to be little doubt that stretch is a most important factor in regulation and maintaining muscle size.

SWITCHING OF SYNTHESIS OF MUSCLE PROTEIN ISOFORMS

As mentioned above the contractile proteins exist as different isoforms. There are different forms of the myosin heavy chain and the myosin light chains, actin, troponin and tropomyosin. These different forms are associated with cardiac, slow tonic, slow

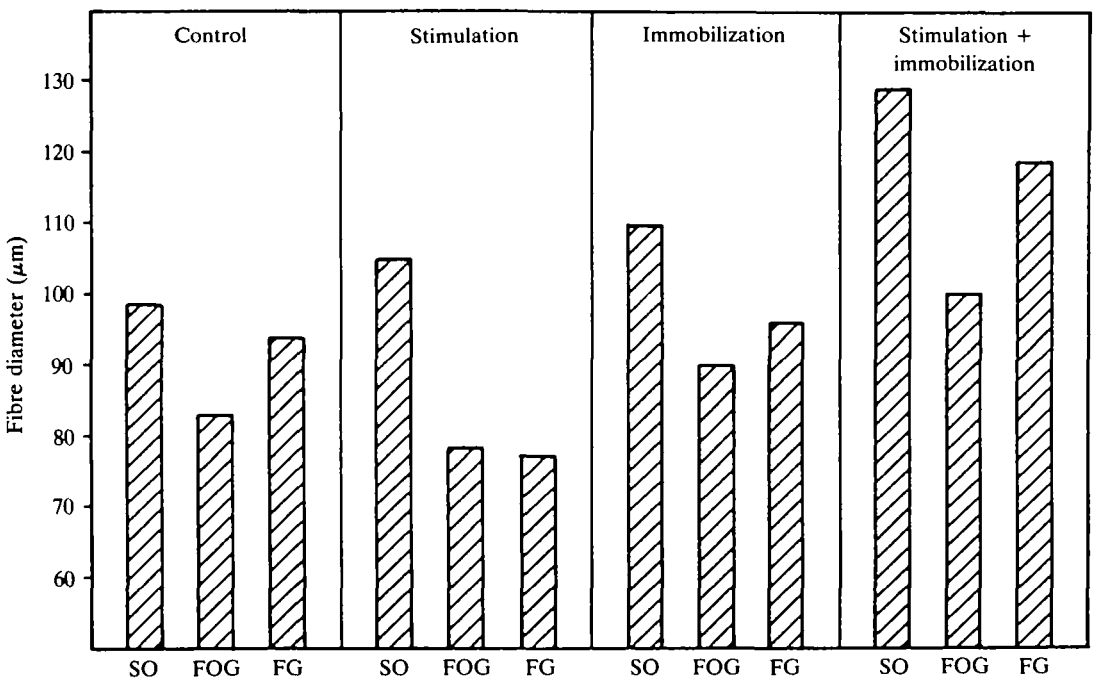

Fig. 6. The effect of stretch combined with stimulation on the fibre diameter of the different fibre types within the anterior tibialis muscle of the rabbit. SO, slow oxidative; FOG, fast oxidative glycolytic; FG, fast glycolytic. Data from P. Williams, P. W. Watt, V. Bicik & G. Goldspink, in preparation.

twitch and fast twitch muscles. So it is assumed that they are involved in determining the different characteristics of these muscle types.

MUSCLE MUTABILITY AND POWER OUTPUT AT DECREASED ENVIRONMENTAL TEMPERATURES

It is often assumed that poikilothermic animals are completely dependent on temperature as far as their metabolism and activity are concerned. However, in recent years it has become evident that certain poikilotherms have evolved strategies for reducing this dependence and for sustaining locomotory ability at decreased temperatures. For instance, our group has shown that some species, e.g. carp and perch, are

Fig. 7. The increase in size of the anterior tibialis muscle of the rabbit within 7 days, when it is stretched and stimulated. The 28 % increase in mass is due to addition of sarcomeres and hypertrophy of all three fibre types. Magnification, ×2 approximately.

