EXPOSURE ASSESSMENT AND MECHANISMS OF PATHOGENESIS IN WORK-RELATED MUSCULOSKELETAL DISORDERS: SIGNIFICANT ASPECTS IN THE DOCUMENTATION OF RISK FACTORS.


ABSTRACT

Epidemiological studies have demonstrated relationships between exposures at the work place and various disorders among the workers. However, only if there is a causal relationship the exposure is a risk factor, and its elimination will prevent the development of disorders. Causal relationships are documented by studying the mechanisms of pathogenesis. Mechanical as well as metabolical mechanisms may be involved in the development of musculoskeletal disorders and mental as well physical loads may play a role. A taxonomy for the classification of physical loads in various categories applicable for identifying risk factors in the work place will be discussed.

INTRODUCTION

The awareness of musculoskeletal disorders being associated with certain jobs dates back to the 18th century in the literature (71). Today numerous epidemiological studies document such relationships. In total these disorders account for the major fraction of reported occupational disorders (fig 1) not only in the Nordic countries (18,36,49,51), but worldwide (30,58,94). Still companies are reluctant to introduce significant changes in the work place and society hesitates to accept occupational musculoskeletal diseases for workers compensation claims. The expenses for society are enormous regarding treatment of and rehabilitation from these diseases, for lost production etc. and ample resources will be saved by investing more into prevention (93). Such preventive strategies must be based on sound scientific knowledge regarding the connection between exposure and effect.

EXPOSURE-RESPONSE-EFFECT MODEL

Risk factors must be identified in the working environment as part of the exposure comprising factors external to the individual. The documentation may be systematized according to the model in fig 2. This simplified model encompasses numerous other models proposed (3,4,49,73,98). Important motives for the model are to clearly distinguish between independent and dependent variables, as well as trying to define the framework for pathogenetic mechanisms. During the last decades many studies have documented relationships between certain jobs and corresponding patterns of musculoskeletal disorders, for references see (4,49,98) and table 2. However, in order to ensure high cost-effectiveness in the prevention we must more specifically identify those exposures which are causally related to musculoskeletal disorders. Such disorders often develop over long periods of time, which makes the precise identification of risk factors so difficult. Therefore, we must include basic knowledge regarding the acute response during the occupational exposure (7). An extensive literature exists regarding such acute physiological responses and the long term effect these may induce on the musculoskeletal system.

EXPOSURE

Exposure falls into two main categories: physical and mental. The physical exposure is in the present context the mechanical load on the musculoskeletal system and can be divided into those external loads that require movements (e.g. work is performed as defined in physics), and those that require tension only. The former may be positive or negative work and is performed as concentric and eccentric.
contractions, respectively, both of which are dynamic contractions. A little confusing is the fact that in some cases dynamic contractions will in a strictly physical sense result in zero work performed on objects external to the body. This is the case when moving objects on a level, i.e. without changes in potential energy, but will still be considered as dynamic work performed by the muscles. The mechanical load which requires tension only is usually called static "work" and is often associated with postural loads. Such loads may be sustained for prolonged periods of time or may be performed repetitively. Based on this the physical exposure can be described systematically and exhaustively as 4 different modes: dynamic concentric, dynamic eccentric, static repetitive, and static sustained. These exposures can be identified in the work place and quantified in classical physical terms as weight, acceleration, deceleration, distance, frequency, and duration (table 1). Quantification of exposure should consider three factors: 1) the exposure level, which is the work or the force performed in each contraction, 2) the frequency or repetitiveness of the contractions, which in combination with the exposure level quantifies the mean work load, i.e. power or mean force, and 3) duration, which in combination with the mean work load quantifies the total amount of work performed or the accumulated dose.

In this context moving the body or parts of the body are considered as external loads. Postural loads include holding objects as well as the body or parts of the body in position. Conceptually the external mechanical load associated with fixation of joints, is the most difficult to categorize. However, fixation of joints required for performing movements of other joints or holding certain parts of the body in position for instance required for precise vision may as well be defined as external loads. This requirement will involve muscles that are also activated to maintain e.g. the shoulder girdle in a fixed position needed for precise hand movements. The importance of this classification lies in the distinction between mechanical and mental loads.

The mental exposure comprises all external factors and stimuli that are subject to mental processing before a physiological response outside the central nervous system is evident. It is extremely difficult to classify all these factors, but a distinction can be made between informational loads and emotional loads. The relevance of specifying mental loads lies in the connection between the stimuli and mainly the activation of motoneurons. This means that under certain circumstances the central nervous system activates motoneurons so that muscle tension develops which serves no mechanical purpose and usually is static co-contraction. Mental exposures also activate stress hormones such as catecholamines and cortisol, which may impede renewal of the cells due to their catabolic influence (48,86). Quantification of mental exposure includes in accordance with the physical exposure duration and frequency. The focus of the present paper is however on physical exposure.

