Theories of musculoskeletal injury causation

SHRAWAN KUMAR*

Department of Physical Therapy, University of Alberta, Edmonton, Alberta, Canada T6G 2G4

Keywords: Theories; Musculoskeletal injuries; Occupational injuries.

Based on the scientific evidence in published literature about precipitation of musculoskeletal injuries in the workplace, four theories have been proposed to explain these afflictions. Central to all theories is the presupposition that all occupational musculoskeletal injuries are biomechanical in nature. Disruption of mechanical order of a biological system is dependent on the individual components and their mechanical properties. These common denominators will be causally affected by the individual's genetic endowment, morphological characteristics and psychosocial makeup, and by the occupational biomechanical hazards. This phenomenon is explained by the Multivariate Interaction Theory. Differential Fatigue Theory accounts for unbalanced and asymmetric occupational activities creating differential fatigue and thereby a kinetic and kinematic imbalance resulting in injury precipitation. Cumulative Load Theory suggests a threshold range of load and repetition product beyond which injury precipitates, as all material substances have a finite life. Finally, Overexertion Theory claims that exertion exceeding the tolerance limit precipitates occupational musculoskeletal injury. It is also suggested that while these theories may explain the immediate mechanism of precipitation of injuries, they all operate simultaneously and interact to modulate injuries to varying degrees in different cases.

1. Introduction

With the industrial revolution, population explosion, technological innovations, and human need for merchandise and commodities, the world economic order has become largely dependent on the industrial sector. The WHO (1995) published that ~45% of the world's population and 58% of population >10 years of age constitute the global workforce. Of these, ~50% of workers work in physically hazardous conditions requiring unreasonably heavy work, exposing them to conditions hazardous to their health and working capacities. Many individuals spend one-third of their adult life in such hazardous work environments.

Humans have evolved over millions of years to be what they are today. The evolutionary pressures and consequent speciation resulted in an upright biped creature with dextrous upper limbs and a highly evolved brain. For a large duration of its existence, the species relied on hunting and gathering as its primary means of sustenance (Davis 1999). With the advancement of science, technology and industrialization, the physical occupational stresses have changed dramatically in

*e-mail: shrawan.kumar@ualberta.ca
an evolutionary ‘flash’. Thus, none of the body systems that one uses today was either designed or evolved for the purpose. As such, demand for force exertion, repetition of activities or assuming postures for prolonged periods places stress on human physical systems, which is inherently unnatural. This is further aggravated by many psychological stresses emanating from financial or interpersonal relation factors.

Thus, humans are neither anatomically adapted to withstand the physical stresses of today nor are they mentally suited to endure such psychological stresses. In addition, most human occupations have become complex, requiring significant organization, and thereby enhancing the chances of errors. Thus, as a function of a multivariate complex system (in many cases under multiple partial controllers) significant chances for unplanned things to happen emerge. These then result in various kinds of accidents with property damage and/or personal injuries. For a meaningful attempt to control such injuries, it is imperative to understand them.

According to WHO (1995), ~120 million occupational accidents with 200 000 fatalities are estimated to occur annually world-wide. Up to 157 million new cases of occupational diseases may be caused by various exposures at work. The number of people with access to remedial occupational health services is also very limited. Even in industrialized and developed countries, only 20–50% may have such a privilege. The corresponding figures in developing countries are 5–10% where injury control and prevention, as an organized effort, is considerably less and rare.

In fact, the occupational injury profile of most countries (which keep statistics) is staggering. The Bureau of Labor Statistics (1999) reported that in 1997 there were 1.8 million loss time injuries in the USA. Of these, 55% of the incidences were in the private sector and workers in the 25–44-year age group constituted 57% of all cases. Also, sprains and strains were the most common cause of loss time cases (43.6%), and most commonly these involved backs. Of sprain and strain injuries, 84% occurred in agriculture, 32% in forestry and fishing sectors and 52% in transportation and utilities. The operators, fabricators and labourers were most frequently affected by such affliction. Trunk (back) was the most affected body part for disabling work injuries in every industry division. Most other injuries occurred to upper and lower extremities. Overexertion while manoeuvring objects led all other disabling events or exposures, constituting up to one-third of cases in every major industry division. Statistics Canada (1995) in its latest report on work injuries reported that 48% of all injuries involved sprains and strains, 18% contusion, crushing and bruising, and 13% cuts, lacerations and punctures. In Canada also, the greatest number of injuries involved the back (27%), followed by the wrist, hand and fingers (22%), and ankle, foot and toes (10%). Overexertion was determined to be the most common cause (31%), followed by being struck (19%) and falls (16%), of all events that led to a work injury. For the proportion of injuries, fabricating, assembling and repairing occupations were highest, followed by service occupations and trades. Manufacturing industries accounted for 35% of all injuries, followed by service industries (19%), and trade (15%). The foregoing account of the nature and type of injuries, people injured, the part of their body affected, and the actions during which such injuries occurred leaves little doubt but to conclude that the physical activities are the most dominant occupational factors which mediate the occupational injuries.

A large majority of the Workers’ Compensation Board (WCB) compensated cases are regional musculoskeletal problems. Workers in different economic sectors generally have injuries characteristic of those sectors. People in forestry, construction and manufacturing have a higher proportion of back injuries. Those working in
office type jobs involving keyboarding have cumulative trauma disorders (also called repetitive strain injuries). Since it does not happen the other way round, i.e. the heavy physical workers developing cumulative trauma disorders and the office workers injuring their backs, it offers credence to the argument that the nature of the physical stress and the region enduring the load largely determine the affected area and probably the nature of injury. If, therefore, one could delineate the mechanisms of injuries and the quantitative details of the relevant variables, one might develop a more effective intervention. An effective intervention would result in a better control of injuries, which clearly has a significant pay-off. Thus, the long-term success in controlling these injuries depends on understanding their causation. A clear understanding and establishment of the mechanism of injury causation has been somewhat elusive. Therefore, an appropriate starting point is to examine the facts and to construct theories of musculoskeletal injury causation.