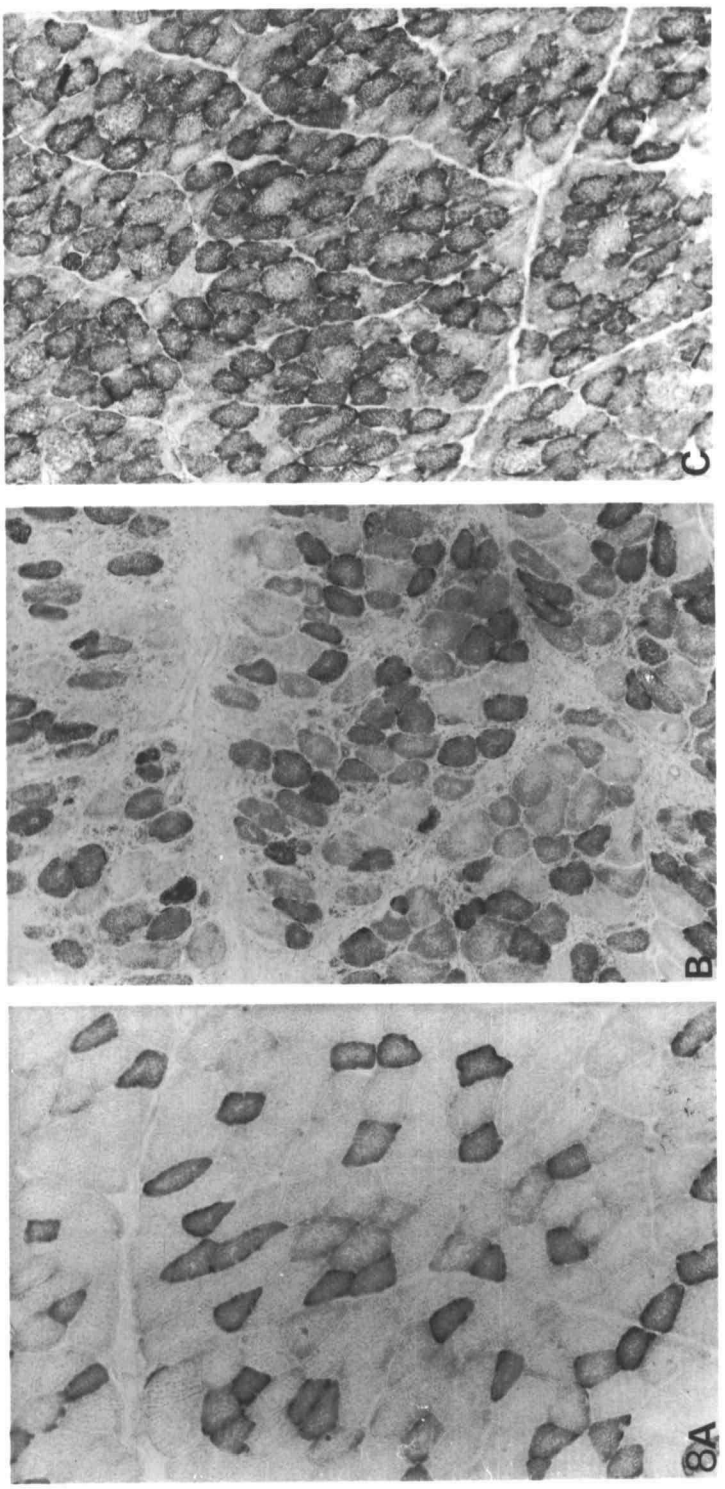

Fig. 8. The transformation of fibre type at the end regions of the fibres. The transformation is achieved only when the muscles are stimulated whilst being stretched. Staining in this case is for succinic dehydrogenase (oxidative capacity). (A) Control; (B) stretched only control; (C) stretched and stimulated (5 Hz). Magnifications, ×approx. 200. Data from P. Williams, P. W. Wall, V. Bicik & G. Goldspink, in preparation. Data for myosin ATPase staining and transformation of contractile protein isoforms will be published elsewhere.

able to produce a different set of myofibrils for low-temperature swimming. Following 1 month or so of exposure to a low environmental temperature, the contractile proteins of these species change so that the specific myofibrillar ATPase is much higher at low temperatures than for non-adapted fish (Johnston, Davison & Goldspink, 1975*a*; Johnston, Walesby, Davison & Goldspink, 1975*b*; Penney & Goldspink, 1981*a*,*b*). Cold-acclimated carp have a myofibrillar ATPase that is approximately three times higher than that of non-acclimated fish. This presumably means that muscle contraction can proceed at a faster rate than otherwise would be the case. Associated with these changes in myofibrillar ATPase, there is also a change in the thermal stability of the myofibrillar system (Johnston *et al.* 1975*a*) (Fig. 9). It seems that one of the consequences of producing contractile proteins with a higher ATPase is that the molecular structure is a more open one and is more susceptible to thermal denaturation (Johnson *et al.* 1975*a*).