Various methods are available for measuring physical or, more precisely, mechanical exposures: force transducers, goniometers, accelerometers, cinematography using film or video techniques etc. Biomechanical modelling then allows calculation of the forces acting on the various body segments. Including detailed anthropometric data gives estimates on actual muscle forces needed to perform the work. The development of force in the muscle is connected to a series of events comprising electrical activation and stimulation of energy turnover. Therefore, the electromyographic response as well as oxygen uptake are measures of acute response. However, these variables can only be used to quantify exposure or external load through calibration procedures. In this context electromyographic recordings may confirm estimates from biomechanical modelling and may additionally give important information on and quantification of mental load. In general biomechanical modelling may be considered as the main link between exposure and acute response.

RESPONSE

Response includes in the present model the acute or immediate physiological changes that occur within the body as a result of the above mentioned physical or mental exposures. Essential for the musculoskeletal system is the development of force. Hence, the primary response to all exposures listed above is the development of active force in the muscles. Biomechanically almost all forms of external mechanical loads will cause counterforces involving muscle activation. In addition to this co-contraction of antagonistic muscles occurs frequently for stabilization of the joints. The active forces developed by
the muscles act on the tendons, connective tissues, joints, and skeleton and enhance or magnify the external forces acting on these structures.

The development of force in the muscles is connected to a series of cascading events: electrical activation of membranes, \( \text{Ca}^{2+} \) release into the cytosol, actin-myosin coupling, ATP breakdown, energy turnover in the mitochondria etc. Secondary to muscle activation a series of responses occur involving a multitude of organs. The respiratory and circulatory responses are important, and a mechanical demand on the musculoskeletal system has secondary consequences for the heart which may be decisive for setting limits rather than the responses of muscle or connective tissues. On the other hand reduced perfusion with increased tension may occur both in muscle and in tendons, and cause specific metabolic changes which may associate with development of disorders.

A major reason for introducing the concept of acute response into the model (fig 2) is that the four different modes of work mentioned above: dynamic concentric and eccentric as well as static repetitive and sustained are known to induce very different physiological responses which are described in detail in the literature (9). Further, an extensive literature exists regarding the effect of such physiological responses over time. Studies documenting relationships between occupational exposures and their effect in terms of musculoskeletal disorders do not identify the mechanisms which cause the disorders to develop. Such knowledge is important on the one hand to establish a causal relationship and on the other hand to identify which specific kind of exposure must be considered a risk factor. Causal relationships between acute physiological responses and musculoskeletal disorders as the effect are to be documented as mechanisms of pathogenesis.

**EFFECT**

Effects are basically either an adaptation or an injury. Adaptation is usually considered as improved capacity or tolerance, i.e. a training effect and injury here includes disorders and pain. The pathogenic mechanisms responsible for the development of pain and disorders depend on the mode of muscle activity. The tissues affected are besides the muscles also the connective tissue including tendons and their sheath, joints, disc, and bone. Perception of pain from the musculoskeletal system is mediated to the central nervous system by sensory afferent nerves. Specific pain receptors can not be distinguished morphologically in these tissues, but some free nerve endings have been demonstrated to respond specifically to noxious stimuli. Free nerve endings connect to neurons which fall within the group III and IV diameter ranges of sensory afferents and have been identified to respond to several types of mechanical as well as a multitude of chemical stimuli, some of which are known to be noxious. The axon terminals of the endings lie in the interstitial space in association with the skeletal muscle fibers, the intrafusal muscle fibers, the capsules of muscle spindles and tendon organs, tendon tissue at the musculo-tendinous junction, the adventitia of arterioles and venules, fat cells and connective tissue (181,89). In muscle interstitial changes within physiological ranges of chemical variables such as pH, inorganic phosphate, potassium, bradykinin etc. have been shown to increase firing frequency in most free nerve endings (61,64). Interestingly, nociceptors and other free nerve endings may become sensitized which means that a given dose algesic substances, which may be endogenous, elicits a stronger excitation, or that they develop a reduced threshold to normal mechanical stimuli such as pressure, stretch, and shear (62,63). This may be an important mechanism in causing allodynia, i.e. pain caused by normally innocuous stimuli.

