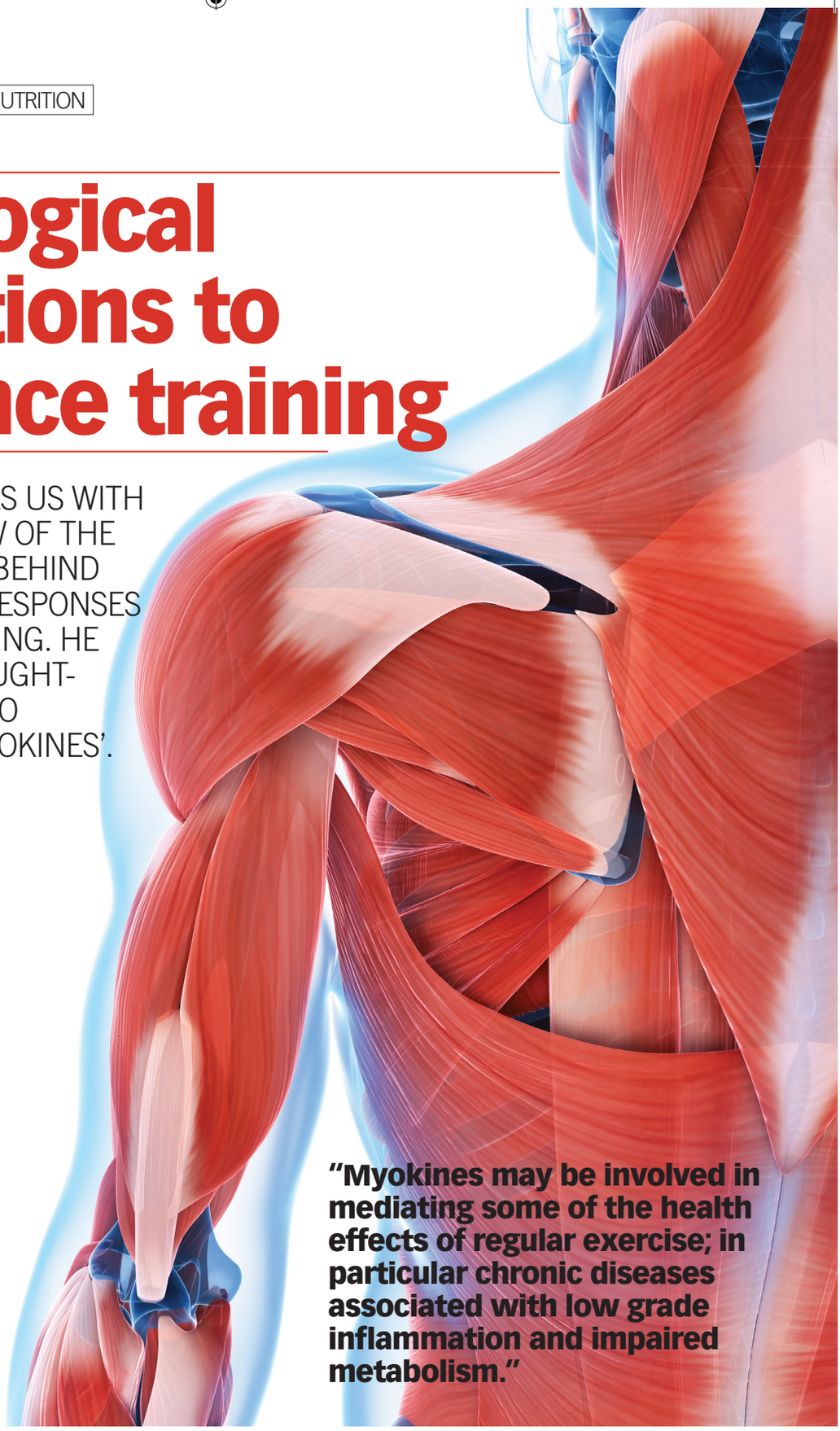
Physiological adaptations to resistance training

PAUL EHREN PROVIDES US WITH A FASCINATING REVIEW OF THE SCIENTIFIC THEORIES BEHIND THE PHYSIOLOGICAL RESPONSES TO RESISTANCE TRAINING. HE FINISHES WITH A THOUGHT-PROVOKING INSIGHT TO RECENT WORK ON 'MYOKINES'.



"Myokines may be involved in mediating some of the health effects of regular exercise; in particular chronic diseases associated with low grade inflammation and impaired metabolism."