Compensations in the metabolic enzymes of fish to changes in environmental temperature have been well documented by Hochachka & Somero (1973), Hazel & Prosser (1974) and Somero (1978). Some of these changes involve the expression of different isoenzymes and therefore it is reasonable to postulate that the changes in the myofibrillar system also involve the production of different species of the same proteins.

Electromyographic studies have shown that fish recruit their red (slow oxidative) muscle for slow cruising but as the speed of swimming is increased, they progressively recruit their pink (fast oxidative) and eventually their white (fast glycolytic) musculature (Davison, Goldspink & Johnston, 1976). At low environmental temperatures, the power

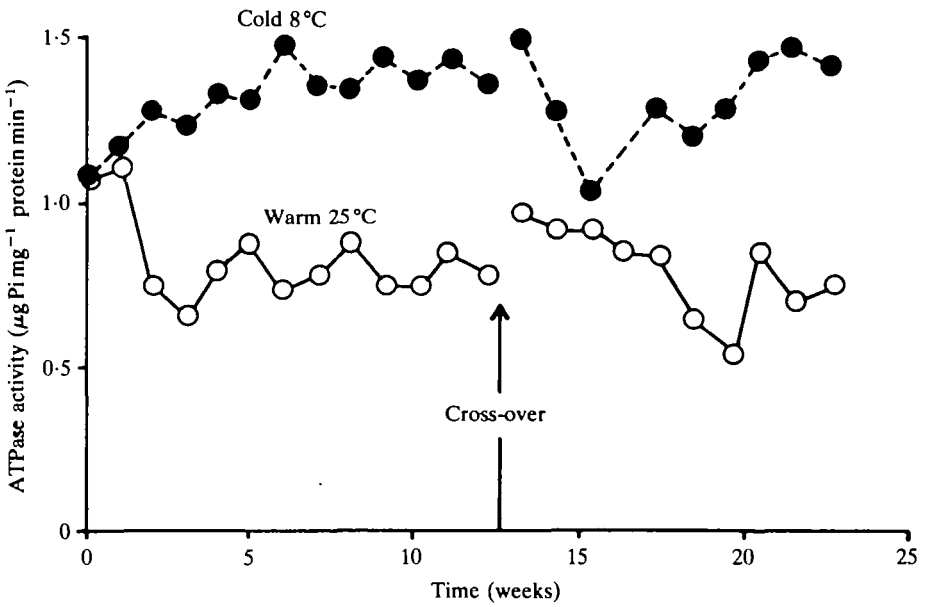

Fig. 9. The changes in the specific myofibrillar ATPase activity in carp myotomal muscle that occur during temperature acclimation. The increased ATPase is associated with an increased rate of shortening and hence an increase in power output of the muscle at low temperatures. Data from Heap, Watt & Goldspink, 1985.

output of the red muscle is reduced and therefore non-acclimated fish have to recruit their white musculature even at low swimming speeds (Rome, Loughna & Goldspink, 1984).

In species that can acclimate by increasing their myofibrillar ATPase (hence increasing their intrinsic rate of contraction of the red fibres), the recruitment thresholds of the white fibres (Table 1) have been shown to be considerably higher (Rome, Loughna & Goldspink, 1985) (Fig. 10). The red fibres are aerobic fibres and it is an advantage to the fish to derive as much power from aerobic means as possible before it has to recruit its anaerobic fast-type fibres. In acclimation, the increase in the power production of the red fibres is derived from the change in the myofibrillar