The lack of objective signs of damage in the peripheral musculoskeletal tissues has been reported regularly and has caused several investigators to place the pain syndromes in the central nervous system. However, either the biochemical damage has so far remained elusive, or chronic states of pain can persist after the initial injury has healed. There is every reason to locate the primary or initial cause of the pain in the peripheral tissues. Today's knowledge is extensive regarding morphological and biochemical changes in muscle, nerve, and connective tissues affecting the functional capacity of these tissues. In the following examples will be given regarding work mode and associated possible pathogenic mechanisms.
Dynamic eccentric

Typical examples of this type of activity in the workplace are within manual material handling when loads are to be moved from high to low level, walking or running down hill or down stairs etc. Of note is that eccentric contractions are often performed also in response to unexpected loading. For instance when carrying patients, who may suddenly tend to fall, as a reflex we try to prevent this by decelerating the falling person. Other examples are slip and fall accidents or near-accidents. Eccentric contractions are then performed and they seem to be much more frequent in daily life activities than we are normally aware of (31). During this type of activity the highest mechanical forces can be developed by the muscles. Mechanical loads may also be imposed by force external to the body which may even exceed maximal muscle strength.

The obvious occupational musculoskeletal injuries may be due to mechanical insufficiencies of tissues resulting in fractures of bones or ruptures of tendons, ligaments, nerves, blood vessels or muscle fibers. When forces imposed on a tissue exceed tissue strength the cells are mechanically torn apart. Inability to continue work is then easily recognised and rest including medical treatment is usually recommended. However, a quite clear distinction should be made between such mechanical overload and normal task related loads.

Repeated exposure to high forces within the physiological range may induce microtraumas causing degenerative and inflammatory changes in tendons, muscles, disks, joints etc. eventually resulting in pathophysiological events when the reparative processes become inadequate. Probably, several types of microtrauma exist, for example demonstrated by morphological changes in muscle on an ultrastructural level as ruptures of myofibrils (Z-band streaming), autophagic vacuoles and abnormal mitochondria (26,27). Further, fiber swelling, fluid accumulation, and elevated intramuscular pressure has been reported (31). Such trauma are related to post-exercise muscle soreness, but the intracellular damage cannot be mediated as pain to the CNS, unless the fibre membrane becomes leaky causing changes in the interstitial space. Evidence for this is presented by increased concentrations of muscle enzymes in the blood (29,66). Further, rupture of elastin and collagen fibers may occur together with changes in the extracellular matrix structure of proteoglycans which encompass these fibers (28,82,83). Also, extravasation of blood from damaged blood vessels leads to liberation of bradykinin from plasma proteins which sensitizes muscle receptors (63). Such changes may trigger reparative processes but the mechanisms are still poorly understood (2). If sufficient recovery is allowed the strength of the tissue may increase but otherwise at some point the degenerative changes occur (fig 3).

High force also is a concern for the other tissues i.e. tendon, bones, and disc. Especially the latter together with the adjacent vertebra have been the focus in many studies associating low back pain with heavy lifting. It is in general recommended to avoid heavy loads and several lifting guides exist worldwide, the most extensive documentation being presented in the NIOSH document (67). In Denmark there is a legal for maximum acceptable loads to be lifted, which is 50 kg provided optimal lifting conditions exist.

Dynamic concentric

Typical examples of high intensity dynamic activity are seen in primary occupations such as agriculture, fishing, forestry, mining and construction work. During this mode of activity humans perform work according to classical physics. Physical work is equivalent to energy and in the human body a conversion occurs from chemically bound energy to mechanical work with an efficiency of maximally 25% as reported for dynamic concentric contractions like for instance during bicycling. During occupational tasks the efficiency usually is much lower for biomechanical reasons even if a relatively high element of dynamic activity is involved. The highest energy turnover can be elicited during this mode of activity. Energy turnover imposes a metabolic load especially on the muscles, which has been reviewed in detail with focus on muscle function (90).

Depletion of chemically bound energy or substrates (especially glycogen) and accumulation of metabolites (especially lactate which affects pH) have been demonstrated to relate to fatigue
development (37,90). The ultimate substrate in the conversion of chemical energy to mechanical energy is ATP, which is broken down in the myofibrils during the actin-myosin reaction. ATP is of significance for the detachment of actin and myosin and insufficiency of this process may cause rigor or contracture with massive pain. Little detailed information is available on the mechanisms of pain in contracture and this may be totally different from work-related muscle pain (24). In normal contractions the actin-myosin reaction is initiated by the release of Ca\(^{2+}\) from the sarcoplasmic reticulum into the cytosol, and has been the focus in a number of studies on muscle fatigue. However, during the last decade attention has been drawn also to the pathogenesis of Ca\(^{2+}\)-induced damage of muscle cells (41). The reuptake of Ca\(^{2+}\) into the sarcoplasmic reticulum is an ATP dependent process, which may be insufficient during prolonged activity since it accounts for up to 30% of the energy turnover during muscle activity. Further, energy crisis may result in an influx of Ca\(^{2+}\) from the extracellular space. Consequently the cytosolic free Ca\(^{2+}\) is likely to be increased above normal during prolonged activity. This has serious implications on the phospholipids including those in the muscle membrane. Ca\(^{2+}\) has a direct effect on phospholipase activity and in addition increases the susceptibility of the membrane lipids to free radicals. Both of these processes promote breakdown of the muscle membrane (20). Finally, prolonged increased cytosolic Ca\(^{2+}\) concentration induces a Ca\(^{2+}\) load on the mitochondria, and may eventually impair ATP formation.