2. Nature of injury

An injury, by definition, means mechanical disruption of tissues resulting in pain. Thus, it is a traumatic event in which the integrity of the tissue in question is violated and its mechanical order perturbed. The latter leads to pain in addition to inflammation and other biochemical responses, hence the difficulty in deploying these structures in any activity including occupational activities. The term ‘injury’ is distinguished from that of ‘disorder’, which is frequently used in any malfunctioning of an organ or an organism. In contrast, to injury, a disorder can result without a mechanical perturbation of the tissues involved. Examples of disorders can be arthropathies, myopathies, neuropathies or several central nervous system problems resulting in improper functioning of the musculoskeletal system (e.g. multiple sclerosis, cystic fibrosis, ALS). Whereas an injury may result in a functional disorder that can be remedied by healing the injury, the injury in itself is not a disorder in a clinical sense.

Another difference between injury and disorder is that, while the onset of a disorder is gradual and mediated by some pathogen or prepathological progression, the onset of an injury is sudden and does not involve prepathogenesis. It may, however, involve mechanical degradation of the tissue due to repeated overuse. Subsequent to injury, inflammation and pathology of healing sets in. In the case of occupational musculoskeletal injuries, the organs or tissues are invariably exposed to factors that place mechanical stresses on the tissues. Most frequently such exposure is repetitive and/or prolonged and forceful, and hence it is considered a hazard or risk factor. The exposure to risk factors and subsequent injury precipitation may follow a dose–response relationship. Numerous studies have reported a strong association between exposure to risk factors and precipitation of injury.

A clear and indisputable causal or dose–response relationship between any of the risk factors and precipitation of injuries has not been demonstrated for any risk factor. Factors likely to confound this situation are the tissue’s ability to undergo adaptation to stress and to recover from the stress exposure. Repeated exposure over a prolonged period may impede complete recovery, causing residual strain. This is most likely to occur when the extent of adaptive changes is insufficient to offset the adverse biomechanical effects of stress exposure. An accumulation of residual strain over years may set the stage for injury even if the stress does not rise extraordinarily. The latter is due to progressive reduction in stress tolerance capacity due to steadily increasing residual strain. Kumar (1990) reported this concept of threshold level of cumulative exposure before injury precipitation.
The tissues that frequently get injured as a result of exposure to occupational biomechanical hazards are ligaments, tendons and muscles, and nerves are affected only secondarily. Other structures affected less frequently are cartilage and bones. All biological tissues are visco-elastic; hence, their mechanical properties are time- and strain rate-dependent. The tissue visco-elastic property determines the duration required for complete mechanical recovery. Any deformation or residual deformation alters the mechanical response characteristics of the tissue in question, most frequently lowering its stress-bearing capacity due to reduction in cross-sectional area and raising stress concentration and injury potential. In contrast, muscles are active organs and undergo voluntary contraction and relaxation rapidly to generate force. Therefore, the contractile elements of muscles are rarely affected by unrecovered deformation. It is the passive structures of muscles, i.e. the sarcolemma, or collagenous harness that can tear due to very rapid contraction or excessive force generation.

Workplace musculoskeletal injuries are broadly divisible under two categories: (1) idiopathic and (2) traumatic. The idiopathic injuries, though mediated through mechanical degradation and possibly confounded by other factors, cannot be assigned to a specific act or incident. The traumatic injuries, on the contrary, can be clearly associated with an incident or an action. Since idiopathic injuries are not assignable to any factor, their further differentiation into subcategories is difficult. However, the traumatic injury category is an assembly of a variety of mechanisms through which injuries occur. Among these are: (a) overexertion, (b) sudden imbalance, (c) pulling apart, (d) crushing, (e) impact, (f) slip and fall, (g) cut, (h) abrasion and laceration, etc.. In either of the two main categories and in all subcategories, a dominance of biomechanical factors is obvious.

### 3. Injury risk factors

From an examination of the published literature, it appears that the epidemiological literature can be quite diverse in its selection of factors and conclusions drawn therefrom. Hildebrandt (1987) identified from the literature 73 individual and 25 work-related factors that were considered as risk or potential risk factors for low-back injury (LBI). Taking the degree of agreement between sources as a measure of significance of the risk factors, only 11% of the individual factors and 25% of the work-related factors emerged as important factors. Millard (1988) reported falling as a cause of back injury. Kumar (1990) reported cumulative load or lifetime exposure to biomechanical loads as an important risk factor for low back pain. Most epidemiological studies are cross-sectional and do not allow a conclusive assessment of cause and effect; hence, they do not have predictive value. Musculoskeletal injuries are under multifactorial control. Whereas most risk factors identified may influence causation (some directly and others indirectly), the one that precipitates the problem must reach its threshold level for the given individual. Since the lifestyle, occupation, leisure activity, nutrition and anthropometry all vary for different people, an array of results has been reported with a variety of conclusions.

All risk factors can be placed in one of four categories: genetic, morphological, psychological and biomechanical. While not much can be done about genetic and morphological factors, knowledge of their role in causation of or association with musculoskeletal injury (MSI), combined with strategies of management of biomechanical and psychosocial factors, could allow a significant and effective control strategy. Unfortunately, however, a comprehensive study of these factors with a view to controlling MSI has not been undertaken. Genetic and morphological
factors (as non-manipulatable factors) and psychosocial and biomechanical factors (as manipulatable factors) can be used for prediction. Such a combined approach is necessary, especially when no single test or small battery of tests can be used to identify the potential MSI.

Disc herniation has been shown to have a genetic association (Lawrence 1977, Varlotta and Brown 1988). Varlotta and Brown found evidence to support the Mendelian pattern of inheritance in disk herniation cases. Annular fissure with disk protrusion was also reported to have a genetic association (Porter 1987). Some of the many common morphological factors are age, body size and spinal canal size. An increasing risk of LBI with age has been reported in many epidemiological studies (Frymoyer et al. 1980, Biering-Sorensen 1982, Burton et al. 1989). Similarly, several researchers have reported a disproportionate amount of back pain among taller men (Tauber 1970, Gyntelberg 1974, Hrubec and Nashbold 1975, Weir 1979, etc.). However, this has been refuted by Pope et al. (1985) and Heliovaara et al. (1988). A reduced vertebral canal size has been attributed as an important predictor of low-back pain (Verbiest 1954, Ehni 1969, Porter et al. 1980, Rothman and Simone 1982, Heliovaara et al. 1988, etc.).