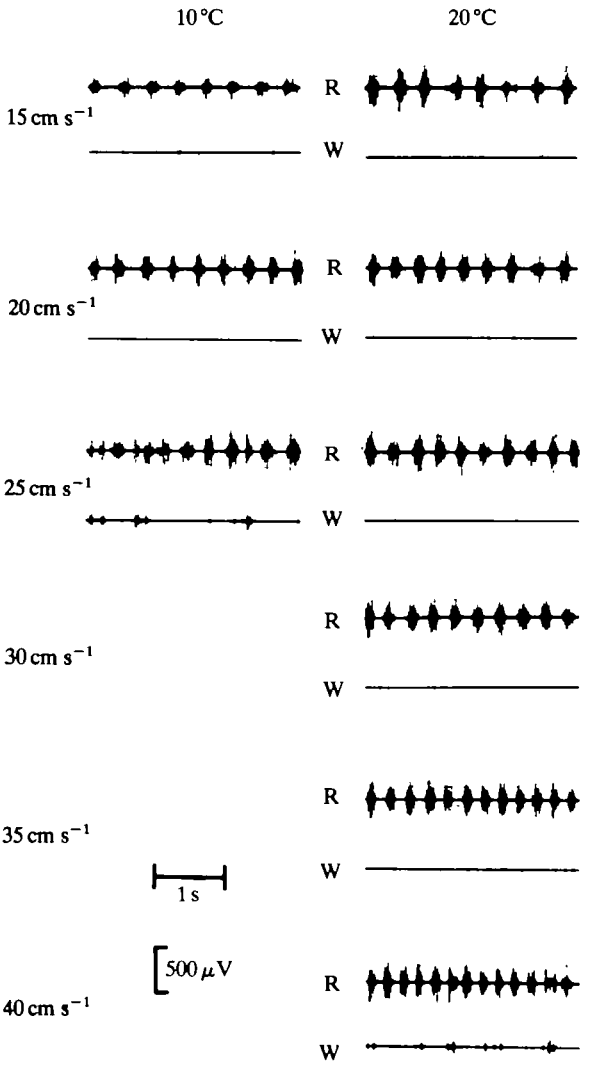

Fig. 10. The way fish maintain power output when the temperature fluctuates. When the temperature is lowered the white glycolytic fibres are recruited earlier (at lower swim speeds). R, red; W, white. Data from Rome, Loughna & Goldspink, 1985.

Table 1. *The swim speed at which the white fibres are recruited at 10 °C and 20 °C in fish acclimated to different temperatures*

Temperature at which fish were acclimated (°C)	White muscle recruitment speed at 10 °C ($cm\,s^{-1}$)	White muscle recruitment speed at 20 °C ($cm\,s^{-1}$)	Q_{10}	N
8	30·80±2·59	44·00±4·18	1·43±0·08	5
15	26·17±2·64	45·83±5·85	1·71±0·11	6
26	21·40±2·07	43·00±5·00	2·01±0·13	5

Taken from Rome, Loughna & Goldspink, 1984.

properties and also from an increase in the number of red fibres. These changes mean that as a result of low temperature acclimation the fish can derive much more aerobic (sustainable) power from its myotomal musculature and this compensates to some extent, for the power reduction due to the lower temperature. This latter type of acclimatory change has been shown to occur in several species of fish examined, including those that cannot change their myofibrillar properties such as the striped bass (Jones & Sidell, 1982).

From these previous studies and recent electrophoretic studies, it is clear that a considerable amount of 'protein remodelling' occurs as a response to temperature. The types of proteins that are produced clearly differ and we are particularly interested in pursuing this problem to find out which proteins are changed and how they are changed. In other words, we wish to determine whether the existing proteins are modified or whether new types of proteins are synthesized.

At the present time the latter seems the most likely as fish fed on protein-deficient diets are unable to acclimate by changing their myofibrillar structure. If new types of proteins are synthesized (e.g. isoenzymes of myosin, troponin), then this means that there are changes in gene expression that are stimulated by environmental temperature. This system may be, therefore, a very useful one for studying the mechanism of differential gene expression in vertebrate cells in general and hence it would be of wider interest than just temperature acclimation.

The muscle mass makes up a large percentage of the bulk of most animals (approximately 50% in mammals and up to 80% in fish) so clearly it is a great advantage if structure can be matched with function. This process of matching structure with function has occurred during evolution but it also, as demonstrated, occurs during the animal's life-time so that the size and characteristics of the muscle mass can be regulated according to the functional demands on the tissue at any given time.

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