The increased energy turnover in the muscles also activates the cardio-respiratory system, which provides the muscles with oxygen and other substrates via the blood flow. The circulatory adaptation is central, and a mechanical demand on the musculoskeletal system has secondary consequences for the heart which may be more decisive for setting limits than the response of the muscles and connective tissues. In fact previous attempts on setting limits for physical loads focus on increases in heart rate and oxygen uptake. On the one hand these variables may be regarded as indicators of the mechanical load and on the other hand they are direct measures of the load on the cardio-respiratory systems. Controversial data exist whether chronic exposure to dynamic muscular work in occupational settings is beneficial or deleterious to cardiovascular health. Dynamic muscular work improves the pumping capacity of the heart and the efficiency of the oxygen transport system as reported in numerous training studies. In line with this epidemiological studies have shown that persons chronically practicing dynamic exercise have a lowered risk of cardiovascular diseases as compared to sedentary persons (69). In contradiction others report high physical load at work to increase the risk of developing coronary heart disease (39,40) and to faster deteriorate the functional capacity of the workers (68). Taking into account the current knowledge acceptable limits for increases in heart rate and oxygen uptake during occupational work have been suggested not to exceed 1/3 of maximum capacity of the workers for an 8 hour working day (45,88).

Static repetitive

The occurrence of extensive static contractions in the work place has increased with increased industrialization i.e. they are common in secondary occupations. Repeated gripping and holding tasks are frequent among sewing-machine workers, meat cutters, chocolate packers and other assembly line workers, just to mention a few occupations in industry in which static repetitive contractions are common exposures. Typically the contraction period is in the order of seconds and the number of contractions often lies in the range of 10,000 to 20,000 per day. The force per contraction as well as the repetitiveness have been shown to be of significance for pathogenic developments (76).

With each muscle contraction tissue pressure in the muscle increases (75). This increase may well be in the same order of magnitude as the blood pressure whereby the perfusion of the muscle is impaired or even occluded. Following each contraction then high perfusion values will be attained for reoxygenation of the hypoxic tissue. Such variations in perfusion may cause short periods of high oxygen tension which can induce the formation of free radicals. As mentioned above free radicals are toxic substances, which increase lipid peroxidation. Studies of the free radical reaction during physical activity has indicated an association between peroxidation of membrane lipids and a number of changes in cell function such as increased membrane permeability, loss of mitochondrial respiratory control, decreased Ca\(^{2+}\) transport in the sarcoplasmic reticulum and formation of toxic metabolites (21,55,77).
The muscle force is transmitted to the skeleton via tendons. At the attachment to the bone or in the
tendon per se repetitive microruptures may occur which lead to the formation of fibrous, granular
tissues, and finally inflammations. These are diagnosed as epicondylitis or tendinitis (36,53,57,65). The
repeated contractions also may induce friction at the tendon especially at the wrist, where the tendons
are kept in place by narrow ligamentous compartments. Friction may cause inflammation in the tendon
sheath as well as in the tendon itself in addition to swelling of the tissues. This swelling will lead to
increased tissue pressure which in the carpal canal also effects the nervous tissue. Increased carpal
tunnel pressure causes an impairment of blood flow in the nerve, which can lead to impaired
conduction and sensory function (19,85). Microscopic studies of tissue in the carpal tunnel in wrist
specimens have revealed changes such as increased thickening of fibrocytes and fibrous connective
tissue in the radial and ulnar bursa and the median nerve (5). The specific diagnose is then carpal
tunnel syndrome, but similar pathogenic mechanisms are seen in other body regions.

Static sustained

Stabilization of the shoulder region is a prerequisite for performing precise manipulations with the
hands. Therefore, sustained static contractions often occur in combination with repetitive hand and arm
movements in industry as mentioned above. Constrained working postures exaggerate such static
loads. In addition static contractions in the neck region are elicited during high visual demands. Thus
not only in secondary but also tertiary occupations sustained static contractions are common. Typical
examples are seen among waiters, dentists, and a large number of other service jobs where for instance
VDU-operation is the dominating task.

