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EDITORIAL

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# The role of muscle activity and mental load in the development of pain and degenerative processes at the muscle cell level during computer work

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## Introduction

The prevention of muscle disorders in the operation of computer input devices, PROCID, is a European Concerted Action Project which held an international symposium in Denmark in November 1999. This issue of the *European Journal of Applied Physiology* contains 16 contributions to the symposium, some presenting new material, others being reviews. These contributions address a number of topics that were deemed to be of particular interest in view of the specific aims of the PROCID. These include:

1. Motor control patterns in humans including methodological aspects (five papers)
2. Motor unit activity during low force contraction and responses to pain and fatigue (four papers)
3. Muscle activity and the effects of psycho-physiological stress (three papers)
4. Mechanisms underlying muscle damage (four papers).

The present editorial is an attempt to put these into a general perspective, thus showing the tight links among them, as well as illustrating the general scope of the PROCID. The authors are very much obliged to the *European Journal of Applied Physiology* for judging the topic as being sufficiently important to deserve publication.

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## Background

Chronic pain in the musculoskeletal system is an adjunct to, or a result of, many different types of exposure during working life, including exposure to mechanical and mental requirements or loads. The majority of these pain syndromes commonly related to the occupation have focused on nerve entrapment and inflammatory reactions in tendinous tissues (Hagberg et al. 1995), where the underlying mechanisms causing pain are comparatively well understood. However, with respect to muscle pain this is not the case, even though chronic muscle pain in recent epidemiological studies has been shown to be prevalent in many occupations. For instance, muscle pain in the shoulder has been found to have high prevalence in welders as well as in computer operators, which illustrates the multidimensional character of the problem (Herberts et al. 1981; Hünting et al. 1981). Pain localized in muscle may not always be based on events within the muscle itself, but may be caused by processes in adjacent tissues, for instance in tendons or ligaments. Sometimes muscle pain may originate in other somatic structures, giving rise to so-called referred pain. However, in the present context we focus on pain that originates within the primary muscle itself in relation to muscle activity and mental load.

Muscle activity is required for performing physical activity at work, during sports, leisure time, etc. and is generally considered to be health promoting. This is because mechanical loading has been said to be essential to ensure the viability of the musculoskeletal tissues (Hargens 1986). Nonetheless we also consider muscle activity to play a significant role in the development of musculoskeletal disorders. This is plausible for tasks with high force requirements, possibly causing mechanical overloading of the tissues, and thereby obviously being causally related to disorders such as sports injuries and occupational accidents. Low force requirements do not constitute equally plausible risk factors leading to muscle disorders; however, a more recent *Cinderella*

hypothesis has proposed that single muscle fibres may be intensely active at force levels considered to be low for the muscle as a whole (Hägg 1991).

