Exercise-Induced Central Fatigue

A Review of the Literature with Implications for Dance Science Research

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Abstract

The complex interplay between cortical and subcortical networks essential to motor performance is altered when muscles fatigue. The construct of exercise-induced human muscle fatigue has been attributed largely to the loss of a peripheral muscle's ability to produce force. Far less understood is "central" fatigue, the result of alterations in central nervous system function. Central fatigue manifests as inadequate motor drive to the muscles and can occur even at levels of sub-maximal voluntary force. This study reviews the literature on exercise-induced central fatigue and its impact on motor performance. While exploring the conditions that may contribute to central fatigue, it addresses perceived exertion, repetitive strain, and their relationship to central fatigue. Evidence supporting possible training protocols designed to offset central fatigue while speculative will be cited as potential areas of investigation for dance scientists.

Human muscle fatigue places highly among the many biological, environmental, and personal factors impacting on skilled motor performance.[1] Of all the physiological and biochemical adaptations occurring in response to physical exercise, muscle fatigue has been relatively under-researched—especially with regard to field research, where multiple conditions affecting performance are difficult to control.[2] The dearth of research on fatigue is particularly apparent in dance science. Although fatigue is considered a contributing factor to the etiology of dance injuries,[3] the effect of any specific dance movement or other environmental factor on the onset and pattern of fatigue is conjectural.[4] A search for evidence to support training protocols to offset fatigue among dancers produced only two articles that dealt directly with protocols potentially bearing on central fatigue.[5,6] Nonetheless, knowledge can be gained from exercise science about the multifactorial nature of fatigue, both central and peripheral.[7,8]

Most research of this nature has been conducted on the delivery of oxygen and the mechanics of maintaining a fuel supply to the peripheral musculature.[9] To produce peripheral muscle fatigue experimentally, subjects are seated and stabilized while they voluntarily execute maximal (or submaximal) single or repetitive efforts (maximal voluntary contractions, or MVCs) of small joint actions (elbow flexion-extension or thumb abduction and adduction) with or without added electrical stimulation.[8,10] Under these restricted laboratory conditions researchers have identified a number of physiological impairments in the contractile mechanism that contribute to exercise-induced, peripheral muscle fatigue. These include problems occurring with neuromuscular transmission and propagation down the sarcolemma, calcium release and uptake in the sarcoplasmic reticulum, availability of metabolic substrates and accumulation of metabolites, and actin-myosin crossbridge interactions.[7,11] These peripheral mechanisms alone, however, cannot account for the decrease in maximal voluntary force that occurs during muscle fatigue.[8,12] A portion of the fatigue phenomenon appears to be due to changes within the central nervous system (CNS) —that is, to "central fatigue" (CF).[12,13] CF is defined as an activity- or exercise-induced decline (progressive reduction) in voluntary activation of a muscle (or muscle group).[8] It differs from peripheral fatigue, which implies a reduction in the ability of muscle fibers to produce force.[14] While peripheral fatigue and CF are interrelated, CF is due to supraspinal and spinal mechanisms that cannot reasonably be explained by dysfunction within the muscle itself.[8]

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Figure 1 Multisystem Impact on Fatigue. Although usually described as descending, it is more realistic to view the process as reciprocal and interdigitated.