This brief review will consider some of the neurological and physiological responses to resistance training (RT). Research in this area has been quite extensive, but still remains nowhere near complete, and of course continues to evolve. It also throws up a number of seemingly contradictory papers and some very interesting academic shenanigans, which I refer to later.

As both force production and hypertrophy are arguably the two most easily recognised responses to RT, I will start with these areas. Force may be defined as the ability to accelerate mass over a given distance. The ability to generate force through the production of muscular tension may, as we will see, be influenced by a number of neurological and physiological factors.

We may view hypertrophy as the increase in cross sectional area (CSA) of muscle fibres. This should not be confused with an actual increase in the number of muscle fibres (hyperplasia) which, to the best of my knowledge, has not been consistently proven as occurring in human studies as a result of RT. We may also fine-tune our definition of hypertrophy by looking at the difference between functional (myofibrillar) hypertrophy and non-functional (sarcoplasmic) hypertrophy.

With functional hypertrophy, we are considering an increase in the size and number of the contractile proteins contained within the myofibrils, leading to an increase in their number, and subsequently an increase in the number of sarcomeres in parallel. We would term this 'functional' because we should experience an increase in force production, but not appreciable muscle size.

Non-functional hypertrophy, however, is obtained through increased volume of the muscle fibres sarcoplasm without the same level of changes to the contractile proteins, providing increases to the muscle size (and weight), but without the concurrent increase in strength.

The ability to promote functional hypertrophy is vital to athletes when power to weight ratios and/or competitive weight categories are important. Non-functional hypertrophy would apply to athletes such as bodybuilders, rugby forwards, American football linemen, wrestlers and sumo wrestlers, where varying degrees of muscle 'bulk' is important.

In practice, pure functional or non-functional hypertrophy would be extremely difficult, if not impossible, to obtain, but manipulation of training parameters such as muscle actions, exercise order, exercise volume, exercise intensity, time under tension, rest periods and frequency, together with nutritional strategies (involving primarily the amounts and timings of macro nutrients), will influence the balance of functional and non-functional hypertrophy achieved. It is therefore extremely important that the coach or strength and conditioning practitioner understands the nature of these variables and how to put them into practice to achieve the desired results with the athletes under his/her care.

Neurological and physiological responses to resistance training

With RT, it's been consistently shown that neurological adaptions are the first to be initiated, and will pre-empt any changes to muscle morphology by some 8 to 12 weeks (1). Neurological adaptions will predominately involve the 'motor units', which are comprised of the motor neuron and the muscle cell(s) it innervates.

If we were to observe a RT novice performing, for example, a bench press, not only would force production be poor, but the movement pattern would be unsure and/or jerky. Improvement in the fluidity of movement as well as the ability to generate increased force production will depend predominately on two things; the number of motor units activated and the frequency in which these motor units fire. Motor units have been shown to be recruited based on the size principle, with the smaller slow twitch motor units being recruited at the onset of exercise and the larger fast twitch units utilised as the force continues to intensify.

Electromyographic (EMG) studies have shown that as RT experience continues, we develop the ability to improve both the number and type of fibres recruited, as well as improve the rate at which these fibres fire. Therefore, assuming our subject is shown correct form in the exercise being performed, initial increases in force production in the targeted muscle(s) will be achieved by way of continued neural stimulation.

Looking at the mechanical stressors of RT; when the intensity of the contractions is sufficient, damage to the sarcomere architecture may occur, leading to Z disc disruption and smearing (2,5). Both the concentric (positive) and eccentric (negative) phases of the repetition are important contributors to the hypertrophic response and need to be applied correctly to achieve the desired effect. The eccentric contractions, in particular, are linked to the phenomenon referred to as delayed onset muscle soreness (DOMS). However, there is still much to be understood about the exact nature and causes of DOMs and reviews have indicated that it should not necessarily be an indicator of muscle damage or a requirement of muscle hypertrophy.

Metabolic stress of RT has been shown to include an increased requirement for energy substrates, an increase in lactate, hydrogen ions, cytosolic calcium ions, decreased mitochondrial activity and increased phosphorylase actions (6,7,8). The muscle contractions which lead to this mechanical and metabolic stress will either directly or via the hormonal and immune system, stimulate protein synthesis and satellite cell activity, leading to increase in the number and size of contractile proteins within the muscle cell.

Protein synthesis has been shown to be initiated by the anabolic hormones testosterone, growth hormone and insulin-like growth factor, plus the inflammatory cytokines interlukin-6, transforming growth factor-β and tumour necrosis factor-α, which stimulate the cellular pathways; primarily the protein kinase mammalian target of rapamycin, leading to the cell nucleus triggering increased protein synthesis (3,9,10).