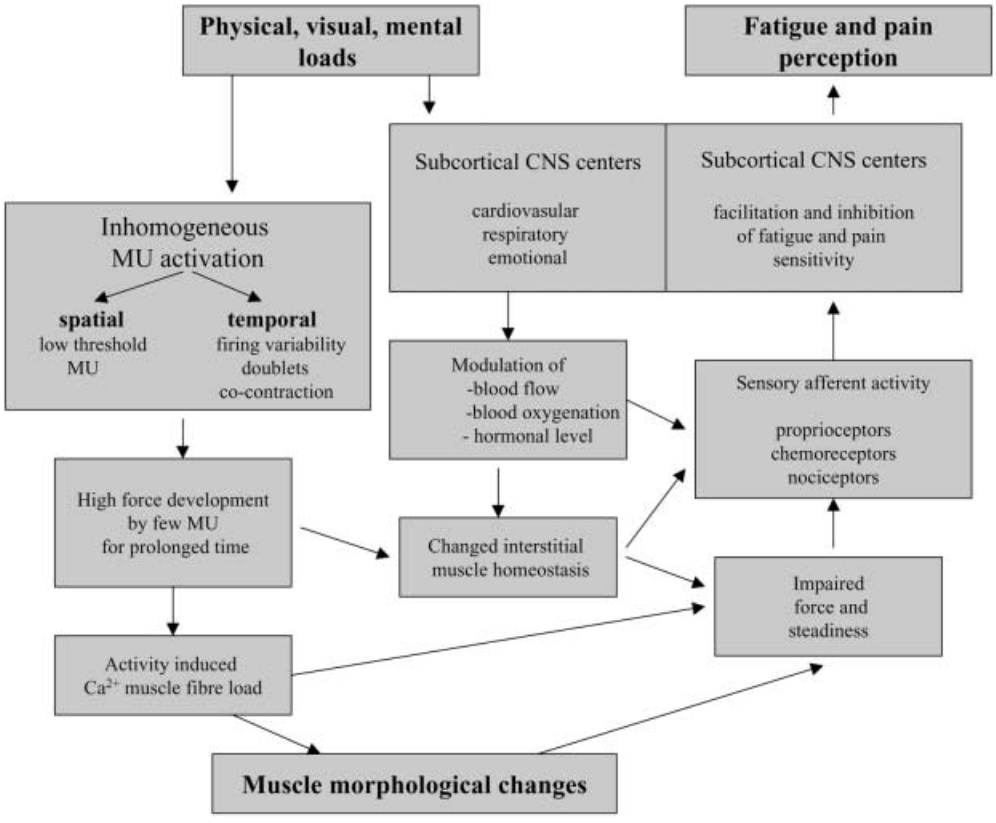
Mental activity is associated with activation of various physiological systems, including the muscles. Such bodily reactions are important for successfully coping with various environmental demands and for the protection and restoring of the body and involve, for example, a decrease in the blood supply to the inner organs and an increase to the muscles and the brain. This response provides resources for muscle effort and for efficient mental performance, but is also assumed to create a link between mental demands and somatic illness. As with heavy physical demands, intense stressors were, according to earlier stress models, assumed to be the major cause of stress-related illness. However, according to more recent models of stress and health, over- as well as under-activity of the allostatic systems has been assumed to contribute to health problems (McEwen 1998). A dynamic stress response, with rapid activation of the allostatic systems in response to a stressor followed by rapid deactivation when the stressor ends, is considered an efficient and healthy way of coping with environmental stress.

Computer work is characterized by low physical and mental exposure levels but none the less extensive health problems have been documented (Bammer 1987; Jensen et al. 1998; Punnett and Bergqvist 1999). Since we can envisage an increasing use of this technology in our working life and leisure time there is an urgent need to understand the mechanisms underlying the development of these disorders. Such knowledge is a prerequisite to the introduction of lasting effective preventive strategies. The inter relationship between the different mechanisms is illustrated in Fig. 1.

## Muscle activation pattern

The prevailing hypothesis is that prolonged activity of single muscle fibres may cause degenerative changes in the muscles. In support of this hypothesis detailed knowledge is required on recruitment patterns of motor units (MU), which are the smallest functional units in the muscle to be activated. Delicate techniques have been required for recording such activity and ongoing development as well as evaluation of these methods are required (Disselhorst-Klug et al. 2000; Pilegaard et al. 2000). It has been shown that descending input from the motor cortex in the central nervous system (CNS) to the MU may be modified by afferent input from the peripheral tissues, for example during fatigue (Taylor et al. 1996), and that voluntary activity does not imply that reflexes are not involved (Chalmer and Bawa 1997). For this reason some papers in the present issue have addressed this area (Bawa et al. 2000; Taylor et al. 2000)

**Fig. 1** Diagram showing the relationships between the various mechanisms that may be involved in the development of occupational muscle disorders



including data on the possible modulation by individual factors such as age and state of training (Patten and Kamen 2000). During video display unit (VDU) operation, for instance, the eyes are continually focusing accurately on moving symbols on the screen and increased neck muscle activity has been shown to improve fast eye movements (Kunita and Fujiwara 1996). This may, during vision-demanding tasks, imply that neck muscle tension is elicited via reflexes. Also, mechanical activity by the hands requires a stabilizing activity of the muscles in the shoulder girdle, although the shoulder muscles themselves do not interface with the actual computer.