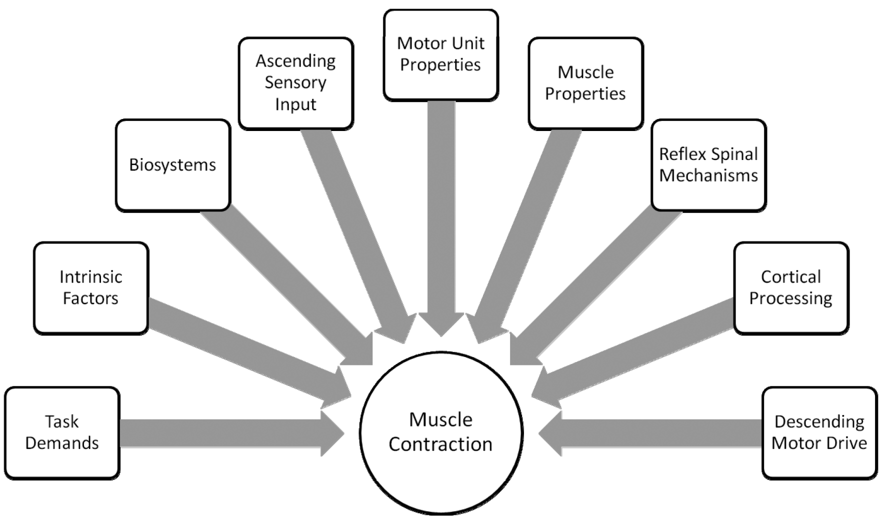

Figure 2 Multiple factors determine a muscle's ability to fire, including the intrinsic factors of the individual (age, previous training, health, etc.), the task and environmental demands, and the number of other biosystems operating simultaneously.

The purpose of this article is to address the causes and manifestations of exercise-induced CF and the evidence underlying the effects of CF on motor performance in healthy (i.e., non-neurologically impaired) persons. For the sake of simplicity of discussion, only the supraspinal (cortical) level of motor control processing within the CNS will be addressed and not the impact of sub-cortical and spinal effects on fatigue or that of other biosystems. While it is necessary to distinguish CF from other fatigue phenomena, readers are advised to consider CF within the larger framework of the ecology of human behavior. Intense exercise stresses all biosystems, challenging researchers in determining the degree of CF involvement in the fatigue process. Reductions in voluntary muscle force, velocity, or power resulting in fatigue can arise from any one of multiple biosystems (or their interactions)—metabolic, cardiovascular, humoral, and neurological.[7] The ultimate state of the contractile potential of a motor unit is the sum of these many factors (Fig. 1). Furthermore, intrinsic variables (e.g., the individual's age, sex, health, past training, etc.) interact with environmental conditions and tasks (Fig. 2).[15] Finally, decision-making is essential in physical exercise participation, and therefore intentional goals, anticipation or expectation of reward, motivation, perceived effort, and other cognitive and psycho-physiological factors affecting action planning and execution figure highly in the relationship of fatigue and performance.[16] For these reasons and to link the topic to issues in dance training, the topic of perceived effort as it relates to CF will also be introduced. Finally, the article will cite evidence (albeit speculative) supporting the development of training protocols to offset fatigue as a possible area of investigation for dance science.

Fatigue: A Confounding Construct

From the neurophysiological perspective, fatigue is regulated by the central nervous system to ensure homeostasis and bodily safety.[17] Five physiological models cover the scope of fatigue research, testifying to the multi-factorial complexity of the subject: 1. the cardiovascular or anaerobic model, 2. the energy supply and energy depletion model, 3. the muscle recruitment and power output model, 4. the biomechanical, and 5. the psychological and motivational models.[2] Within this array, CF belongs most readily to the third category, involving the neurophysiology of CNS-generated muscle recruitment and power output. Secondarily, CF resonates with the cognitive and emotional aspects of motor performance represented in the fifth model.[2] It is improbable, however, that any one of the five fatigue models or combination thereof fully explains human exercise performance under all conditions. Knowledge is lacking as to the precise factors that underlie fatigue and limit performance in a variety of types of exercise.[2]

Between 1998 and 2010 at least 15 definitions for exercise-induced muscle fatigue evolved from neurophysiological research.[7] These definitions reflect the involvement of both peripheral and central levels of motor control in the fatigue process. For example, muscle fatigue is defined as: 1. the "loss of ability to produce *voluntary* muscle force either with a single muscle or muscle group"[18]; 2. "the exercise-induced reduction in the ability of a muscle to produce power or force,[14] *irrespective of task completion*"[19]; and 3. "the failure of a muscle to maintain a required or *expected* force"[20] (italics the author's). The notion that peripheral fatigue occurs independently of task completion suggests that central CNS processes contribute to the onset of fatigue and its fluctuating pattern of expression.[19,21] The italicized phrases suggest that task conditions, as well as higher level processes such as perceptual and cognitive aspects of motor control, are important modulators of nervous system activity in the fatigue process.[7]