Job dissatisfaction has been reported as an important psychosocial factor that has a strong association with low-back pain (Andersson et al. 1983, Vallfors 1985). Lloyd et al. (1979) and Frymoyer et al. (1985) reported increased anxiety, neurosis, depression and heightened somatic awareness among low-back pain patients. Biering-Sorensen (1984) found that previous hospitalization, surgery, restlessness, frequent headaches, living alone, stomach ache and fatigue were among the factors of psychosocial stress among low-back pain patients. However, their causal association with the injury remains largely speculative. Bigos et al. (1989) indicated that a significant portion of the problem antedates the actual disability.

A large array of variables constitutes biomechanical factors. Strength relative to job demand has been reported as a factor for LBI (Chaffin and Park 1973, Ayoub et al. 1978, Keyserling et al. 1980). Biering-Sorensen (1984) reported the protective effect of good isometric endurance of spinal muscles for back problems. Porter (1987) stated that the strength of the trunk muscles protects an individual from disc protrusion. However, heavy manual work has been widely reported as associated with low-back pain injury (Bergquist-Ullman and Larson 1977, Snook et al. 1978, Frymoyer et al. 1980, 1983, Schaffer 1982, Manning et al. 1984, Ayoub and Mital 1989, etc.). Exposure to vibration is reported to be contributory to low-back pain problems (Frymoyer et al. 1983, Svensson and Andersson 1983).

Similarly, a considerable body of work exists in the literature to indicate that many factors with a biomechanical impact are strong risk factors for musculoskeletal injury to the upper extremities. Repetitiveness of the work activity has been shown to be a strong risk factor for cumulative trauma disorders (repetitive strain injury) (Armstrong 1986, Stock 1991, Hales and Bernard 1996, Malchaire et al. 1996, Latko et al. 1999). Forcefulness of job activities has similarly been strongly associated with these upper extremity injuries (Armstrong 1986, Stock 1991, Hales and Bernard 1996, Malchaire et al. 1996, Sjogaard and Sogaard 1998, Viikari-Juntura 1998, etc.). However, in a study designed to assess the relative roles of repetitiveness and forcefulness, various combinations of high and low repetitiveness and forcefulness were assessed (Silverstein et al. 1986). Silverstein et al. determined that simultaneous exposure to high repetitiveness and high forcefulness of job activities was most hazardous followed by high repetitiveness and low forcefulness, low repetitiveness
and high forcefulness, and low repetitiveness and low forcefulness. In addition Schoenmarklin et al. (1994) reported that the angular velocities and accelerations of the wrists during vocational activities were also risk factors for these injuries.

Duration of exposure was reported by Hales and Bernard (1996) and Spurgeon et al. (1997) as an important variable in precipitation of MSI of the upper extremity. Hales and Bernard stated that sustained activities with insufficient recovery time led to such afflictions. Exposure duration has also been reported as an important factor in precipitation LBI by Waersted and Westgaard (1991), who found that people who worked at the same jobs part-time had significantly delayed injury onset when compared with those working full-time. Overall increased biomechanical loads whether due to posture (Armstrong 1986, Malchaires et al. 1996, Hales and Bernard 1996, Li and Buckle 1999) or to differential exposure due to handedness (Kucera and Robins 1989) or to another combination of factors (Stock 1991, Fransson-Hall et al. 1995, Grieco et al. 1998) is a significant risk factor in precipitation of MSI of the upper extremity.

4. Biomechanical basis of injuries

Occupational activities are generally kinesiologically complex, involving a large number of muscles, bones, ligaments, tendons and joints. However, to exemplify various actions taking place simultaneously at multiple joints in a human body during an occupational activity, a schematic diagram of one joint is presented in figure 1. At the musculoskeletal level, the motion of any segment at a joint is initiated by the contraction of muscle. The rate of motion at the joint will be determined by the rate of muscle shortening, and the strength of contraction determines the amount of force developed. If muscle 1 contracts, its force will be transmitted through the tendon of the muscle. Bone D will move along the path depicted by A. During this motion, bone D will glide on bone P at the joint. To facilitate this gliding, the joint cavity is filled with a biological lubricant, i.e. ‘synovial fluid’. As the motion occurs at the joint, the ligaments that encapsulate and surround the joint will undergo stretching in some parts and folding or buckling in others. To control this for preventing ballistic motion, muscle 2 will also contract but to a much lesser extent than muscle 1, and only as much as is required for a smooth and controlled motion. To return bone D to its original position or to move it in the opposite direction, there will be a role reversal between muscles 1 and 2. With motion in the opposite direction, a previously taut ligament will become lax and the other ligament will become taut. This seemingly simple phenomenon is accomplished by the use of several complex biological structures, each with their unique structures and mechanical properties interacting in a kinesiological dynamic equilibrium.

4.1. Muscle

The challenge to describe mechanical characteristics of this contractile tissue is enhanced by the range of elastic properties exhibited by each muscle. The differences are not only in gross morphology, but also in ultimate structural profiles. Regardless of differences, Hill’s model describes each muscle with a contractile element, a series element and a parallel element (Hill 1938) (figure 2). In the active state, the actin filaments slide on the myosin filaments during contraction causing muscle length shortening. Skeletal muscles can shorten up to 50% of their resting length. This shortening pulls the tendon, which in turn moves the bone at the joint, causing the segment motion. The muscles have a viscous element as well. It shows a transient
tension when muscle is quickly extended from a relaxed state. This is followed by tension decay with a rate constant closely related to intrinsic maximum speed with which the muscle can shorten when tetanized (maximal contraction). The force of contraction of muscles is dependent on the rate of the neural input and is maximum when muscle is stimulated at 100 Hz. While shortening of the muscles takes place due to contractile element behaviour, the return to its resting position after the contraction is achieved by the release of stored energy in the parallel and series elastic components.

