

Muscular Fatigue

Considerations for Dance

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Abstract

Muscular fatigue can be defined as the failure to maintain an expected power output. It is a multifaceted phenomenon that incorporates metabolic, neural and neuromuscular components, among others. Metabolic causes of fatigue are associated with the ability to maintain energy supply during exercise, the speed at which homeostasis is achieved post-exercise, and the effects of high intensity exercise by-products on the peripheral neuromuscular system. Research has indicated that the central nervous system plays a protective role in preventing catastrophic muscle damage by reducing the intensity and frequency of propagation founded on biofeedback from the muscle cells. The duration and particularly the type of physical activity play a role in the development of muscle fatigue, with impact or weightbearing exercises, such as dance, producing increased symptoms compared to non-impact or non-weightbearing equivalents. The effects of prolonged exercise and the associated increased levels of muscle fatigue that may lead to compromises in neuromuscular propagation need to be considered in dance.

Muscular fatigue occurs when power output or exercise intensity cannot be maintained at a pre-set rate, and if exercise continues, it is at a lower intensity. More technically, Hultman and coworkers described fatigue as the reduction in muscle force production resulting from a decrease in the number of cross-bridges attaching simultaneously in the force generating state.¹ They attributed this phenomenon to a number of different mechanisms, including: alterations in calcium (Ca^{++}) release from the sarcoplasmic reticulum during excitation contraction coupling; lack of ATP supply to the calcium and potassium pumps, as well as to the actin-myosin cross-bridges; and inhibition of cross-bridge cycling by increased levels of dihydrogen phosphate (H_2PO_4). However, there is a lack of universal agreement as to the causes of fatigue; they could be peripheral (muscular metabolism), central (impaired central drive changes in afferent neural signals), or a combination of the two.^{2,3} Muscu-

lar fatigue also depends on the type, duration, and intensity of exercise, and on the individual's muscle fibre profile and level of fitness, in addition to environmental and psychological factors.^{4,5}

Studies have demonstrated that dance is a high intensity intermittent exercise,⁶⁻¹² with dance periods during class and performance lasting from 1 to 6 minutes followed by rest periods of 2 to 9 minutes.^{6,9,10,12-14} Exercise intensity during dance class ranges from 46% to 80% of maximum aerobic capacity,^{6,8,9,13,14} while stage performances include short periods of maximal exercise and periods of passive rest.¹² Classes and rehearsals are usually 90 to 120 minutes long, with daily dance time totalling up to 7 hours.¹⁵ It should also be considered that dance is a high-skill activity, where physical prowess is seen solely as a foundation for the demonstration of complex movement sequences. As the complexity of the skill increases within a sequence, the physiological intensity of that sequence has to decrease.¹⁶ This makes the application of muscle fatigue research, which has generally focussed on relatively low-skill maximal exercise (e.g., running, cycling),¹⁷ difficult in dance. Thus, the current literature offers little information that is directly relevant to dance and muscular fatigue. As the deleterious effects of long-term fatigue include overtraining in active individuals¹⁸ (including dancers¹⁹), this review will explore two

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likely causes of fatigue, i.e., energy production for muscular contraction and the role of the nervous system, making relevant references to dance when possible. Special reference will also be made to fatigue in relation to dance injury.

Energy Systems in Dance and Fatigue

The muscle's ability to convert chemical energy from food into muscular work is directly related to performance in sport and dance alike. However, the efficiency of the body to convert chemical energy to energy for muscular work is only about 25%; the remaining cellular chemical energy appears as heat.²⁰ The cell obtains energy with the help of specialized energy-carrier molecules expressed by adenosine triphosphate (ATP), which is considered to be the body's energy currency. Three phosphate molecules are attached to an adenosine molecule through a series of high-energy bonds. It is the breakdown of one of these bonds to form adenosine diphosphate (ADP) and phosphate (P_i) and the subsequent release of energy that allows muscular contraction to occur.²⁰ As there are limited stores of ATP within cells, approximately 25 mmol of ATP per kilogram of dry muscle,²¹ ATP needs to be continually regenerated during physical activity. The role of the energy systems, therefore, is to maintain this ATP store, and the availability of oxygen at the cellular level determines which pathways are used.