Overview of Neurophysiology in CF

For a muscle to produce force, it must receive an adequate stream of motor impulses from the CNS. The firing of the motor neuron during voluntary muscle contraction depends on the appropriate level of descending (efferent) output to the motor neurons in the ventral horn of the spinal cord. Known as "central drive,"[7] these impulses arise from supraspinal motor areas (the motor cortex and subcortex) and descend through the spinal cord to the peripheral motor neuron pool governing the muscles involved in the activity. Cortical CF manifests as the inability to generate and maintain an adequate number, magnitude, and sequencing of neuronal impulses to sustain maximal drive of the working muscles. This failure of central drive is accepted as the most likely explanation of exercise-induced CF.[13] A hallmark sign is the demonstrated ability of a muscle to generate additional force after "task failure." Task failure is defined as the voluntary or involuntary stopping of an assigned task before its anticipated completion.[22]

The research paradigm for describing the decline in sustained force generation is arrived at by observing either one maximal contraction (MVC) or a series of submaximal (SMVC) voluntary contractions. SMVCs utilize a lower percentage of motor units over time than MVCs, the magnitude of which varies with research parameters. In assessing CF under research conditions, artificial stimulation is added to the brain at the point of task failure to evoke further twitches (known as "motor evoked potentials" or MEPs). The appearance of MEPs on electrophysiological recordings suggests that the muscle still possesses the capability to generate force.[23,24] Regardless of the type of muscle action, the appearance of CF implies that the descending impulses from the motor cortex are less than maximal, or more realistically less than optimal, to meet task demands.[12] The additional contractile force generated either voluntarily or through mechanical stimulation (electrical or magnetic) indicates that the muscle is not completely fatigued despite a momentary loss of adequate CNS drive.

One difficulty in localizing the scope and magnitude of synaptic transmission in CF is that few research studies have tracked these impulses through time.[25] CNS function is a manifestation of continuous brain activity: past, present, and future.[26] CNS and motor unit responsiveness is labile, and what governs the optimal degree of excitation of these descending impulses depends on central processes as well as environmental factors (e.g., temperature, altitude), local and systemic metabolic factors, and the current demands of the task.[7] The exact cause of altered responsiveness of descending motor drive in CF is not understood.[8] With repetitive action, it is not clear whether loss of maximal voluntary force is due to changes at the level of the cortex and the motor neuron.[27] Variations in voluntary force generation result from a sequence of neural events that interact reciprocally with multiple sensory-motor pathways to alter motor neuron firing properties.[24] In CF, the decline in central motor drive results in a decrease in excitability or an increase in central inhibitory processes or both.[8] Altered synaptic strength during fatiguing voluntary contractions can include changes in excitatory or inhibitory inputs to motor cortical neurons[28] and depletion of neurotransmitters at the synaptic vesicles.[29] Multi-modal synaptic processes have time courses varying from milliseconds to minutes. Time alone can facilitate and depress the release of transmitters, altering membrane properties.[30] Further, impairments at any level of motor control (ascending or descending) can potentially limit the magnitude and duration of muscle contraction.[7] Local changes in the motor units themselves, as well as ascending (afferent) feedback, can in turn alter the motor cortex.[24] Peripheral input from local interactions of muscle receptors (e.g., muscle spindle or other somatosensory input), recurrent inhibition, and ascending input back to the CNS can alter sensorimotor topography and result in a net decline in descending drive to the muscle.[24]

Several neurophysiological theories help to explain the loss of central drive. All of these appear to support the functional capacity of the brain to maintain body safety (homeostasis).[2] First, an accumulation of brain metabolites (such as an increase in ammonia) can alter local brain pH, causing a reflex decrease in force in order to prevent muscle damage.[31] Second, altered concentrations of serotonin and other neurotransmitters (such as dopamine and acetylcholine) affect the density neural impulses and the synaptic connectivity available to reach the exercising muscles.[32] Third, inhibitory reflexes arising from exercising muscles are feedback to the spinal cord and supraspinal levels, resulting in reduction of skeletal muscle recruitment at the alpha-motor neuron level.[32] The extensive interplay between central and peripheral processes in motor control implies that the presence of CF not only affects muscle strength and power but also impacts negatively on cognition.[13] proprioception,[33] normal physiological tremor,[34] and postural control.[35]