4.2. Tendons and ligaments
The most frequently injured tissues are soft tissues (Praemer et al. 1992). Citing data from National Health and Nutrition Examination Survey II, the authors reported that the prevalence of back pain among US men and women was 16%. Using the same data, Deyo and Tsui-Wu (1987) found that 1.6% had back pain with features of sciatica. The average annual number of persons reporting intervertebral disc disorders in the USA was 1.7% (Praemer et al. 1992). Others (Leavitt et al. 1971, Vallfors 1985) reported that in most back pain cases a specific cause or a lesion could not be determined. Dillane et al. (1966) reported that in a study of low back pain seen by general practitioners in London, no specific reason for pain could be
identified in 79% of first attacks of low-back pain in men and 89% in women. Bergquist-Ullman and Larson (1977) and Damkot et al. (1984) also reported that the majority of patient with symptoms of pain had no radiation, implying no disc involvement. Statistics Canada (1991, 1995) also reported that the most frequent injury to Canadian workers was overexertion injury. Owing to their lower deformability, ligaments and tendons are most frequently affected. Both ligaments and tendons are made of the same protein—collagen. The difference between them lies largely in the density of the collagen fibres and in their arrangement. In ligaments, a smaller number of collagen fibres is arranged largely in the form of a flat sheet with fibres running in different directions. Ligaments are generally well supplied with blood vessels and nerve fibres. Tendons on the other hand are densely packed collagen fibres running in one direction in the form of a rope. Tendons are proportionally less vascular. Owing to the common building block, they have similar mechanical properties but with a difference in magnitude. A brief description of their mechanical properties is given here to emphasize the information needed in integrating the theories of musculoskeletal injury.

Tendons are located between muscles and bones to transmit the muscle tension to the bone with minimal loss of force. Hence, these structures have a very high modulus of elasticity and very high tensile strength. However, the tensile strength may vary from tendon to tendon depending on the tendon’s morphology and composition. Tensile strength has been reported to range between 45 and 125 N.mm$^{-2}$. Pure collagen has been estimated to have an ultimate tensile strength

![Figure 2. Hill's three-element muscle model.](image-url)
up to 500 N.mm$^{-2}$. Its modulus of elasticity is close to that of mild steel but the material is soft and pliant. Despite collagen’s inert appearance, its mechanical properties are quite complex and well adapted to a biological system’s needs (figure 3). Figure 3A demonstrates the visco-elastic property of collagen within its physiological range, reaching yield point and undergoing plastic (unrecoverable) deformation before undergoing rupture. Figure 3B also demonstrates the hysteresis and progressively increasing deformation with repeated cycles of loading. Owing to its visco-elasticity, collagen shows pronounced strain rate dependence (figure 3C), creep (figure 3D), stress relaxation (figure 3E) and load relaxation (figure 3F). All these properties and other mechanical behaviour of collagen (not described here) are explained by Viidik’s (1973) mechanical analogue model (figure 4).

Ligaments on the other hand provide joint support and stability, and hence have a sheet-like arrangement of fibres. They are designed to accommodate the normal range of joint motion and to bear considerable load. High-density collagen tissues, such as tendons and many ligaments, have a physiological and reversible range of deformation between 4 and 6%. Deformations beyond this value, even upon full recovery, show residual deformation. Rupture of these tissues occurs at $\sim 8-10\%$ of deformation. The combination of the foregoing basic properties of collagen serves to explain most of the common occupational injuries to the musculoskeletal system. Infrequently, the magnitude of a single load is so high that it exceeds the ultimate tensile strength of the collagenous structure and causes failure of the tissues. More commonly, repetitive exertions set the scene for occupational injuries. With repeated exertions and inadequate time for recovery from the deformation, there will be reduction in the cross-sectional area of the connective tissues stressed, thereby increasing the stress concentration. This will lower the stress tolerance of the tissue and heighten the chances of injury. Prolonged static loading resulting in creep will also render these tissues vulnerable in a similar way, in addition as well as joint play.

4.3. Bone
Bone is an anisotropic, non-homogenous, fluid-filled (perhaps hydraulically strengthened) composite material constituting the skeleton of all vertebrate animals. Owing to its greatest requirement to support loads in compressive mode, it is strongest in compression, followed by tension and shear. Like other biological components, bone possesses visco-elastic properties. It has a very high modulus of elasticity and undergoes plastic deformation after 1% strain (Evans 1973). Its mechanical behaviour can be modelled by spring, dashpot and friction bodies (Sedlin 1965) (figure 5A). On load application, spring A undergoes immediate elastic deformation proportional to the load. Dashpot C alters the response of spring B when load begins to be transferred beyond spring A. The friction blocks (D) do not move until the load exceeds some minimum level, which varies with the condition of loading. The deformation obtained by the movement of friction blocks is unrecoverable due to plastic deformation. The resulting mechanical behaviour of a bone is shown in figure 5B (Evans 1973).

4.4. Cartilage
Cartilage is a tough and pliant material with its external surface very smooth and without any geometrical irregularities. Pads of articular cartilages line the terminal ends of hard and mineralized bones at their articulating ends with other bones. The geometrical congruence of two opposing cartilage plates in any joint is extremely
well adapted to functional movements. Mechanically, cartilages are adapted to resist and endure compression and bending forces. Structurally, they are made of two

![Graph showing mechanical properties of collagen](image)

Figure 3. Mechanical properties of collagen (from Viidik 1973). (A) An idealized stress and strain plot for collagenous tissues for a single cycle of load to ultimate tensile strength. (B) Stress-strain plot of collagen when subjected to repeated cyclic loading. (C) Stiffness of collagen under different strain rates: 3, 6, 10 and 20%. (D) Creep phenomenon of collagen at a constant level of load. (E) Creep in collagen with application of a sudden fixed load. (F) Stress relaxation by holding the strain constant.
major components. First collagen fibres are arranged in wide arches with their domes directed outward (figure 6). The intervening space is densely packed with a protein—
carbohydrate complex (proteoglycan), which has enormous affinity for water and, hence, maintains a high turgor pressure. Owing to the structural arrangement, any compressive or bending force on the collagen fibres, which at the surface have a parallel arrangement, is felt as tensile load. Any ensuing deformation is easily absorbed and supported by deformable proteoglycan. With bending forces, one cartilage slides on the other, preventing any bone-on-bone rubbing.