Muscle forces can only be sustained for prolonged periods of time if they are relatively low.
Consequently, the increase in intramuscular pressure is far below the blood pressure and insufficient
blood perfusion often is not considered a problem. In numerous work place studies the static
contraction levels reported are in the order of 5-10% of maximum strength. In the shoulder muscles
this will cause tissue pressures of 40-60mmHg or more (42). Causal relationships between prolonged
moderately increased tissue pressure and pathogenic changes have been studied extensively in relation
to compartment syndromes (70). Pressures above 30mmHg maintained for 8 hours have been shown
to induce necrotic changes in the muscles even if no active contraction was performed and energy
demand therefore was minimal (32). One possible mechanism is that although initially blood flow is
sufficient during low level contractions this may not be the case when the contraction is maintained
for prolonged periods. Conditions with low flow and low perfusion pressure may provoke granulocyte
plugging in the capillaries which seriously effects the microcirculation (43,74).

Another aspect of importance during low force development is that although the muscle as a whole
may not be metabolically exhausted this may well be the case for single muscle fibers. Various motor
units may be alternating in activity pattern during a submaximal muscle contraction preventing fatigue
to develop in each of the involved fibers. However, performing highly skilled movements and accurate
manipulations it is likely that the very same motor units are being recruited continuously.
Additionally, contractions may be elicited due to reflexes causing an even more stereotype recruitment
pattern than during voluntary contractions. Mental load has been demonstrated to generate non-
postural muscle tension and the same holds true for visual demands (54,95). Also reflexes originating
in the contracting muscle itself from chemos as well as mechanoreceptors may play a role and recently
the gamma-loop has been proposed to play a role in developing a potentially vicious circle (44,62). The
muscle fibers being continuously activated have been termed cinderella fibers, since they are working
from early to late (33). A high energy turnover occurs in these fibers and most likely they receive the
least blood flow because pressure increases in their vicinity due to the mechanical contraction (80,100).
The pathogenic mechanisms described above for the muscle as a whole regarding accumulation of Ca²⁺
and free radicals may here be a concern on the single fibre level. Prolonged activity of specific motor
units throughout an 8 hour working day may cause insufficient time for full recovery of these motor
units due to a long lasting element of fatigue (23). This may cause necrosis and finally cell destruction
of these fibers. In line with this fibers with marked degenerative characteristics have been found more
frequently in muscle biopsies from patients with work-related chronic myalgia than in normal subjects

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(52). Interestingly, the degenerative fibers identified are slow twitch fibers which connect with low threshold motor nerves (35).

Adequate perfusion is essential not only for muscles and nerves but also for tendons. The major blood vessels supplying a tendon often runs through the corresponding muscle as it is the case for the supraspinatus muscle and tendon. Tendon circulation therefore is dependent on muscle tension in that it is inversely proportional to the tension and ceases at high tensions. Mechanical compression of tendons may further impede the circulation. For example during elevation of the arms, the rotator cuff tendons and the insertions on the greater tuberosity are compressed under the coracoclavicular arch. It is believed that this compression or impingement is sufficient to impair perfusion of the tendon and an inflammatory reaction may develop. This may be true in the tendon as well as in the bursa above the tendon and swelling of both structures may further increase the tissue pressure in this narrow space. Over the years zones of avascularity may develop, seriously predisposing for tendon disorders (56,72). Degeneration of tendon is caused by impairment of perfusion and nutrition, in addition to mechanical load. Inflammation forming debris in which chalk can deposit is the integral part of cell death and degeneration.

BODY REGIONS

Epidemiological studies report prevalence of disorders in the various body regions which are different among occupations with different exposures (30,51). Due to methodological differences various studies may be difficult to compare. However, the Nordic questionnaire has been used extensively in many occupations (50). The results from several studies are presented in table 2.

The one-year prevalence is highest in the neck and shoulder region in occupations with monotonous tasks inducing a high static load on the muscles in this region throughout the day. The reasons for this relationship are probably two fold: firstly, static continuous contractions in working life are the most extensive in this body region and secondly, the tissues in the neck and shoulder region are most susceptible to this kind of exposure due to the special anatomy. Examples are given above regarding the supraspinatus muscle and tendon. Similar arguments may be used regarding the high prevalence of elbow and wrist disorders in occupations with highly repetitive contractions of the forearm muscles. The carpal tunnel is here an example of how the special anatomy may cause certain tissues to be particularly vulnerable to repeated contractions. In many industrial tasks these exposures and disorders are often found in combination.

Regarding the low back the highest prevalence of disorders are reported among workers with a high level of physical activity including a high degree of manual material handling. Very high compression forces have been measured in the tissues of the low back which may imply a risk of injury. But interestingly also workers in constrained sitting working postures with only small loads applied to the hands and thereby also only small loads effecting the low back, report high prevalence of low back disorders. Multifactorial causes of these disorders are likely to exist which need further elucidation. Malnutrition of the intervertebral discs and back muscle fatigue are factors which may coincide with low back trouble in jobs with persistent low graded back muscle activity (38,47). In line with low back trouble, disorders in the legs are frequent among workers performing physically heavy work often with a high degree of eccentric contractions.