Brain Stimulation and CF

With the advent of non-invasive neuro-stimulation technology, researchers can infer the presence of CF and distinguish it from other physiological phenomena.[8] By pairing electromyography (EMG) with these technologies, brain behavior can be observed "online." Of the variety of non-invasive technologies now available, paired-pulse Transcranial Magnetic Stimulation (TMS) has proven particularly practical in fatigue research generally,[24] although no fatigue studies have been conducted on dancers. TMS has provided the first concrete evidence in humans of the existence of direct monosynaptic connections from the motor cortex to spinal motor neurons.[26] Using pulsed electromagnetic fields, TMS helps discern to some degree the loss of force due to inadequate descending motor drive from the motor cortex. TMS stimulates the (motor) cortex, activating intra-cortical circuitry as well as cortico-fugal (descending) axons, eliciting excitatory responses in target muscles (MEPs). The level of activation of cortical neurons can be inferred from increments of magnitude and duration of these MEPs. By observing the spatiotemporal patterns of these MEPs, researchers can extract various phases of motor planning and execution such as the onset, magnitude, amplitude, and duration of contraction, and the relaxation and recovery phases.[13,21] Latency patterns of activity indicate the presence of CF.[35]

When TMS is conducted over the motor cortex during an isometric MVC of the elbow flexors, for example, a small twitch-like increment of force is evoked over and above the subject's maximal effort.[36] This additional force generation suggests that (at the moment of stimulation, at least) the level of cortical excitation needed to drive the motor neurons to produce maximal muscle force is insufficient. While TMS provides a window for evaluating motor pathway strength and muscle fiber recruitment, research results need to be viewed with caution. Mechanisms underlying the diagnostic value and development of CF during exercise are hypothetical for several reasons.[24] First, total body stabilization is necessary to avoid artifacts. Therefore, MEPS are most readily accessed in smaller muscles such as the ab- and adductors of the thumb. Second, in analyzing data from TMS studies the topography emerging from elicited responses is not a hard-wired map of corticospinal efferent projections. The profile of MEPs is highly variable and context-dependent.[37]

For example, considerable variability in MEP recordings can exist depending on the anatomical, physiological, and contractile characteristics of the muscles and the patterns of agonist-antagonist interaction.[10] Maximal efforts from respiratory muscles produce a very different CF profile than grip efforts, for example.[38] Central motor drive may adjust to match the force generating capacity of the muscle fibers during slow movements and isometric contractions.[10] Further, CF and peripheral fatigue profiles are not equivalent. Peripheral fatigue levels can be high despite the lack of CF and vice versa.[39] In studies involving persons with chronic fatigue syndrome performing submaximal, symptom-limited cycling exercise, experimental subjects' syndrome showed little or no impairment in their ability to exert muscle force if they were highly motivated,[40] These subjects generally rated their levels of PE higher than controls, even though muscle laboratory values were normal before and after exercise.[41]

The fatigue pattern appears dependent not only on the type of task but also the body parts involved in the task.[42] Subjects trained in a kicking task, for example, experienced less fatigue when the same kick was performed consistently with one leg than with alternating legs,[43] a finding which suggests that motor commands involving one leg are significantly different from those involving two legs, and that training-induced decreases in fatigue involve adaptations within the CNS as well as the muscle.[43] More clarification is needed to distinguish physiological parameters from neuropsychological factors (e.g., perceived effort and motivation), differentiating the onset of muscular exhaustion from fatigue-induced task failure.[44,45]

Perceived Effort

The fact that CF can bring exercise to a halt before the muscles are fully fatigued physiologically suggests that "mental fatigue" contributes to the pattern of CF expression.[32,46] Many central neural processes have an impact on motor performance, including arousal, mental effort (attention), motivation, perceived effort, and pain.[47] Within this array, mental fatigue is an elusive concept, encompassing subjective feelings related to physical exertion (e.g., the perceived effort occurring with weightlifting), mental concentration (e.g., the effort involved in computing statistics), or self-restraint (e.g., quitting smoking).[47] The voluntary effort required to maintain maximum exertion during high intensity exercise reportedly is unpleasant.[48] The conscious sensation of fatigue does not arise directly from the accumulation of peripheral metabolites but rather emerges from subconscious parts of the brain.[17] This implies that the sensation of fatigue is more an emotional phenomenon than a physical one.