Figure 5. (A) Mechanical analogue of bone (after Sedlin 1965). (B) Load deformation behaviour of bone (Evans 1973).
4.5. **Synovial fluid**
Synovial fluid is present in the capsules of all articulated joints and acts as a dynamic lubricant. This fluid is secreted by the synovial membrane, which lines the inside of the joint capsule. The synovial fluid is a thixotropic material and hence its viscosity is shear rate-dependent (figure 7). In rapid motions, its viscosity drops instantly to allow appropriately thinned lubrication, and in slower motions it maintains a high viscosity.

4.6. **Joint capsule**
Joint capsules are made of a strong sheet-like layout of collagen fibres surrounding the entire joint from all sides. These provide stability to the joint and limit the joint motion beyond the physiological range. These collagen fibres are laid in a bed of proteoglycan, which serves to bind the fibres together, much like cement and concrete do for a net of metal rods or wire mesh. The property of collagen in a joint capsule is the same as the mechanical properties of collagen in tendons. However, their arrangement is different and they are interspersed with elastic fibres, which together allow the joint to move within the physiological range.

4.7. **Motion at the joint**
When motion is to be performed at a joint, the muscle controlling it contracts (the intensity and velocity of contraction regulated by the central nervous system). During contraction, due to the shortening (up to 50% of the muscle’s resting length), a tension is generated in the ‘springs’ of the muscle by their stretching (figure 8). This tension is transferred to the tendon, which is much smaller in length and has a much higher modulus of elasticity than the muscle. Owing to the laxity (through crimping of collagen), the initial extension of the collagen fibres will require little force, simply
moving the front-end friction block (figures 3A and 8). Once this slack has been taken up, the non-linear springs of collagen will be engaged, which will extend through the entire elastic region.

Most muscle contractions and joint motions stop in this range and return to neutral. With continued contraction, the deformation proceeds into the visco-elastic and plastic range. The initial (visco-elastic) part of the collagen is relatively small, beyond which the forces are transmitted to friction blocks. As the latter begin to move, the collagen would have traversed the yield point and any additional deformation will be a permanent one. Since the collagenous tendon is short in length and has a physiological range of reversible deformation of 4%, very little, if any, shortening is absorbed. Most of it is then passed to the bone to which the tendon is attached (figure 8).

As one bone is pulled on the other across a joint, it begins to move by their cartilaginous ends gliding on each other, facilitated by the lubrication from synovial fluid. In motions where one segment is moving on the other with load, the cartilages at the point of contact will experience a compressive load. The latter, at the cartilage surface, with any deformation, is converted into a tensile load on the constituent superficial collagen. The extent of the motion is controlled by the degree of muscle contraction, the joint configuration, and the deformability of the ligamentous joint capsule that surrounds it. In any motion one part of the joint capsule will undergo buckling (in the direction of motion) and the opposite part will become taut as the collagen fibres are stretched, controlling the motion beyond the safe range (figure 8).

Given these structures and functions of muscles, collagen, bone, cartilage, synovial fluid and the joint capsule, an injury may precipitate at many places along the chain. This may include extremely rapid or powerful muscle contraction causing strain or sprain to its series or parallel element. Similarly, due to the visco-elastic property, the stress in collagen rises drastically for the same magnitude of

Figure 7. Mechanical behaviour of thixotropic synovial fluid compared with Newtonian materials such as water, oil or other lubricants.
deformation with a higher strain rate. Thus, the deformation magnitude may remain within physiological limits but the strain rate may exceed the tissue tolerance. The reverse may be true as well. Load, well within the physiological range, applied for a prolonged period would cause a large deformation due to creep (figure 3D). Such deformation will increase the stress concentration in the tissue for the same load due to a decreased cross-sectional area of the tissues. In addition, lengthening of the collagen may reduce the stability at the joint, causing non-physiological motion or motion beyond physiological range potentiating precipitation of injury (figure 8). Since the bones are made of strong, hard, mineralized material with a physiological range of deformation of only 1%, they rarely show change in length or injury. However, if the forces exceed the ultimate strength of the bone in the relevant mode of loading, it will undergo fracture. Bone is strongest in compression, followed by tension and shear. Repetitive or prolonged load may also affect the safety of bone by cumulative changes leading to fatigue failure at a load commonly endured without adverse consequence.

5. Theories of injury precipitation
Any advanced biological organism (e.g. human) is a consequence of a large number of variables interacting simultaneously in biological, mechanical and behavioural
domains. In any such multivariate system, it is extremely unlikely that the path or mechanism of failure will be singular and narrowly defined and controlled. The musculoskeletal system, with its various components, varying composition, and complex organization and function, can be perturbed and injured at many levels (figure 8). Which element or link in the chain will fail may vary in different circumstances. Recognizing this reality, to address many situations, if not all, the following four theories of injuries are being proposed.

5.1. Multivariate Interaction Theory of Musculoskeletal Injury Precipitation
From the foregoing consideration of the nature of injury, the biomechanical basis of injury and the risk factors one may state that a precipitation of musculoskeletal injury is an interactive process between genetic, morphological, psychosocial and biomechanical factors (figure 9). Within each of these categories are many variables, which potentiate and may effect precipitation of a musculoskeletal injury. Since the

![Diagram of Multivariate Interaction Theory of Musculoskeletal Injury Precipitation]

Figure 9. Multivariate Interaction Theory of Musculoskeletal Injury Precipitation.
permutation and combination of so many variables is extensive, there are many ways in which such an undesirable event may happen. However, it is speculated that an interaction between the relative weightings of the variables and the extent to which they have been stressed in any given individual determines the final outcome as depicted in figure 9.

5.2. Differential Fatigue Theory

Occupational activities are designed to meet occupational demands and not to optimize biological compatibility. For these activities to be of any economic and industrial value, they have to be repetitive. Any of these activities employ a large number of muscles at various joints in such industrially relevant human physical motions. Asymmetric motions are very frequently components of these repetitive industrial activities and are common in workplace (Garg and Badger 1986, Kumar 1987, McGill 1991, Kumar and Garand 1992, Marras et al. 1993, Waters et al. 1993, Kumar 1996, etc.). Depending on the activity, different joints are also differentially loaded, and depending on the motion to be performed, different muscles operating the joints may also be differentially loaded. This differential loading of muscles may also not be proportional to the individual muscles’ capabilities. Such differential prolonged and/or repeated loading is likely to do two things. In the short-term, due to the disproportionate demands on different muscles, it is likely that the different muscles operating a joint may undergo different amounts of fatigue and the rate with which they fatigue may also be different. In the long-term, however, if such activities were to continue, the foregoing altered muscle kinetics may result in joint kinematics and loading pattern different from the optimum and natural loading pattern according to the geometry and design of the joint.