The rate of ATP depletion and the levels of ADP, AMP, and P_i determine the sources from which ATP is regenerated. Specifically, ATP is generated aerobically (slow glycolysis) via the mitochondria, but as the rate of energy demand increases, this system becomes less capable of restoring ATP. Within this environment of increasing levels of ATP breakdown by-products, the anaerobic systems (fast glycolysis and creatine phosphate) are activated to supplement ATP production. However, if these systems cannot maintain the ATP store, the ability of the muscle fibres to maintain their contraction rate is compromised. This results in

reduced force production, decreased velocity of shortening, and slowed relaxation,²² a combination of factors that affects the performance of rapidly repeated movements typical of dance.

It becomes clear from the above that the rate of demand for energy by the muscle cell will determine the dominant energy production system. Hill and Smith,²³ when they examined the different energy systems during a 30-second maximal Wingate test, noted that the aerobic contribution was 16%, the glycolytic 56%, and the ATP-CP 28%. They also found that the glycolytic system peaked within 15 seconds. For the same Wingate test, Boobis²⁴ noted that after 6 seconds the anaerobic energy production was 10.5 mmol ATP·kg⁻¹ dry weight·sec⁻¹, which dropped to 6.3 mmol·kg⁻¹ dry weight·sec⁻¹ after 30 seconds. The glycolytic contribution was estimated at 49% after 6 seconds and 80% after 30 seconds. At 2 minutes maximal exercise, the ratio between aerobic and anaerobic energy production was 50:50.²⁵⁻²⁷ During steady state maximal exercise lasting for more than 4 minutes, the aerobic system largely dominates in the provision of energy. Consequently, the energy systems utilized during periods of physical activity are dependent on several variables: the intensity and duration of the activity itself and the length of rest between activity periods.^{28,29} System utilization also depends on levels of physical fitness; fit dancers reach the fatigue causing anaerobic status significantly later and at the same time demonstrate better dance-related skills than their less fit counterparts.³⁰⁻³²

Dance class has two distinct parts, the warm-up at the barre and the center section. The warm-up is characterized by 5 to 10 minutes of low intensity exercise periods,^{8,14} incorporating exercises such as pliés, port de bras, and rond de jambe, which suggests that the aerobic system is meeting most of the muscular energy demands. The center section of class, however, is typified by periods of intense activity lasting 10 to 40 seconds (petite and grande allegro) interspersed between longer periods

of rest (2 to 5 minutes),^{8,14} during which both anaerobic and aerobic energy systems are involved. Dance performance data suggest that the intensity of performance is often similar to that seen during the center phase of dance class,^{7,9,12} although the duration of exercise is longer (1 to 4 minutes).¹⁰ This is accompanied by increased lactate levels of up to 11 mmol·l⁻¹,⁹ with concomitant decreases in muscle force production that have been linked to fatigue in both athletes and dancers.³³ It must be stressed that the dance data mentioned above were derived from a limited number of studies of ballet and contemporary dance and therefore cannot be construed as a true reflection of the diversity of styles and performances seen in dance generally.

Hultman and colleagues¹ suggested that high-intensity exercise is not limited by the ATP resynthesis capacity of the contracting muscle but by the utilization of available ATP by the contractile mechanism, which has been linked to the decreased release of Ca⁺⁺ from the sarcoplasmic reticulum (SR).³⁴ Furthermore, Westerblad and associates³⁵ hypothesized that it is the release of inorganic phosphate (P_i , from the breakdown of ATP) rather than lactic acid causing intracellular acidosis that is a potential cause of fatigue. The inorganic phosphate enters the myoplasm before moving into the SR, where the P_i combines with Ca⁺⁺ to form calcium phosphate (Ca P_i). This leads to a reduced SR Ca⁺⁺ release, which subsequently impacts actin-myosin cross-bridging and the resulting decline in force production.²² It would take 1 to 2 minutes of maximal fatiguing exercise (e.g., the jump section in dance class) for the precipitation of Ca P_i to occur.²²

Energy Restoration Following Repetitive High Intensity Exercise

The energy required for one bout of high intensity exercise is provided through anaerobic pathways (CP and glycogenolysis).³⁶ When the exercise is comprised of repetitive brief high intensity bouts, the relative contributions to ATP resynthesis need to be

reassessed. Preliminary data suggest that limited periods of recovery (e.g., about 30 seconds) would lead to a reduction of CP muscle content with a greater emphasis on glycogenolysis to anaerobically produce ATP in subsequent bouts.³⁷ This is in line with work by Gaitanos and coworkers,³⁸ who concluded that a 30-second recovery phase is not enough to allow for sufficient replenishment of the CP stores to cope with maximal-intensity short-bout exercise.