Training plays a role in being able to allay feelings of fatigue.[48,49] The ability to sustain exercise arises in part from a conscious effort to override discomfort and maintain focused concentration on the goal. Pacing strategies appear to be vital tools in enduring self-regulated, high intensity exercise.[49] Maximal central drive can be difficult or impossible to sustain, even for highly motivated, well-trained individuals.[11] Additional contractile force can be evoked voluntarily by asking subjects to generate an extraordinary effort.[17] Even with training, however, maximal voluntary effort never recruits 100% of the motor units available.[23]

Perceived effort (PE) is a well-recognized, measurable phenomenon in exercise science.[47] The experience of physical exertion is a feeling of energy being exerted, and the gold standard of measurement is Borg's Ratings of Perceived Exertion (RPE).[50] When a muscle contracts, actual physical "work" is produced and energy expended. PE, however, appears to be unrelated to the work completed. PE is believed to be one motor control strategy the nervous system adopts for pacing to avoid irreversible tissue damage as exercise intensity increases.[50] It is also a useful tool for moderating exercise intensity by providing direct feedback about task difficulty, allowing the subject to adjust exertion appropriately. Feelings of physical effort (RPE) then further promote conservation of energy, and finally, effort contributes to the sense of conscious will or agency.[47]

PE is important in the neuropsychology of fatigue because it can be a limiting factor in task completion and thus mistaken for true fatigue.[51] From a neurophysiological perspective, the perception of force appears to be cued centrally at the highest levels.[52] Central motor pathways, referred to as "corollary discharge"[53] or "efference copy,"[54] generate feelings of effort. Evidence suggests that the sense of effort is strongly influenced by the magnitude of the corollary discharge (efferent activity from the motor cortex related to the motor command).[52] The greater the efferent activity, the greater the feeling of effort.[47] Intuitively, it would appear that the harder one exerts effort, the higher the rate of tension and the expected rate of perceived exertion would be.[51] A one-to-one correlation does not exist, however, between task difficulty or the level of exertion of effort and the consequent onset of fatigue. Many different factors can affect the outcome.[55] In a study comparing the responses of males and females to a repetitive knee extension task, female subjects reported greater feelings of "exhaustion" and "weakness," neither feeling being fatigue proper.[10,56] Some reserve always exists, even in persons with compromised health, such as those with fibromyalgia, who in an endurance cycling study showed no detectable CF despite complaints of extreme tiredness.[56]

Effort perception results from the integration of multiple afferent signals from a variety of perceptual cues, both central and peripheral.[51] In healthy athletes, these feelings can easily be overridden. A very high degree of motivation can boost maximal CNS drive. Additional muscle force also can be generated and sustained voluntarily by use of encouraging instructional feedback,[57] as well as by the subject's belief that he or she is producing maximal effort.[10] Further, exertion can be energizing, a positive benefit of exercise that fosters a sense of vitality and improves mood in persons who are unfit or depressed.[58] These emotional and psychological benefits appear to be much more strongly associated with "moderate" and "light" exercise,[59] however, whereas dancers (and other elite athletes) are more likely to experience burnout from the intensity of rehearsal schedules.[60] Pacing appears to be the strategy of choice for enduring self-regulated, high intensity exercise.[49]