Further, mental loads may elicit non-postural and non-voluntary muscle activity. The influence of mental activity on muscle activity was demonstrated early on and a series of recent experiments have confirmed and extended these findings. They have shown that various kinds of psychological stress and cognitive demands may cause elevated muscle activity in the trapezius muscle, although with considerable differences between individuals in the magnitude of response (Lundberg et al. 1994; Svebak et al. 1993; Wærsted et al. 1996). The muscle activity induced by mental stress is relatively low compared to that caused by heavy physical demands. However, psychologically induced muscle tension may cause overactivity of single MU and it has been thought that individual differences in responsiveness may be of clinical relevance (Veiersted et al. 1993). This area has been further reviewed in this issue (Wærsted 2000), and also a number of original contributions regarding mechanical and mental loads and pain have been presented (Birch et al. 2000; Jensen et al. 2000; Kitahara et al. 2000; Palmerud et al. 2000; Rissén et al. 2000; Sandsjö et al. 2000; Schnoz et al. 2000).

Particular patterns of motor control may play important roles. In this context inhomogeneous activation of a muscle exerting low forces is a characteristic that may be identified as a risk factor. The inhomogeneity can be specified in two dimensions: the spatial and the temporal.

Spatial inhomogeneity refers to the activation of only few MU of the whole muscle or a lack of load sharing between all MU during low force development. Those MU activated may develop relatively high forces while other MU remain silent and at rest. It has been considered that a stereotype recruitment order of the MU according to the size principle then implies activation of always the same low threshold MU (Henneman and Olson 1965). During prolonged low level contraction a few MU may then become fatigued or exhausted and this has led to the Cinderella hypothesis being proposed (Hägg 1991).

Temporal inhomogeneity refers to firing patterns not being uniform throughout voluntary contractions. In humans most MU firing patterns have been studied during static contractions at constant force, due to the limitations of the techniques for recording from single MU. This has led to reports of certain constant firing rates relating to specific force levels. However, on cat muscle preparations elegant experiments have been conducted showing that the level of force development is not only dependent on the mean firing frequency but in particular on the inter-firing interval of the initial 2–3 firings (Zajac 1981). Very short inter-firing intervals of less that 20 ms are termed doublets or double discharges, and the occurrence of doublets early in the contraction will cause large initial increases in the rate of force development. Further, the force is maintained at a higher level even after the firing frequency subsequently has been reduced to 10–15 Hz. Probably it is the requirement for high velocity finger movements that elicits recruitment patterns with doublets, and which have been observed during double clicking of computer mouse buttons (Søgaard et al. 2000). Other temporal inhomogeneities are the synchronization and firing variability that have been found to increase during prolonged, sustained forces. It has been thought that such changes in firing pattern may induce larger force fluctuation or impaired steadiness, and may elicit co-contraction for stabilization in order to be able to perform a steady contraction in spite of fatigue (Semmler et al. 1999; Yao et al. 2000). It has been suggested that this strategy may be common particularly in older workers (Patten and Kamen 2000). In total these mechanisms may result in the activation of a larger number of MU and thus develop larger muscle loads which may further increase fatigue development.

## Muscle fatigue and pain

Muscle fatigue limiting endurance occurs as a result of prolonged muscle activation at high voluntary effort. Fatigue may be accompanied by pain during ischaemic work situations that occur at muscle activation levels impairing blood flow. It has been shown that intramuscle pressures exceeding 20–40 mmHg are likely to cause partial or total arrest of blood flow, and that in high pressure muscles this may correspond to only some 10%–20% of maximal voluntary effort (Järvholm et al. 1988). It has been suggested that recurrent occupational exposure to static work may, with time, cause damage to poorly vascularized areas of muscle and tendon systems through inflammatory processes and subsequent development of compartment syndromes in muscles surrounded by rigid structures (Jensen et al. 1992; Järvholm et al. 1989). Therefore, studies further elucidating relationships between various occupational postures and intramuscular pressures to enable the setting of acceptable limits are highly relevant and welcome in this context (Palmerud et al. 2000).