Kumar and Narayan (1998) studied 14 trunk muscles (erector spinae at T10 and L3 levels, latissimus dorsi, external and internal obliques, rectus abdominis and pectoralis bilaterally) in a posturally stabilized axial rotation in a single fatiguing contraction, maintained at 60% of maximal voluntary contraction (MVC) of the individual. To achieve this, 50 subjects (27 male, 23 female) were seated and stabilized in an adjusted Axial Rotation Tester (AROT). The AROT was placed in isometric mode and subjects exerted 60% of their previously measured MVC. The subjects were provided with visual feedback of their level of effort. The EMG was sampled at 1 kHz and analyzed using a Fast Fourier Transform (FFT) routine. The median frequency (MF) was plotted against time (10% of the task cycle). A decline in median frequency (an indicator of fatigue) was tracked through the entire duration of the contraction. Raw data normalized for each muscle and plotted against time in the task cycle, demonstrated a significant decline in the median frequency; and, the rate of decline was also different in different trunk muscles (figure 10). In yet another experiment, combined motions of simultaneous flexion and rotation, and extension and rotation from 40° flexed and 40° rotated postures were studied. Subjects maintained 80% of their previously measured MVC for a maximum of 2 min. The EMG of trunk muscles was recorded at 1 kHz and subjected to spectral analysis. The MF was plotted at every 10% of the task cycle to reveal a significant decline in MF with time which occurred at rates different in different muscles, demonstrating differential fatigue (figure 11). If EMG fatigue leads to force fatigue, a differential fatigue is likely to affect two aspects of the joint. First, as each muscle
acts on the joint by its own connective tissue, a component which is adapted to transmit the normal and natural forces is likely to be affected differently in different parts of the joint structure. By a proportionally higher prolonged or repeated loading, the connective tissue component will be called upon to perform at a proportionally higher rate or for a longer time. The friction blocks and viscoelastic elements of connective tissues (figures 4 and 8) may cause disproportionate deformation of these connective tissues, thereby altering the joint stability. Second, as the fatigue progresses, the muscle concerned will be able to generate less and less force. Such a situation may create a kinetic imbalance. Superimposition of this kinetic imbalance on previously created kinematic imbalance (connective tissue deformation) will potentiate uncoordinated, sudden and perhaps even abnormal or unnatural motion at the joint. This can create a significant stress concentration in some tissues, causing an injury. It is probably for these reasons that >60% of all injuries to the back involve rotation of the trunk (Manning et al. 1984). Others have also reported trunk rotation to be the predominant mechanical factor in LBI and pain (Ralston et al. 1974, Snook et al. 1978, 1980, Frymoyer et al. 1980, 1983, Schaffer 1982, Duncan and Ahmed 1991, etc.)
5.3. Cumulative Load Theory

Biological tissues are like all other physical materials with a finite life, and similarly subject to wear and tear. They are capable of self-repair but undergo mechanical degradation with repeated and prolonged usages. All biological tissues are viscoelastic and their prolonged loading may result in permanent deformation. Repeated load application may also result in cumulative fatigue, reducing their stress-bearing capacity. Such changes may reduce the threshold stress at which the tissues fail. Kumar (1990) reported a strong association between cumulative load (biomechanical load and exposure time integral over entire work life) and LBI/pain \((p < 0.01)\). Among nursing aides employed in public sector in Alberta, Kumar reported 42.8% of males and 64.6% of females were having back pain. Data on length of service before the onset of the first episode of back pain in age, sex, body weight, height, occupational activities and recreational activities matched sample revealed in the pain group that the number of cumulative years worked at which pain precipitated was significantly greater than the number of cumulative years worked by the no pain group \((p < 0.01)\). The mean cumulative compression and shear loads for subjects were obtained from the biomechanical analysis of each of the job tasks performed. Each of the task cycles was biomechanically analysed for compression and shear load on the spine at the discrete time intervals dividing each task cycle into 200-ms
chunks. By summing the load time product for 200-ms chunks, the total load time product for one task cycle was obtained. This load time product (N.s) was multiplied by the number of cycles performed on a shift to obtain the cumulative load of the shift, both for compression and shear. By similarly analysing all other tasks performed by the subjects in the sample’s total cumulative compression and shear loads in units of Newton second (N.s) were obtained. Such mean cumulative compression and shear loads sustained by the no-pain and pain groups for male and female samples in the study are presented in figures 12 and 13. A Student t-test of the comparison of the means revealed that the cumulative compression at thoraco-lumbar and lumbosacral discs was significantly higher in the pain group (p < 0.05 or better). The cumulative shear in the male pain group was also significantly higher than that of the male no-pain group (p < 0.02). The mean cumulative daily compression and shear loads and their standard deviations at the thoracolumbar and lumbosacral regions of the male sample respondents with and without pain were not significantly different. The data also revealed that there was no significant difference between pain and no pain group in any of the biomechanical spinal load variables when compared for one task cycle. However, the total time spent working by the pain group was significantly higher than that of the no-pain group, thus causing a significant difference in cumulative loads (p < 0.001). The data for such lifetime cumulative load exposure for pain and no pain groups are presented in figure 14.