Hill proposed that exercise is limited by uncompensated metabolic acidosis within the muscle fibers during short duration maximal exercise.²⁷ Holmyard and colleagues³⁹ also investigated the differences between 30- and 60-second rest periods between multiple sprints. When mean power output was analysed, the 60-second rest period tests produced significantly higher scores than the 30-second rest periods, although there was no difference between the two when peak power was considered. This discrepancy was due to greater CP regeneration stores and to more time for translocation of H⁺ ions out of the muscle cell; a low cell pH will inhibit creatine kinase, thereby impairing CP resynthesis. Other studies^{40,41} have revealed similar results, noting that CP stores are fully recovered after a 2 minute rest period following a high intensity exercise bout, while another set of data indicated that the majority (70%) of CP restoration occurs in 30 seconds, reaching its full capacity within 3 to 5 minutes.⁴² However, Hermansen and Vaage⁴³ reported that muscle's CP stores can take from 5 to 60 minutes to recover fully depending on the amount of depletion and are therefore of little importance during a shorter recovery period. Maughan and associates²⁷ noted that the slower resynthesis of CP in type II muscle fibers than type I, in the initial minutes of recovery, was possibly due to the greater acidosis in these fibers.

Full restoration of glycogen can be a long process, depending on the type of exercise. The rate of glycogen resynthesis after prolonged exercise is much slower than that observed after

high intensity, short duration exercise.^{44,45} The resynthesis of glycogen is thought to be linear for prolonged exercise^{42,46,47} and curvilinear after high-intensity short-duration exercise.^{42,47} However, given that glycogen is an import fuel for the function of vital organs, such as the brain, sharp declines of its relatively limited body depots may trigger central-driven protection mechanisms, perceived as muscular fatigue, which can prevent muscle from using large amounts of this precious fuel by reducing the intensity and frequency of propagation founded on biofeedback from the muscle cells.

The Role of the Nervous System (The Central Governor Theory)

Noakes and coworkers⁴⁸ argue that fatigue during exercise mostly occurs without evidence of the above mentioned metabolic limitations. Indeed, it has been demonstrated that isolated muscle fibers with no CNS control, when stimulated tetanically to pH levels of around 6.8, only result in mechanical failure when temperature is reduced to approximately 12° C; at normal temperatures force output is not affected by pH levels.^{49,50} This suggests that cellular pH is not a significant contributor to fatigue, despite Hill's proposed association between metabolic acidosis and the conscious decision to stop exercising.⁵¹ Noakes and colleagues⁵² reported that despite limited muscular ATP stores, ATP concentrations never dropped below 50% of resting value under any conditions, including ischaemic conditions, even in individuals with reduced capacity to generate ATP aerobically or anaerobically. The investigators suggest that ATP concentrations are as tightly regulated during exercise as they are at rest. A decrease in the muscle's pH may also contribute to central fatigue, as an association between pH and electromyography (EMG) has been shown; pH provides feedback to the central nervous system (CNS), with low pH causing a subsequent alteration in central motor drive.⁵³

The findings described above led to the Central Governor Theory, accord-

ing to which exercise is not limited by peripheral events, but rather is regulated within a neurally calculated safe exertion by the body.⁵⁴ The CNS is possibly protecting the muscle from becoming damaged by reducing the maximal voluntary activation⁵⁵ or preventing hypoglycaemia from glycogen depletion.⁵⁶ Noakes proposes that the CNS is constantly monitoring biochemical changes in the muscle and regulating the muscle's activity accordingly.⁵⁴ However, research by Miller and colleagues^{17,57,58} indicated that central fatigue alone cannot totally explain the MVC decrease, suggesting the involvement of E-C coupling failure, alteration of neuromuscular propagation, and contractile apparatus modification. Nevertheless, studies examining motor unit requirement during exhaustive exercise have shown that, at the point of exhaustion, approximately 30% of the available units are active in a recreational population,^{59,60} and this might rise to 40% to 50% in elite athletes. This corresponds to Gandevia's⁵⁵ conclusion that muscular fatigue in humans is not solely decided in the muscle. Noakes and coworkers⁴⁸ suggest fatigue should be understood as a "sensory perception that results from the complex integration of physiological, biochemical, and other sensory feedback from the periphery and which may or may not be associated with any alteration in muscle force production." Thus, all changes in exercise intensity and the cessation of exercise occur as part of a regulatory strategy that is continuously changing to protect the body from damage⁴⁸ and allows the individual to "calculate" the intensity and duration of exercise that can be achieved within a few minutes of starting, but also alters these variables based on the continuous feedback received by the central governor.