The theoretical discussion with respect to the relationship between muscle pain and muscle tension is extremely complex, and no attempt will be made here to summarize it. Let it just be said that on the one hand there is the view that tonic muscle hyperactivity is the very cause of, or is at least contributing to, pain through reflex action, building a vicious circle. On the other hand

there is the view that pain, through inhibition, reduces the ability to activate a muscle, thereby forming a protective adaptation mechanism.

It is generally understood that muscle pain originates in the nociceptive free nerve endings located in the muscle interstitium. Nociceptors respond to potentially tissue-damaging stimuli and can be looked upon as pain sensors. They are sensitive to chemical or mechanical inputs, but activation is essentially non specific and may be triggered through release of different metabolic products related to muscle contraction, i.e. potassium, hydrogen, and lactate ions, and arachidonic acid. Changes in the chemical environment in a muscle in sustained contraction may cause a lowering of the nociceptor activation threshold. The pain sensation is mediated by groups III and IV sensory pathways, i.e. by thin myelinated and unmyelinated muscle afferent nerves. They connect in the dorsal horn to pathways leading to the CNS, but they may also connect to motorneurons.

Muscle pain of the myalgia type is particularly prevalent in occupations characterized by monotonous or repetitive work, not necessarily involving a high muscle activity but rather an ongoing psychological stress. In myalgia of the trapezius muscle the descending part of the muscle is tender, often with trigger points distributed over the muscle surface. It has been hypothesized that this pain syndrome has a background in reflex mediated muscle stiffness (Johansson and Sojka 1991). As a result of muscle activation, groups III and IV muscle afferent nerves are activated. It has been suggested that they have excitatory effects on $\gamma$-motorneurons, eliciting an increased firing of the muscle spindle afferent nerves or increasing the muscle spindle sensitivity to stretch. This will in turn raise the activation level of the $\alpha$-motorneurons innervating the muscle. Activation is thus evoked by sensitization of muscle spindles via the $\gamma$-loop. Provided there is a positive gain in this feedback system, a vicious circle may be set up, and muscle pain may spread, This may develop not only in the primary affected muscle, but even in neighboring muscles through heteronymous activation of the $\gamma$-motorneurons.

This hypothesis aligns well with some of the neurophysiological findings made by other researchers. The $\gamma$ muscle spindle system has a key role in regulation of muscle stiffness. It has been noted that muscles known to be rich in spindles include muscles of the shoulder and neck where muscle pain is common (Boyd 1985; Eriksson and Thornell 2000). In myofascial trigger points of the upper trapezius muscle, it has been demonstrated that there is an increase in spontaneous activity compared to adjacent areas of the same muscle (Hubbard and Berkoff 1993). This supports the hypothesis that trigger points are areas where there are sympathetically activated contractions of intrafusal fibres. On the other hand, Lund et al. (1991), in a critical review, concluded that there is evidence that pain does not cause muscles to become tonically hyperactive. They proposed an alternative neurophysiological model built on motor programming of excitatory and inhibitory interneurons supplied by high-threshold afferent fibres, and focusing on the pain inhibition mechanism. This model proposes that pain will cause a reduction in muscle activity; however, in conditions where a certain amount of muscle force must be exerted to accomplish the task it has been thought that this mechanism is unlikely to play a role (Birch et al. 2000).