![Figure 12. Mean cumulative compression and shear loads in pain and no pain groups in the male sample in mega-Newton seconds (MN.s).](image-url)
loads of between 20–30, 30–40, 40–50, 50–60, 60–70 and 75% of the estimated ultimate compressive strength (UCS) of spinal units were applied at a frequency of 0.25 Hz. They found that both the magnitude of the load and the number of cycles affect the spinal unit failure. At lower loads high repetition and at higher loads low
repetition produced fatigue failures. When their specimens were loaded between 50 and 60% of the UCS, 92% suffered fatigue failures after 5000 cycles. A 91% fatigue failure rate was reported after 500 cycles when the load was increased by an additional 10%, and at a load of 75% of UCS the fatigue fractures were precipitated only in 10 cycles. Long-range, low-grade loading of the spine will be difficult to control and measure. However, looking at the results of Brinckmann et al. (1987, 1988), it would appear that physiological limitations strongly favour biological safety by preventing cumulative load from rising rapidly as the maximum voluntary contraction for level can neither be held for a long time, and nor can it be repeated in quick succession. In addition, the compression generated by the maximum voluntary contraction ranges between 68 and 77% of the ultimate compressive strength (Kumar and Mital 1992). When one considers that the MVC can be sustained only for a few seconds and that it decays exponentially with the duration of the hold (Rohmert 1973a, b), it is obvious that such cumulative compressions cannot rise rapidly. Also, the MVC cannot be repeated without long rest pauses. Rapid repeated trials of force exertion degenerate quickly with a drastic reduction in magnitude, thereby preventing the total exposure (load × cycle) from rising. It must be borne in mind that rapid cyclic loading does not allow much needed recovery time to the visco-elastic biological tissues. This in turn progressively accentuates the deformation, rendering the tissues more vulnerable to injuries.

5.4. Overexertion Theory
Exertion implies physical effort and thus overexertion will mean excessive exertion which exceeds the tolerance limit of the system or system components. Every physical activity requires force generation (or application) from one position (posture) to another (motion) for a certain length of time (duration). Thus, overexertion by definition will be a function of force, duration, posture and motion. All these variables are complex entities and have been discussed in detail before (Kumar 1994). However, a brief account is given here to make a case for this theory.

5.4.1. Force: With respect to the validity of the concept, a large number of epidemiological studies reported in the literature reveal beyond doubt a strong association between exertion and injuries to the various regions of the human body. Statistics Canada (1991) reported that the largest portion of all occupational injuries (48%) was overexertion injuries. A number of cross-sectional and case studies have shown that musculoskeletal disorders were caused in the neck and shoulder regions by occupationally increasing muscle contraction. Other musculoskeletal conditions have also been reported associated with exertion, such as supraspinatus tendonitis, myofascial syndrome and cervicobrachial disorders and neck and shoulder regional muscle disorders. The argument of association is supported by the observation that when muscle and joint loads were reduced in some interventions and eliminated in others through job redesign, the incidence and severity of neck and shoulder disorders also decreased.

The relatively recent and epidemic increase of upper limb repetitive strain injury (RSI) or cumulative trauma disorders (CTD) in many occupations has been largely attributed to the external loads, postural load levels, and repetition of posture and/or force application. All these three factors that have been reported to be causally associated with injury also develop exertion of the structures. Awkward, fixed,
constrained or deviated postures can overload the muscles, ligaments, and tendons and also load the joints in an asymmetrical manner.

In a cross-sectional study, Silverstein et al. (1986) reported that industrial jobs involving low force and little repetition had the fewest CTD cases and that those involving high force and frequent repetition had ~30 times greater morbidity, indicating an interactive behaviour of these risk factors. Jobs requiring high force and little repetition or low force and frequent repetition had morbidity rates in between the extremes.

The magnitude of the mechanical load has been associated with low-back pain incidence by many authors (Hult 1954, Magora 1970, 1972, 1973, Chaffin and Park 1973, Chaffin 1974, Snook 1978, Andersson 1981, NIOSH 1981, Heliovaara et al. 1987, Kumar 1990, Statistics Canada 1991, etc.). In the USA overexertion was claimed to have caused low-back pain in >60% of low-back pain patients (Jensen 1988). Of all heavy load handlers, 45% were reported to have sought medical help for low-back pain in a 10-year study by Rowe (1969).

Forceful static or repetitive contraction of muscles causes their corresponding tendons to stretch, thereby compressing their vascular epitelen, peritenon and endotenon microstructures. This in turn causes ischaemia, fibrillar tearing, and inflammation. Frictional damage to the sheaths can occur with repetitive motion. Awkward postures contribute to muscle tendon inflammation by compression of the microstructure and by increasing the force requirements of the tasks. Tissue injuries are known to occur in maximal exertions; tissue tolerance characteristics are therefore considered to be factors of paramount importance. Evans and Lissner (1959) and Sonoda (1962) reported that mean ultimate compressive strengths of human spinal units are age dependent (3400 N for those > 60 years, and 6700 N for those < 40 years). On this basis, the National Institute for Occupational Safety and Health developed its Work Practices Guide for Manual Lifting (1981), which was updated by Waters et al. (1993).

Different levels of strength exertion have different levels of physiological demand. Rohmert (1973) demonstrated that the duration for which a muscular contraction can be sustained depends on the level of contraction. Contractions of the levels of 15–20% only can be held indefinitely as a continuous hold. Higher levels of contraction impede the blood supply and thereby availability of nutrients and oxygen to the muscles doing the work. Furthermore, such an occlusion of the blood supply also interferes with the removal of metabolites, which results in a sensation of pain. Except for static postural demands to manage a dynamic task, industrial jobs are not prolonged static holds; rather they generally require short time repetitive exertions. These exertions remain constant for the task in hand (constant level, CL) though they may represent a different proportion of the MVC for different workers. Ayoub et al. (1978) and Chaffin et al. (1978) reported that as the strength required on the job increases, the injury incidence also increases. It is, however, unclear about what level of strength requirement may be considered risk neutral. It has been suggested that due to an integrative capacity, the human perception of a preferred work level may optimize the balance of physical and physiological factors in favour of system safety (Kumar and Mital 1992). Kumar and Simmonds (1992) also reported that people underestimate precision, power and gross motor efforts < 40% of MVC and overestimate efforts greater than that value. The pattern of perception was repeatable and reliable. It may, therefore, follow that an assessment of a preferred level (PL) based on the perceptual sense may provide a level of exertion
that may be risk neutral. An exertion above PL will increase the job-mediated risk (JMR) of overexertion injuries while exertions below PL will remain risk neutral (figure 15).