Not surprisingly, the Central Governor Theory is very controversial and has led to heated debate,⁶¹ with other research groups proposing a "peripheral governor"⁶² or that exercise regulation can occur without somatosensory feedback.⁶³ The above commentary shows that muscle fatigue

is a multifaceted phenomenon that has biochemical and neuromuscular components. The research seems to favor CNS playing a protective role of preventing catastrophic muscle damage by reducing the intensity and frequency of propagation based on biofeedback from the muscle cells. The type and duration of physical activity in question plays a role, with impact (e.g., running or jumping) causing increased fatigue even when exercise intensity is standardized with non-impact exercise (e.g., cycling). After 2 or more hours of impact exercise, significant levels of peripheral muscle fatigue and compromised neuromuscular propagation have been detected that would need to be taken into account, for example, during dance class and rehearsals. In particular, strength or power moves, such as jumps and lifts, as well as highly intricate moves including *sissonne* and balancing en *pointe*, may be compromised so that jumps will be lower and leg speed and accuracy diminished.

Fatigue and Dance Injury

It has been repeatedly reported that injury is a fact of dancers' lives. In a period of 12 months, almost 50% of a large sample of professional dancers reported 1 to 6 days away from dance due to a musculoskeletal injury, with the lower back, pelvis, legs, knees, and feet the most frequently injured sites.^{33,54} Although there are no published reports directly related to dance, available data lead us to believe that fatigue, via overwork and burnout,¹⁹ may have contributed to chronic injury patterns in dance. Indeed, it has been found that both dancers and athletes are likely to become overtrained toward the end of their stage performance⁶⁴ and competition⁶⁵ seasons, respectively. It is probable that overtraining and the associated fatigue contributed to these findings as reported in two large scale self-report injury surveys.^{66,67} Allen and associates⁶⁸ noted that reducing the incidence of chronic injury in a professional ballet company led to a slight increase in traumatic injuries. They postulated that this was because

the occurrence of chronic injuries allowed dancers to have a reduced workload and therefore more rest. By reducing this benefit the dancers were becoming more fatigued, and this had a direct effect on limb alignment during activities, such as landing from jumps, causing the observed increase in traumatic injuries.

Fatigue is also associated with a lack of optimal physical fitness,^{69,70} which in turn has been linked to increased injury rates in dance.^{71,72} However, supplemental fitness training may have a significant effect in combating dance injury, as the detrimental effects of fatigue would be less pronounced.⁷³⁻⁷⁶ It should also be noted that fatigue is task dependent, with eccentric and shock-absorbing movements having lasting effects after the cessation of exercise. Long duration low level impact exercise has been shown to cause muscular fatigue that can be explained by muscle damage.⁷⁷

Practical Applications

Dance teachers, artistic directors, and choreographers need to understand aspects of muscle fatigue, especially because dance utilizes both high intensity impact and high skill activities. The dancer's body will try to protect itself from incurring damage by compromising and reducing the intensity of exercise. Evidence suggests that high intensity impact dance classes and rehearsals should be of short duration (less than 60 minutes), with accompanying rest periods after exercise to allow the muscles to recover fully. During class and rehearsal, the work to rest ratio needs to be set so that muscular metabolic recovery is possible. For instance, during center jump sequences a 1:3 work to rest ratio is recommended,¹⁶ as this will allow the anaerobic energy system to recover enough to avoid compromising the subsequent movement repetitions. These elements should also be taken into account during the choreographic process as, without adequate rest in between, a dancer will not be able to finish one high intensity sequence and then attempt another without compromising the move-

ment quality and intensity.⁷⁸ In general, the individual's physical fitness characteristics and how the training of dancers brings about appropriate adaptations of the energy systems within the muscle cells should be of critical concern.¹⁶ Evidence suggests that within the hierarchy of ballet companies certain positions require specific physical abilities to accomplish the roles assign them, and this is manifested in the physical fitness characteristics of these dancers; corps and principal dancers tend to have a more fully developed aerobic system compared to soloists, who in turn have greater anaerobic capacities.⁷⁹ The importance of supplemental fitness training for dancers has been demonstrated by a number of studies^{31,32,80,81} and reviews^{16,33,74,75,82,83} and therefore need not be re-examined here. Finally, by combating fatigue the current injury patterns may be changed in favor of dancers.^{73,84}