## Morphological changes

Irrespective of the pain mechanisms, prolonged muscle activity resulting in muscle fatigue or pain has been proposed as a possible precursor to muscle disorders. Consequently, prevention of the development of perceived fatigue has been considered a preventive strategy and the general trend in the work place has been to try to decrease the requirement for the exertion of muscle force. This may be effective during high intensity activities; however, when reducing low to very low force development, it has been found that the sensory feed back mediating fatigue may become subliminal (Sjøgaard 1990). In line with this, the introduction of micropauses in sustained, moderate to high level muscle activation may, through hyperaemia, allow for the quick washout of metabolites and restitution of the composition of the interstitial fluid. This may reduce or eliminate the fatigue experienced or the ischaemic pain. Controversially, it has been hypothesized that such a pain relieving strategy may have the adverse effect of *fooling* the physiological protection mechanisms, and that introduction of micro-pauses may in fact allow the occurrence of degenerative processes causing damage to the muscle cells (Byström et al. 1991).

In short, homeostasis in the interstitium, where the sensory afferent nerves are located, may be largely maintained at the resting level during contractions where a relatively high blood flow is maintained compared to the small number of active MU releasing minimal amounts of metabolites and electrolytes. Thus the sensory free nerve endings mediating fatigue or pain perception are not being excited. Lack of such sensory feedback of muscle fatigue implies that we do not know that some muscle fibres are being exhausted and thus we will not voluntarily prevent single MU being activated to an extent that may cause serious changes in the intracellular compartment. No sensory nerve fibres are located in this compartment and the state of changes here cannot be conveyed to the CNS. For more details see (Sjøgaard and Søgaard 1998).

The activation of muscle fibres for a prolonged time will compromise intracellular homeostasis even if this cannot be sensed. Morphological changes have been identified in human muscle in occupational myalgia and a review on the state of the art has been included in this issue (Hägg 2000). However, the mechanisms or *intracellular risk factors* that elicit these abnormalities are poorly understood. The depletion of substrates or the

accumulation of metabolites are unlikely alone to account for degenerative tissue changes. Rather, electrolyte disturbances may be involved and, among these, the modulation of $Ca^{2+}$ transients has been a candidate for several years (Gissel and Clausen 1999; Jackson et al. 1984; Westerblad et al. 1991). The toxic effect of $Ca^{2+}$ may cause breakdown of intracellular and surface membranes causing massive muscle disorders. These processes are difficult to study in human muscle in vivo and knowledge from animal experiments has been useful in shedding some light on this area (Gissel 2000; Westerblad and Allen 2000). Profound differences in activation patterns during voluntary contraction and electrical stimulation have been found, although repeated bursts of high firing rates may be elicited also voluntarily, for example, during double clicking of computer mouse (Søgaard et al. 2000). Thus, the basic mechanisms for the breakdown of morphological structures at the level of a single muscle fibre need to be elucidated by in vitro experiments and therefore have been included in this issue.

In vivo, a large number of modifying factors, such as cardio-respiratory and hormone responses that normally relate to physical activity may have protective or aggravating effects on the breakdown of muscle cells. It is of note that during low level physical activity the effect of psychophysiological responses to stress may be particularly important. It has been said that stress can be induced by an imbalance between external demands and the individual's resources to meet those demands (Frankenhaeuser et al. 1989), by an imbalance between effort and rewards (Siegrist 1996), or by a combination of high demands and low control (Karasek 1979). Two major systems responding to stress, the sympathetic-adrenal-medullary (SAM) and the hypothalamic-pituitary-adrenocortical (HPA) systems, have been of particular interest from a health perspective (Henry 1992). Activation of the SAM system, with elevated blood pressure and heart rate, increased secretion of adrenaline and noradrenaline and release of energy (blood lipids, glucose) into the blood stream, can be described as an *active defence mechanism* preparing the body for battle (*fight-or-flight*). The best-known health problems associated with over-activation of this system are cardiovascular disorders, such as hypertension, myocardial infarction and stroke. However, in situations where the muscles are not active to an extent that would call for the physiological responses, even this tissue may be at a greater risk of developing abnormalities. Activation of the HPA axis, with elevated secretion of cortisol from the adrenal cortex, represents a *defeat reaction* and loss of control. Long-term overactivity of this system has been linked to increased risk of diabetes (insulin insensitivity), cardiovascular illness (elevated blood lipids), infections (impaired immune function) and cognitive deficiency (degeneration of hippocampus). The catabolic effect of, for example, cortisol, of course may also affect muscle tissue in ways causing deterioration. Lack of a response in one system due to, for example, exhaustion, may cause a compensatory overactivation of other systems. In contrast to the catabolic effects of the stress hormones, anabolic activity through steroid sex and growth hormones may help to repair damaged muscle fibres and thus reduce the risk of inflammations and pain development.