5.4.2. *Duration of exertion:* The significance of the time variable of exertion is dependent on the type of contraction, the magnitude of contraction, the recovery period and the repetition of the activity in question. With any activity there will be phosphagen and glycogen depletion from the intrinsic muscular sources before the aerobic glycolysis ensues, depending on the circumstances. Such metabolic response will also result in accumulation of lactate. Following high intensity activity, up to 70% replenishment of phosphagen on the one hand and removal of lactate on the other may take place in 30 s, while near complete replenishing may take up to 5 min (Astrand and Rodahl 1977). The endurance times of submaximal contractions as a percentage of MVC are widely reported (Rohmert 1973a, b) even though the exact time required for near full recovery is unclear. The duration of submaximal contractions (at different levels of MVC) and the corresponding time durations by which no significant adverse physiological and metabolic change has taken place are unclear. However, Molbech (1963) reported that the strength of isometric exertions (MVC) declined from 85 to 60% of MVC as the frequency of exertion was increased from 5 to 30 min$^{-1}$.

![Figure 15. Generic relationship between the overexertion caused by the job stress variables and safety of the system.](image-url)
5.4.3. Job range of motion: The geometric relationships of the muscle, tendon and bone with respect to the joint vary with the degree of motion at the joint. At the extremes of their range, the joints are at the greatest mechanical and physiological disadvantage. Though the exact angle at which the best mechanical and physiological advantage is available may vary somewhat from joint to joint, generally the mid-position of the range is perceived to require the lowest effort for performance. This can be designated the risk neutral position. Deviations from the mid-range position to either side will represent increasing hazard. The 20% range around the mid-position, that may either be subjectively considered as the comfortable zone or be the zone in which the effort required to move increases (by virtue of the position), is small. Thus, this zone may be designated the risk neutral zone. Deviations from this or motion in excess of the mid-range may constitute a job mediated motion risk. Deviation from the mid-range beyond the risk neutral zone will be considered hazardous.

In addition to failure in compression mode, the tissues can be strained beyond their physiological limit and precipitate injury if utilized beyond the physiological range of motion. To investigate this aspect, Adams and Hutton (1986) compared the maximal in vivo range of flexion of the lumbar and lumbosacral vertebral joints with that of osteoligamentous preparations. The active ranges of flexion of the vertebral joints were reported by them to be 10% short of that of the osteoligamentous preparation. Such a difference between extreme forward flexion and elastic limit of osteoligamentous preparation ensures the safety from possible strain injuries by preventing excessive deformation and generation of high tensile stresses. Adams and Hutton (1986) also reported that in a typical lumbar motion segment, a 2% reduction in flexion at its elastic limit reduces the resistance to bending moment by 50%. This 2% withdrawal of flexion causes a 50% reduction in the tensile stresses on the intervertebral ligaments and the annulus. At the limit of the active flexion, the osteoligamentous preparation provides half of the resistance to bending moment exerted by the upper body in forward bending (Adams et al. 1980). Considering most activities of daily living and occupational activities, it is obvious that only modest ranges of motion are commonly used. Thus, such interplay between posture and material properties ensures at least a safety margin of 50% in force enduring capacities. A given degree of muscle contraction is evoked for postural stability and readiness to move to the next phase of activity. Any sudden force may, however, tend to overcome the visco-elastic resistance of the muscle due to high strain rate deformation. Such forces may result in sprains and strains as minor injuries before structural damage can occur.

5.4.4. Force, duration and posture/motion interaction: At work, the biomechanical hazards comprising force, effective exposure and postural load interact to create a composite job mediated risk of injury. When the magnitude of this risk exceeds the tissue tolerance capacity, an injury is precipitated. The latter can happen in a single forceful exertion by either exceeding the ultimate strength of any component in the system (figure 8) or exceeding the tolerable strain rate (figure 3c). An overexertion injury precipitation can also occur in a situation when the combination of exertion and repetition does not allow adequate recovery and leads to overexertion.

Based on this logic, Kumar (1994) proceeded to integrate three components (force, effective exposure time and postural load) of injury causation into an integrated index (II). He proposed that the sum of margin of safety (MOS) and job-mediated risk of
injury (JMR) will be in unity due to their reciprocal relationship. Thus, since MOS + JMR = 1, it follows that JMR = 1 − MOS and MOS = 1 − JMR. Thus, the magnitude of JMR (overexertion) can be obtained by subtracting the total safety from 1. If there are three risk factors (R₁, R₂, R₃), there will be three corresponding safety factors (S₁, S₂, S₃). Each of the three safety factors can be expressed as:

\[
S_1 = 1 - R_1 \\
S_2 = 1 - R_2 \\
S_3 = 1 - R_3
\]

The MOS of the entire system will be proportional to the product of the three safety factors described above. A multiplicative relationship accommodates the interaction among the relevant variables, which is a necessity of a multifactorial system. This relationship also ensures a decline in MOS as the number of risk factors rises. Kumar (1990) reported such a phenomenon in the case of safety of the low back. According to the relationship described above, the MOS will be product of the three safety components according to the following expression:

\[
\text{MOS} \propto S_1 \cdot S_2 \cdot S_3
\]

Since JMR = 1 − MOS, it can be written by substituting the MOS with its individual components:

\[
\text{JMR} \propto [1 - \{(1 - R_1)(1 - R_2)(1 - R_3)\}].
\]

By inserting the proportionality constant, \( K \), this equation can be written as:

\[
\text{JMR} = K[1 - \{(1 - R_1)(1 - R_2)(1 - R_3)\}].
\]

JMR is the risk of causing overexertion due to the job and as such is the overexertion.

Thus, the Overexertion Theory is different from the Cumulative Load Theory resulting from an interaction between the force, the exposure time and posture/motion proposed by Kumar (1994).

6. Conclusion

In the foregoing description four theories of musculoskeletal injury precipitation have been described: (1) Multivariate Interaction Theory, (2) Differential Fatigue Theory, (3) Cumulative Load Theory and (4) Overexertion Theory. It needs to be stated that in each individual all four mechanisms described in these theories will be simultaneously operative. The actual injury precipitation in any given incident may occur as a result of events from any of the four theories described. However, an injury is likely to be mediated through the interaction of variables in all of the four theories and their bearing on the interactive behaviour of the multiple visco-elastic components in a complex biological system. Control strategies, to be effective in any given situation, may need to assess different operative mechanisms and their possible role in the injury precipitation during interaction of the biological system and occupational stresses.

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