Based on this it seems relevant to identify exposure categories relative to the mode of work and the body regions. A distinction between the four major body regions may be sufficiently detailed for including documentation of causality from a large number of physiological studies, while in some cases especially when including clinical studies a more detailed approach may be needed.

INDIVIDUAL FACTORS

Genetically determined capabilities include gender, age, anthropometric measures, inherited diseases etc. and can not be changed by the individual. Similarly, different patterns of psychosomatic responses
exist and the tendency to develop muscle tension and hence muscle pain is probably quite variable and dependent on inherited capabilities. Muscle strength and anaerobic as well as aerobic power are trainable (even by the work task itself) but probably not beyond certain genetically determined limits. The individually different capacities determine the physiological responses to different mechanical and mental loads as well as transitions to long term effects (fig 2). Experience, knowledge, and motivation can of course further modify the response and cause any load to become unbearable or much too light, and coping is a key determinant of any type of physiological response.

A given work task consequently implies very different relative loads for the different workers and physiologically the relative load is decisive for the development of fatigue during the work. Fatigue is probably a key factor for longlasting injury that may be based on incomplete biochemical recovery. Various definitions of fatigue exist, the most common concepts being related to the mechanical response. However, biochemically speaking a muscle is fatiguing from the beginning of a contraction (10,22). The rate of fatigue development depends on the work mode and intensity. By manipulating the duration of work and rest cycles the endurance time can be extended dramatically and the amount of work performed in one work bout can then be increased significantly (11,16). The biochemical state of the muscle may be much more deteriorated when exhaustion is attained after prolonged fatiguing work compared to short-term fatiguing work. Interestingly, with submaximal repetitive static contractions fatigue may develop gradually due to an intrinsic muscle process causing a continuous reduction in maximal voluntary contraction force from the beginning of the muscle activity. Little or no change is seen in high energy phosphates (ATP and CrP) or lactate during this type of muscle contraction until suddenly the target force can no longer be maintained (91). At this point CrP is rapidly reduced, but still little lactate is produced. This kind of fatigue, developing slowly throughout the day is associated with increased oxygen consumption of the muscle, by as much as 100% (92). Hence although no work in a strict physical sense is performed, the muscle uses much more energy at the same force output. We do not know the exact mechanisms for these changes, but the data point to altered aerobic metabolism as a key pathogenic factor.

Of note is further that the time needed for attaining full recovery may be especially long following prolonged low intensity work (15). This may be due to not only slow recovery of substrate and electrolyte homeostasis but also to the slow restoration of the sarcoplasmic reticulum function and the element of low frequency fatigue (23,25). Following an 8 hour day of continuous activity for some muscles the subsequent 16 hours may not be sufficient time for recovery. Fatigue may play a significant role as an protective mechanism (59,78) and efforts of changing the work merely to postpone the perception of fatigue development can not be recommended.

Both central and peripheral fatigue reduce capacity and make the work task an increasingly larger load. This is illustrated in fig 3. During “work 1” fatigue develops and muscle function is reduced. Following the work muscle function will recover and the work may cause a training effect, but only if sufficiently long time for recovery is allowed. If the next work bout starts before full recovery is attained muscle function may be further decreased compared to the first working period. If work bouts are repeated with such a frequency that full recovery is never attained between successive bouts eventually injury, disorders, and pain may develop.

A strategy aimed merely at reducing mechanical and mental loads can not be correct, however. Physical activity is related to lower incidence of several diseases, and atrophy and degeneration occurs in the absence of such exposure (8). Hence, no physical exposure or inactivity may be considered as a risk factor. Pathogenic mechanisms in inactivity has not been included above, but ample documentation does exist and we may conclude that exposure to mechanical loads is necessary, otherwise reduced capacity and illness may ensue. Also deprivation of mental loads are unfortunate. Provided we can cope with the demands we thrive on challenges (48). The fact that even large external loads of mechanical or mental type may have beneficial effects on the individual, is of course a basic knowledge that must perpetuate the work on setting limits of ergonomic character. Indeed, one can turn the problem upside down and ask in what way regulations on physical and mental loads in the working environment can be used to promote health!

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This relates to several important conditions in working life: How long is acceptable to spend on the same job task? How long are pauses required? Need for micropauses and restitution? Job rotation? Hence any evaluation of exposure needs to incorporate these variables very accurately, which in combination constitute a major part of work organization.