CF with Submaximal Repetition

The disproportionate increase in PE reported during prolonged low-force contractions is one indicator of the nonlinear relationship between force and effort.[21] CF manifests differently in maximal and submaximal efforts,[8] and may or may not have an impact on performance.[25] One-to-one correlations between the magnitude of CF and the degree of muscle work accomplished do not exist.[12] Fatigue can occur with brief maximal voluntary contractions as well as with very low force efforts.[8] Low-force, prolonged isometric contractions (20% MVC), for example, have been shown to induce greater levels of CF than high-force contractions (80% MVC) in both men and women.[21,61] In a study of repetitive submaximal isometric contraction of the knee-extensors, subjects showed reductions in MVC ranging from 35% to 70%, but equal time to exhaustion, and negligible central fatigue.[45] Sustained performance of very low-force contractions (isometric elbow flexion of 5% MVC for 70 min) produced a progressive inability to sustain motor drive and the onset of CF.[62] The time course of fatigue in the quadriceps of long distance runners after a 20 km run showed no decrease in the contractile properties of the muscle, despite the onset of CF (decreased voluntary activation).[25] These findings were attributed to a centrally-mediated pacing strategy.[25]

Why voluntary muscle force also declines with prolonged submaximal contractions is not clear.[21] At low levels of exertion, the sense of PE may amount to little more than perception of body movement in overcoming inertia.[47] Yet, these low levels of exertion may be critical to the continuous need to sculpt descending motor drive through (non-conscious) proprioceptive monitoring of motor performance to ensure accurate levels of motor drive.[37] Moshe Feldenkrais designed a somatic system, Awareness Through Movement, in which persons attend to executing small-range movements at force levels below that of perceived muscular effort. Feldenkrais predicted that low-force actions would promote positive changes in the sensorimotor cortex (that is, in the brain's sensorimotor maps, which Feldenkrais called the "body image").[63] Neuroscientists now recognize these sensorimotor maps as very highly organized, discrete regions of neural assemblies representing the various body parts that control sensations and movements. While little is known about how the brain codes for force,[64] evidence does suggest that brain structure and function are far from static, changing dynamically in response to use, learning, or injury.[65-67] New patterns of synaptic connectivity are formed as a result of learning and experience, altering the size, synaptic organization, and connectivity of sensorimotor maps of muscles and actions.[66] This stimulation induces alterations of cortical representations of body parts, muscles, and actions (topographic maps). Extensive adaptive plasticity is possible in response to motor learning and retraining (rehabilitation).[68] Changes in the brain's topography can result from learning a new skill, improvement of a skill, degradation of a skill, or simply from non-use (prolonged immobility).[69] Neuroscientists refer to this dynamic pattern of brain mapping activity as use-dependent[67] or activity-driven[65] plasticity.

The nature and locus of this plasticity in the sensorimotor cortex and the corticospinal system is dictated by the specifics of motor experience—not only on the characteristics of the stimulation (magnitude, intensity, frequency, duration), but also on aspects of the task.[67,70,71]

To paraphrase Feldenkrais, the sensation of effort is not a measure of the actual work done but an indication of the *organization* of the effort (italics the author's).[72] Feldenkrais believed that attending to the proprioceptive feedback during Awareness Through Movement lessons helps to organize these neural assemblies in ways that promote improvements in function. Proprioceptive feedback arising during these lessons is purported to induce positive changes in the brain's sensorimotor maps and lead to improved skill,[72] although no imaging studies exist to support Feldenkrais' claims.

What do exist are examples of "aberrant" learning hypothesis, or the "repetitive strain model," in which particular conditions of repetitive force lead to fatigue and injury. These include rapid, alternating, repetitive loading of the fingers (as in computer typing) or hand squeezing under conditions of highly focused attention,[73] conditions commonly reported from improper use of a computer or other manual equipment. Implosive loading of the fingers ("clacking" on the computer keyboard) with high cortical demand (paying attention to small detail), poor postural support, and poor environmental conditions (such as an overly cold office) conspire to create repetitive strain injury. With manual use of machinery mechanical forces not only can be repetitive, but also the voluntary effort often is near maximal and invariant. Such repetition purportedly causes brain fatigue and negative changes (degradation) in the cortical maps.[73] The neurons, normally discretely organized in these sensorimotor maps, become blurred and chaotic, resulting in a breakdown in function and consequent physical injury.[73,74]