## Occupational muscle disorders

Muscle disorders include the many conditions affecting muscle tissue, resulting in dysfunction, pain or discomfort as well as clinical diseases with objective morphological abnormalities. There is a strong and growing body of evidence associating muscle disorders with workplace exposure to mechanical and mental loads. It has been reported that physically monotonous or repetitive work is associated with increased risk of shoulder and neck pain (Bernard 1997), but recent studies also report an association between psychosocial factors at the work place and muscle disorders. Conditions typical of many low status jobs have been reported often to characterize jobs associated with a high risk of muscle problems (Bammer 1990; Bongers et al. 1993; Lundberg et al. 1999). The high prevalence of musculoskeletal disorders in psychologically stressful but light physical work, such as assembly work and computerized data entry, has indicated that mental stress plays an important role (Moon and Sauter 1996; Schleifer and Ley 1996). Repetitive and monotonous blue collar jobs yielding a high prevalence of neck, shoulder and back pain problems have been associated with elevated levels of psychophysiological stress and a slower physiological deactivation after work, compared with more flexible jobs (Lundberg and Johansson 2000). Thus, psychosocial and psychological factors may prevent the individual from shutting off their physiological activation and returning to baseline during breaks at work and after their work shift. This will reduce restitution and contribute to sustained muscle activity.

Differences in individual susceptibility imply that some groups in the population are at higher risk than others. Women have been found to be at particular risk (Lundberg 1996), possibly due to the fact that they, more often than men, are involved in repetitive work and suffer the extra burden of unpaid work at home (Punnett and Herbert 2000). It has been suggested that this may be relevant to the greater health problems of women in general and for musculoskeletal disorders in particular (Melin and Lundberg 1997). Another group more prone to develop disorders is the elderly. It is of note in this context that the average age of the work force will increase in the coming years. The elderly are physically perfectly qualified to perform computer work. However, for example, the characteristics of their vision have often changed and they frequently use multi-focal glasses, narrowing their range of focus and thus affecting head postures. Also it has been shown that their basic motor control may have changed (Laidlaw et al. 2000; Patten and Kamen 2000; Spiegel et al. 1996).

## Conclusions

Any factor that provokes maintained muscle activity may increase the risk for contracting work-related muscle pain. Thus, it is relevant to explore to what extent mental and physical aspects of work tasks contribute to the maintained muscle activity, in sedentary work with low physical demands as well as in physically more demanding jobs. Workers carrying out repetitive work who lack influence and control seem to be exposed to higher levels of chronic stress and, consequently, to experience more lasting muscle tension than workers in more flexible and stimulating jobs.

In the modern work environment, with its emphasis on time pressure, competitiveness, lean production, and downsizing, it is possible that the lack of relaxation is an even more important health risk than is the absolute level of muscle contraction or the frequency of muscle activation. This is because of the way lack of muscle relaxation may prevent rest and also the recovery of the allostatic systems. As mental stress is often more lasting that physical activity, psychosocial factors at work may be of particular importance in keeping low threshold MU active to an extent causing homeostatic disturbances, morphological damage and pain perception. More knowledge and understanding of these mechanisms may allow us to design computer soft- and hardware that serves humans so that humans do not have to suffer when serving computers.

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