References

1. Hultman E, Bergstrom M, Spriet LL, Soderlund K. Energy metabolism and fatigue. In: Taylor AW, Gollnick PD, Green HJ, et al (eds): *Biochemistry of Exercise* (Vol VII). Illinois: Human Kinetics, 1990.
2. Bigland-Ritchie B, Furbush F, Woods J. Fatigue of intermittent submaximal voluntary contractions: central and peripheral factors. *J Appl Physiol*. 1986 Aug;61(2):421-9.
3. Koutedakis Y, Frischknecht R, Vrbová G, et al. Maximal voluntary quadriceps strength patterns in olympic overtrained athletes. *Med Sci Sports Exerc*. 1995 Apr;27(4):566-72.
4. Fitts RH, Metzger JM. Mechanisms of muscular fatigue. In: Poortmans JR (ed): *Principles of Exercise Biochemistry* (2nd ed). Basel: Medicine Sport and Science, 1993, pp. 248-268.
5. Paschalis V, Koutedakis Y, Baltzopoulos V, et al. The effects of muscle damage on running economy in healthy males. *Int J Sports Med*. 2005 Dec;26(10):827-31.
6. Angioi M, Twitchett E, Metsios G, et al. Fitness level in contemporary dance: a systematic review. *Int J Sports Med*. 2009 Jul;30:475-84.
7. Cohen JL, Segal KR, McArdle