Impact on Motor Control and Performance

Determining just how much CF impacts the overall fatigue pattern in any physical profile (skill, strength, endurance, power) is difficult.[13,24] While most of the actual force lost during fatigue occurs distal to the motor axon, between 10% and 50% of force has been attributed to inadequate central drive to the muscle.[8,41] Such a wide discrepancy in percentage of force lost appears due not only to differences in research methodology but also to the complex context-dependent nature of CF.[17,42] Although CF alone cannot explain the totality of task failure with strenuous exercise, it unquestionably has serious consequences for neuromuscular control and therefore motor performance.[7,75,76] CF has been shown to impair coordination,[25] postural stability,[35] concentration,[46] and ability to perform both simple (pull ups) and complex (cutting drills) motor skills.[77] In an examination of the effect of fatiguing lower extremity exercise on postural stability, for example, researchers found increased postural sway and instability up to 30 minutes post-fatigue protocol.[35] Postural responses undergo an adaptive process in CF, designed to preserve postural control in conditions of altered neuromuscular function.[38] When balancing in upright stance during center floor dance exercises, for example, this may manifest as a strategy of shifting more weight to the non-dominant leg, which may not be as "skilled" or strong as the dominant one, placing the dancer at risk for injury.

What can be deduced from this physiological data is that the combination of CF *with* peripheral fatigue places the elite athlete at risk of injury, especially during prolonged repetitive, highly implosive exercise (e.g., jumping).[78] Borotikar and coworkers examined the interaction between neuromuscular fatigue and decision-making in dynamic single-leg landings from jumps and the relationship to anterior cruciate ligament injury. Their progressive fatigue protocol first involved having subjects perform repetitive squats and jumps to fatigue lower limb muscles, followed by a random series of light-cued jumps to land on a force plate. The cued jump was a forward unilateral landing with an anticipated or unanticipated cut to the right or left (onto a second force plate). Findings suggested that neuromuscular fatigue induced significant increases in initial landing contact, hip extension, and internal rotation, with more pronounced hip rotations and peak knee abduction angles during unanticipated landings. The substantial altering of hip control during fatigue led to decreased hip flexion and increased hip internal rotation during the shock-absorbing phase of landing. While fatigue-induced landing with the hip relatively more extended may have prevented the lower limb from collapsing (in this experiment), the knee was left to absorb the impact through hazardous joint postures. The researchers speculated that cognitive deterioration resulted from fatigue in decision-making in response to the repetitive, especially unanticipated, cueing.[78] Exposure to repetitive, impulsive joint loading with a change in direction not only affects the lead limb, but also has a crossover effect on the contralateral limb.[77] These biomechanical outcomes have added strength to the speculation that an unanticipated landing in the presence of CF represents a worst-case scenario for risk of non-contact injury to the anterior cruciate ligament of the knee joint.[77]

From Science to Studio Practice?

Outcomes from various studies of CF have led researchers to suggest ongoing training and injury prevention methods to offset the debilitative effects of CF.[49,77] These suggestions, especially from the field of sports and exercise science, have important implications for dance training. Since dancers are also exposed to prolonged, complex (quick changes in effort, direction, or landing conditions), cognitively-demanding movement tasks, addressing the cognitive and psychophysical components implicated in CF would be of vital importance in sustaining endurance, preventing injury, and promoting overall motor control. Although repetition in dance often is counterbalanced by variability of muscular effort, practice hours are lengthy, and protocols similar to those used in exercise science to offset CF, such as self-pacing, higher rest-to-activity ratios, and intervallic training, are non-existent.[5,79]

Mental practice (MP) has been suggested as an effective means of developing central control strategies that quickly transfer to ongoing performance demands.[77] Neuroimaging studies have shown that the cortical substrates underlying imagined practice of a motor task are the same as those in actual physical movement, except for the end execution of the task (i.e., the degree to which the primary motor cortex is activated to initiate efferent pathways to the muscles).[80] MP has been shown to increase the excitability of the cortical areas involved in movement and motor planning[81]; it is also a non-fatiguing approach to motor learning in improving motor skill,[82] speed,[83] and strength.[84] The impact of MP on CF *per se* has yet to be investigated, though MP of motor imagery[85] (Ideokinesis[86]) is a well-substantiated practice in dance for enhancing performance while offsetting fatigue.[87] MP also is often used in dance as a strategy for recovery, recuperation, and neuromuscular re-education, but its actual use in moments of intense, complex, and cortically demanding activities is under-researched. Attempts have been made to integrate mental training with dance practice, such as the program called "Conditioning with Imagery for Dancers,"[88] although again the emphasis in application appears to be on facilitating movement rather than offsetting CF.