DURATION

Only exposures with high forces which may cause acute mechanical injury of the tissues are per se dangerous. For all other exposures it is the duration of the exposure which must be considered as a potential risk factor. Consequently, we can minimize the risk factor by setting time limits regarding the maximal acceptable dose for a given task (97-99). Based on this it is suggested to quantify the work relative to the duration it can be sustained. Basic physiological knowledge exists regarding maximal endurance times for dynamic and static contractions, respectively (fig 4). Operationally three different intensities may be identified in the work place besides no activity or inactivity. The latter corresponds to "0", while "1" is low, "2" is medium, and "3" is high working intensity. An intensity may be considered as high if the work can only be sustained for a few minutes, medium if it can be sustained for around one hour, and low if it can be sustained for several hours. Referring to fig 4 an order of magnitude of these intensities may be given. The intention is not that maximum endurance time during a given work must be measured directly in the work place, a rough estimation on a three graded scale is probably possible and sufficiently accurate. Physiologically the three levels of intensities by and large correspond to the following % of the maximal individual capacities, which for static contraction is expressed in % of maximum voluntary contraction (MVC) and for dynamic work in % of maximum oxygen uptake ($\dot{V}_{O_2,\text{max}}$).

"intensity 1": a few % MVC or <30% $\dot{V}_{O_2,\text{max}}$
"intensity 2": <20% MVC or 30-80% $\dot{V}_{O_2,\text{max}}$
"intensity 3": >20% MVC or >80% $\dot{V}_{O_2,\text{max}}$

In the present taxonomy the term "intensity" is used for both static and dynamic contractions. The latter is measured in liters of oxygen per minute, which is a measure for intensity in a physical sense also, since it corresponds to energy per unit time or Watts. According to table 1 the equivalent for static contractions is force measured in N, which justifies to use "intensity" here as well. Alternatively, the term work "level" could be used. The absolute values suggested above may be further discussed. Large variations in endurance times exist depending on genetic capabilities, state of training, active muscle group etc. The curves in fig 4 represent rough mean values but may be sufficiently precise for a three graded scale of activity. In addition inactivity may occur for each body region.

Exposure assessment can then be quantified by combining work mode and intensity for the various body regions. Examples are given in fig 5 for a monotonous assembly task in welding (a) and a physically heavy task in manual material handling (b). Differences are easily recognized. The cube presented contains 4x4x4=64 categories or entities. Each job can be described in terms of a combination of several entities which may be considered as the exposure pattern of the job in question. Not all entities imply a risk for developing musculoskeletal disorders in the work place. However, for those which represent the highest risk high priority should be given to systematize the current knowledge in criteria documents. Based on the present taxonomy the general knowledge can then be used for the different specific tasks in the work place. A similar slightly simplified model has been proposed previously by the present authors (96). Optimization of job design may then be accomplished by adjusting the work load of the body region at risk (18) or by setting time limits for work and recovery ensuring an acceptable dose (59,99).

A maximal acceptable dose may be considered for each work task or on an accumulated basis for a day, a week, or a month. However as mentioned above the duration of recovery between successive work tasks is important. This emphasizes that not only the accumulated working dose but also the accumulated recovery must be considered. The ratio between work period and the following period allowed for recovery determines the mean work load. But besides this ratio also the absolute duration

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of work and recovery is of physiological importance for the rate of fatigue development. This shows that many degrees of freedom exist for modifying the exposure pattern.

CONCLUSIONS

In conclusion, we need to systematize our knowledge in a way that allows us to distinguish between exposures in the work place which are causally related to the development of disorders and those which are not. A lack of understanding the pathogenic mechanisms and a lack of guide lines to identify those exposures which carry a health risk are major reasons for lack of preventive strategies. The cube presented is a simplification which for several purposes is sufficiently detailed. The cube may be a useful tool for categorizing and assessing exposures in the work place and for systematizing future criteria documents needed for preventive strategies on musculoskeletal disorders.

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FIGURE TEXT

Figure 1.

Reported occupational diseases in Denmark in 1991, total number 14110. Source: The Danish Working Environmental Service.

Figure 2.

Exposure-response-effect model

Figure 3.

Muscle function in relation to exercise and recovery.

Figure 4.

Maximal endurance times for dynamic work and static contractions expressed in % of maximum oxygen uptake ($\dot{V}O_2^{max}$) and % of maximum voluntary contraction (MVC), respectively.

Figure 5.

Exposure pattern categorized in various entities for
a) a monotonous assembly task in e.g. welding and
b) a physically heavy task in e.g. manual material handling
<table>
<thead>
<tr>
<th>1) External load per contraction:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>For dynamic work</td>
<td>For static contraction</td>
</tr>
<tr>
<td>force * distance (Nm * J)</td>
<td>force * time (Ns) or peak force (N)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>2) Mean work load:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>For dynamic work</td>
<td>For static contraction</td>
</tr>
<tr>
<td>force * distance * frequency (J s ^* W)</td>
<td>force * time * frequency (N)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>3) Total amount of work:</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>For dynamic work</td>
<td>For static contraction</td>
</tr>
<tr>
<td>force * distance * frequency * duration (Nm * J)</td>
<td>force * time * frequency * duration (Ns)</td>
</tr>
</tbody>
</table>

Table 2. One year prevalence of musculoskeletal disorders in studies using the Nordic questionnaire for musculoskeletal troubles.