Textbook representation of hypertrophy

Let's begin to pull the various threads together and consider what may be considered the current 'textbook' view of the hypertrophic response to RT. I would like to concentrate on the long-term changes, as opposed to the single session changes, as these will arguably be of much greater relevance to performance, health and aesthetics.

Neurological changes are likely to see an increase in motor unit recruitment and firing rate, and EMG amplitude should demonstrate an increase during maximum voluntary contraction. Changes to skeletal muscle tissue should include an increase in force production, an increase in type I and type II fibre cross-sectional area and potentially a shift between the fast twitch sub types.

The possible changes to connective tissue, such as tendons and ligaments, includes additional synthesis of collagen. However, many apocryphal accounts lead to the conclusion that there is a substantial 'time lag' between the point of force production in skeletal muscle and the ability of the connective tissue to maintain integrity when subjected to the increased loads placed upon it. This is particularly the case when performance-enhancing substances such as anabolic steroids are employed.

Changes to the skeletal system have been studied at some length, concentrating to a large extent on post-menopausal women, in an effort to lessen the incidence of osteoporosis. The theory that research has attempted to prove is that the strain imposed by the application of RT will lead to positive bone remodelling. My own studies in this area appear to demonstrate that stronger individuals, men and women, do tend to have thicker and stronger bones, but the evidence is mixed. As ever, selection issues, population and the exact nature of the RT utilised will influence the results, but there does appear to be sufficient evidence that RT will have a positive effect on bone health.

As RT is primarily an anaerobic form of exercise, metabolic changes triggered should relate to either the phosphagen or glycolytic systems. Research has shown mixed results on substrate and enzyme activity, complicated by the fact that the increase in muscle cross sectional area has the effect of diluting these substances even though their absolute level have increased. My own experience, working with power athletes and bodybuilders, shows a consistent increase in both creatine kinase (CK) and lactate dehydrogenase (LDH), although these may well be influenced by acute spikes, caused by individual exercise sessions.

Another area which is causing me some interest at the moment is the influence of RT on the hepatic enzymes aspartate aminotransferase (AST), alanine aminotransferase (ALT) and gamma-glutamyltranspepidase (GGT). The conventional medical view is that increases in both AST and ALT are an indicator of hepatic stress. However, other research shows that high-intensity RT, leading to rhabdomyolysis (severe muscle breakdown), may promote changes in both these enzymes (11).

Chronic hormonal changes are subject to influence from a number of factors, such as the acute hormone spikes caused by RT and the hormonal effect of possible overtraining, which will affect adrenal function in particular. There does, however, appear to be a body of evidence showing that a chronic elevation in testosterone levels is experienced, as well as an up-regulation of androgen receptor sites in both skeletal muscle and neural tissues. These changes will depend on the type of RT undertaken: athletes taking part in weightlifting/powerlifting-style training, including low repetitions, heavy weights and long rest periods, will experience a different hormonal profile to those who may employ a bodybuilding-style workout with medium to high repetitions, much lower poundage's and shorter rest periods.

The conventional view, with regard to cardiovascular changes, is that RT will not have any major positive effect on peak VO_2 performance or oxidative enzyme activity and may even decrease the level of myoglobin and mitochondrial density. However, a correctly applied RT programme will improve the ability of an athlete to perform aerobic activities and will therefore have a positive, albeit non-direct, effect on cardiovascular performance.

Finally, body composition should also be heavily influenced by RT with an increase in fat free mass, resulting from a correctly constructed and applied RT and nutrition programme.

New research

I would now like to re-direct my gaze to some of the new research which had, until recently, brought about a whole new paradigm to the influence of RT on general health. I would ask you, however, to ensure that you read the entire article, as you will discover that this research comes with a very large 'be aware' notice attached to it. Let's start with a little background; the medical and exercise science communities have long sought an 'exercise factor' over and above the ability of exercise to balance energy intake, that may explain the differences in mortality and morbidity between sedentary and active individuals. This would include the increased incidences of the so-called sedentary conditions; type 2 diabetes, high blood pressure, obesity, certain cancers, cardiovascular disease, depression and dementia – it has been summed up by the phrase "diseasome of physical inactivity".

The basic hypothesis drawn up by research is that a cascade effect is produced, with "physical inactivity leading to the accumulation of visceral fat and consequently to the activation of a network of inflammatory pathways, which promote the development of insulin resistance, atherosclerosis, neurodegeneration and tumour growth, leading to the development of the diseasome of physical inactivity" (12).