How MP can be used to train hard-wired spinal control mechanisms to further combat fatigue in the dance studio remains on the frontier of dance science, awaiting methods of recording how the brain can be paired with actual performance of dance movements. One precedent has been set by the research of Blaser and Hockelmann on the effects of mental rehearsal of choreography and its effects on physiological fatigue in dancers.[6] Dancers were asked to mentally rehearse 120 seconds of choreography with eyes closed, first to the music associated with the choreography and then the dance movements alone. Using electroencephalography, and monitoring heart rate and breathing, the researchers found that an increase in mental concentration was associated with an increase in brain activity of the most utilized body parts, as well as increases in physiological (vegetative) parameters.[6] Their research points to the fact that even mental rehearsal itself is fatiguing, placing demands on cardiac, respiratory, and muscular systems, as well as the neural.[89]

Another more speculative approach to offsetting fatigue is action observation,[90] in which observing motor activities activates the mirror neuron system.[91] If the particular movement is within the observer's repertoire, stronger activation of the mirror neuron system results. Studies comparing the activation of mirror neurons in ballet and Capoiera dancers showed stronger activation when the dancers watched their own genre.[92] The activation patterns were stronger across gender lines as well; steps typifying professional male dancers evoked stronger mirror activation when male dancers watched males than when the steps were performed by females (and vice versa).[93] The mirror neuron system is not merely imitative ("monkey see, monkey do"), but inferential, in that the observer can predict the intention of the mover, implying the potential role of motor programming.[94]

In summary, the skills needed to sustain psychophysical and mental endurance and offset fatigue (both peripheral and central) are inter-related and of crucial importance in dance training. Dancers need to develop a set of cognitive, psychological, and perceptuo-motor tools for detecting the onset of CF, in order to modulate its effects and recover from it. These tools should address various timing intervals of pacing and scheduling comparable to those adopted by sports psychologists in training athletes. For example, at a minimum the dancer should develop and use an ongoing "**body blink**"—a momentary, one-second focused effort at central inhibition—that is used for "refreshing the page," much as we automatically refresh the page on a computer. One-second rests have been shown to improve performance during mentally demanding bimanual hand-tasks.[95] Five-minute rests are routine and substantiated to improve performance in repetitive weightlifting intervals.[96] Twenty minutes is the normal time frame suggested for Ideokinetic constructive rest.[86] A short nap also suffices as a multi-system refresher.[96] One hour is a solid lunch break, ideally without multitasking or attention harnessed to cell phones and other forms of social networking. Finally, 8 hours of sleep needs little defense in the scientific literature in terms of its importance for motor learning[97] and cognitive health.[98]

Author: "body blink" had ref 95, superscript removed; see notes in reference list.

Conclusion

CF remains an elusive mechanism in the overall profile of exercise-induced fatigue. While researchers have shed much light on the mechanisms of CF, its behavior appears largely nonlinear and context-dependent. Future insights can be gained as researchers move away from single factor fatigue paradigms toward theories and practices within the psycho-physiological realm of effort-related decision making in motor skill learning.[42,46] CNS appears to employ different strategies to execute different motor tasks, and every individual task is affected by its own unique variables. Further, CF cannot be studied fully without considering its dependence on peripheral neurological and metabolic phenomena. Research investigating the interaction between fatiguing muscles and central motor command during whole body exercise, while still in its infancy,[1] alludes to the importance of perception, cognition, and emotion in the onset and development of fatigue. Ultimately, many questions still remain as to appropriate training regimens to offset fatigue in demanding, repetitive, prolonged activities such as dance.[7] The future of fatigue research in dance science lies in uncovering fatigue factors that are specific to dance contexts in order to design exercise-specific training protocols. More research utilizing imaging studies in dance-specific contexts is warranted, especially using controlled and standardized methods and protocols as well as multimodal measurement approaches.

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