<table>
<thead>
<tr>
<th>Job</th>
<th>Country</th>
<th>Material</th>
<th>One year prevalence of musculoskeletal disorder</th>
<th>Ref</th>
</tr>
</thead>
<tbody>
<tr>
<td>VDU operators</td>
<td>S</td>
<td>125</td>
<td>49%</td>
<td>49%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>433</td>
<td>25%</td>
<td>22%</td>
</tr>
<tr>
<td>Bank assistance</td>
<td>S</td>
<td>31</td>
<td>20%</td>
<td>48%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>256</td>
<td>18%</td>
<td>18%</td>
</tr>
<tr>
<td>Secretaries</td>
<td>S</td>
<td>645</td>
<td>48%</td>
<td>48%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>125</td>
<td>18%</td>
<td>18%</td>
</tr>
<tr>
<td>Office workers</td>
<td>DK</td>
<td>69</td>
<td>50%</td>
<td>48%</td>
</tr>
<tr>
<td>Postal workers</td>
<td>DK</td>
<td>79</td>
<td>55%</td>
<td>60%</td>
</tr>
<tr>
<td>Sawmill workers</td>
<td>DK</td>
<td>303</td>
<td>68%</td>
<td>64%</td>
</tr>
<tr>
<td>Electronic industry</td>
<td>S</td>
<td>125</td>
<td>81%</td>
<td>82%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>433</td>
<td>34%</td>
<td>33%</td>
</tr>
<tr>
<td>Packers</td>
<td>S</td>
<td>125</td>
<td>50%</td>
<td>21%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>256</td>
<td>41%</td>
<td>67%</td>
</tr>
<tr>
<td>Meatcutters</td>
<td>S</td>
<td>256</td>
<td>72%</td>
<td>72%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>114</td>
<td>39%</td>
<td>62%</td>
</tr>
<tr>
<td>Train drivers</td>
<td>S</td>
<td>241</td>
<td>20%</td>
<td>18%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>677</td>
<td>34%</td>
<td>33%</td>
</tr>
<tr>
<td>Bakers</td>
<td>S</td>
<td>433</td>
<td>40%</td>
<td>37%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>125</td>
<td>34%</td>
<td>33%</td>
</tr>
<tr>
<td>Machine fitters</td>
<td>S</td>
<td>216</td>
<td>51%</td>
<td>59%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>220</td>
<td>29%</td>
<td>22%</td>
</tr>
<tr>
<td>Nurses</td>
<td>S</td>
<td>216</td>
<td>20%</td>
<td>29%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>114</td>
<td>11%</td>
<td>16%</td>
</tr>
<tr>
<td>Railway staff</td>
<td>S</td>
<td>660</td>
<td>16%</td>
<td>22%</td>
</tr>
<tr>
<td>Railway staff</td>
<td>D</td>
<td>508</td>
<td>24%</td>
<td>29%</td>
</tr>
<tr>
<td>Railway staff</td>
<td>SF</td>
<td>630</td>
<td>35%</td>
<td>47%</td>
</tr>
<tr>
<td>Wood and furniture industry</td>
<td>DK</td>
<td>22</td>
<td>20%</td>
<td>29%</td>
</tr>
<tr>
<td>Physical education teachers</td>
<td>DK</td>
<td>26</td>
<td>34%</td>
<td>25%</td>
</tr>
<tr>
<td></td>
<td>D</td>
<td>35</td>
<td>34%</td>
<td>25%</td>
</tr>
<tr>
<td>Airplane loading</td>
<td>DK, B, N</td>
<td>808</td>
<td>35%</td>
<td>31%</td>
</tr>
<tr>
<td>Welding operators</td>
<td>SF</td>
<td>5</td>
<td>94%</td>
<td>49%</td>
</tr>
<tr>
<td>Welding operators</td>
<td>D</td>
<td>320</td>
<td>34%</td>
<td>31%</td>
</tr>
<tr>
<td>Fisherman</td>
<td>S</td>
<td>1243</td>
<td>19%</td>
<td>30%</td>
</tr>
</tbody>
</table>
Cardiovascular diseases (2%)
Allergic disorders (4%)
Neurotoxic disorders (4%)
Lung diseases (6%)
Other diseases (10%)
Dermatological diseases (15%)
Musculoskeletal disorders (39%)
Noise-induced hearing loss (20%)

External

Internal

EXPOSURE

INDIVIDUAL FACTORS

ACUTE RESPONSE

LONG-TERM EFFECT