The recent research has been led, to the best of my knowledge, by Bente Klarlund Pedersen, Professor of Integrative Medicine at the University of Copenhagen. Experiments identified hormone-like secretions produced by contracting skeletal muscle, referred to as 'myokines', which act in an endocrine, paracrine and autocrine fashion, affecting the original secreting cell, adjacent cells and those on a system-wide basis.

The first of these myokines indentified appears to have been Interleukin-6 (IL-6), which in itself becomes interesting because this substance is normally considered a pro-inflammatory cytokine produced as a result of macrophage activity. However, it was concluded that this was environment-dependent and when IL-6 was produced and released by muscle cells, the classical pro-inflammatory pathways were not activated and in these instances IL-6 acted in an anti-inflammatory fashion and therefore, inter alia, as a protective agent against the above mentioned diseasome of physical inactivity. Other myokines were subsequently identified, including, IL-15, IL-8, brain-derived neurotrophic factor and fibroblast growth factor-21.

The conclusion of the research papers in my possession is:

"The finding that muscles produce myokines, creates a paradigm shift and reveals new scientific, technological and scholarly horizons. We are convinced that the characterisation of the biological effects of known and unknown peptides, constituting the muscle secretome, will dominate the coming decade. Moreover, we suggest that myokines may be involved in mediating some of the health effects of regular exercise; in particular chronic diseases associated with low grade inflammation and impaired metabolism."(12)

However, and it is a big however, as part of my own investigations I normally do a background check on the lead authors of the research that I am relying upon. Unfortunately, various internet posts, including the *Times* Higher Education supplement, report that Professor Pedersen was: "found guilty of scientific dishonesty in relation to six articles of which she was senior author. The articles describe the release of proteins called myokines during exercise, which is purported to explain its health benefits."

I have absolutely no way of substantiating these claims on Professor Pedersen's integrity, and I include it here purely as a warning shot that, as ever, research should not be taken solely at face value. At the very least, it has opened up a direction of thinking that has many fascinating aspects. We all need to continue our own investigations and draw conclusions on what is an ever-changing and expanding subject. FSN

REFERENCES

1. Moritani T & deVries H (1979). Neural factors vs hypertrophy in the time course of muscle strength gain. *Am J Phys Med.* 58(3):115-130.
2. Nosaka K et al (2003). Muscle damage in resistance training – is muscle damage necessary for strength gain and muscle hypertrophy. *Int Journal Sport Health Science.* 1(1):1-8.
3. Spiering B et al (2008). Resistance exercise biology, manipulation of resistance exercise program variables determines the responses of cellular and molecular signalling pathways. *Sports Med.* 38(7):527-540.
4. Stone M et al (2007). Principles and Practice of Resistance Training. *Human Kinetics.*
5. Cheung K et al (2003). Delayed onset muscle soreness: treatment strategies and performance factors. *Sports Med.* 33(2):145-164.
6. Siff M (2003). Supertraining. Supertraining Institute.
7. Zatsiorsky V (1992). Intensity of strength training facts and theory, Russian and Eastern European approach. *Journal of Strength & Conditioning.* 14:46-57.
8. Zatsiorsky V & Kraemer W (2006). Science and Practice of Strength Training. 2nd Ed. *Human Kinetics.*
9. Rommel C et al (2001). Mediation of IGF-1 induced skeletal myotube hypertrophy by PI(3)K/Akt/mTOR and PI(3)K/Akt/GSK3 pathways. *Nat Cell Biol.* 3(11):1009-1013.
10. Stone M et al (2003). Power and maximum strength relationships during performance of dynamic and static weighted jumps. *J Strength Cond Res.* 17(1):140-147.
11. Dickerman R et al (1999). Anabolic steroid induced hepatotoxicity: is it overstated? *Clin J Sport Med.* 9(1):34-39.
12. Pedersen B (2011). Muscles and their myokines. *J Exp Biol.* 15;214(Pt 2):337-346.

About the author

Paul K Ehren has run his personal fitness practice in London/Essex for the last 15 years and is a founding director of Physical Frontiers, who specialise in the health and performance of strength athletes and martial artists. Paul remains a competitive athlete and as a Masters Bodybuilder has won one British title (UKBFF), placing 2nd twice, along with two South East titles (NAABA), the latest being this year, a Midlands title (UKBFF) and has represented GB in Europe (WAABA). He is currently in an off-season phase of training, which will hopefully culminate in the retention of his South East title in 2015. Paul may be contacted directly on PEhren@aol.com.