- WD. Heart rate response to ballet stage performance. *Phys Sportsmed*. 1982;10(11):120-33.
8. Cohen JL, Segal KR, Witriol I, McArdle WD. Cardiorespiratory responses to ballet exercise and VO₂max of elite ballet dancers. *Med Sci Sports Exerc*. 1982;14(3):212-7.
9. Schantz P, Astrand P-O. Physiological characteristics of classical ballet. *Med Sci Sports Exerc*. 1984 Oct;16(5):472-6.
10. Twitchett E, Angioi M, Koutedakis Y, Wyon M. Video analysis of classical ballet performance. *J Dance Med Sci*. 2009;13(4):124-8.
11. Twitchett E, Koutedakis Y, Wyon M. Physical fitness and classical ballet performance: a literature review. *J Strength Cond Res*. 2009 Dec;23(9):2732-40.
12. Wyon M, Head A, Sharp NCC, et al. Oxygen uptake during modern dance class, rehearsal, and performance. *J Strength Cond Res*. 2004 Aug;18(3):646-9.
13. Dahlstrom M, Inasio J, Jansson E, Kaijser L. Physical fitness and physical effort in dancers: a comparison of four major dance styles. *Impulse*. 1996;4:193-209.
14. Wyon M, Head A, Sharp C, Redding E. The cardiorespiratory responses to modern dance classes: differences between university, graduate, and professional classes. *J Dance Med Sci*. 2002;6(2):41-5.
15. Twitchett E, Angioi M, Koutedakis Y, Wyon M. The demands of a working day among female professional ballet dancers. *J Dance Med Sci*. 2010;14(4):127-32.
16. Wyon M. Cardiorespiratory training for dancers. *J Dance Med Sci*. 2005;9(1):7-12.
17. Millet G, Lepers R, Maffiuletti N, et al. Alteration of neuromuscular function after an ultra-marathon. *J Appl Physiol*. 2002 Feb;92:486-92.
18. Parry-Billings M, Budgett R, Koutedakis Y, et al. Plasma amino acid concentrations in the overtraining syndrome: possible effects on the immune system. *Med Sci Sports Exerc*. 1992 Dec;24:1353-8.
19. Koutedakis Y. Burnout in dance: the physiological viewpoint. *J Dance Med Sci*. 2000;4(4):122-7.
20. Koutedakis Y, Sharp NCC. *The Fit and Healthy Dancer*. Chichester: John Wiley and Sons, 1999.
21. Hultman E, Sjoholm H. Substrate availability. In: K, HG, Vogel J, Poortman J (eds): *Biochemistry of Exercise*. Champaign, Ill: Human Kinetics, 1983, pp. 63-75.
22. Allen D, Westerblad H. Role of phosphate and calcium stores in muscle fatigue. *J Physiol*. 2001 Nov 1;536(Pt 3):657-65.
23. Hill DW, Smith JC. Calculation of aerobic contribution during high intensity exercise. *Res Q Exerc Sport*. 1991 Mar;63(1):85-8.
24. Boobis LH. Metabolic aspects of fatigue during sprinting. In: Macleod D, Maughan R, Nimmo M, et al (eds): *Exercise: Benefits, Limits and Adaptations*. London: E&FN Spon, 1987, pp. 116-143.
25. Keul J. Limiting factors of physical performance. In: Keul J, Ambrosoli G (eds): *International Symposium on the Limiting Factors of Physical Performance*. Gravenbruch: Thieme, 1971, pp. 113-125.
26. Mader A, Heck H, Hollmann W. Evaluation of lactic acid anaerobic energy contribution by determination of postexercise lactic acid concentration of ear capillary blood in middle-distance runners and swimmers. *Exerc Physiol*. 1976;4:187-199.
27. Maughan R, Gleeson M, Greenhaff PL. *Biochemistry of Exercise and Training*. Oxford: Oxford University Press, 1997.
28. Bangsbo J. Physiological factors associated with efficiency in high intensity exercise. *Sports Med*. 1996 Nov;22(5):299-305.
29. Bompa T. *Theory and Methodology of Training: The Key to Athletic Success* (3rd ed). Dubuque, Iowa: Kendall/Hunt Publishing, 1994.
30. Angioi M, Metsios GS, Twitchett E, et al. Association between selected physical fitness parameters and esthetic competence in contemporary dancers. *J Dance Med Sci*. 2009;13(4):115-23.
31. Koutedakis Y, Hukam H, Metsios G, et al. The effects of three months of aerobic and strength training on selected performance- and fitness-related parameters in modern dance students. *J Strength Cond Res*. 2007 Aug;21(3):808-12.
32. Twitchett E, Angioi M, Koutedakis Y, Wyon M. Do increases in selected fitness parameters affect the aesthetic aspects of classical ballet performance. *Med Probl Perform Art*. 2011 Mar;26(1):35-8.
33. Koutedakis Y, Jamurtas A. The dancer as a performing athlete: physiological considerations. *Sports Med*. 2004;34(10):651-61.
34. Bergstroem M, Hultman E. Energy cost and fatigue during intermittent electrical stimulation of human skeletal muscle. *J Appl Physiol*. 1988 Oct;65(4):1500-5.
35. Westerblad H, Allen D, Lannergren J. Muscle Fatigue: lactic acid or inorganic phosphate the major cause? *News Physiol Sci*. 2002 Feb;17:17-21.
36. Jacobs I, Tesch PA, Bar-Or O, et al. Lactate in human skeletal muscle after 10 and 30 s of supramaximal exercise. *J Appl Physiol*. 1983 Aug;55(2):365-7.
37. Wootton SA, Williams C. Influences of recovery duration on repeated maximal sprints. In: Knuttgen HG (ed): *International Symposium on the Biochemistry of Exercise; 1983*. Boston: Human Kinetics Publishers, 1983, pp. 269-273.
38. Gaitanos G, Williams C, Boobis L, Brooks S. Human muscle metabolism during intermittent maximal exercise. *J Appl Physiol*. 1993 Aug;75(2):712-9.
39. Holmyard DJ, Cheetham ME, Lakomy HK, Williams C. Effects of recovery duration on performance during multiple treadmill sprints. In: Reilly T, Lees A, Davids K, Murphy WJ (eds): *Science and Football*. London: E&FN Spon, 1988, pp. 134-142.
40. Bogdanis GC, Nevill ME, Boobis LH, et al. Recovery of power output and muscle metabolites following 30-s of maximal sprint cycling in man. *J Physiol*. 1995 Jan 15;482 (Pt 2):467-80.
41. Tabata I, Irisawa K, Kouzaki M, et al. Metabolic profile of high intensity intermittent exercises. *Med Sci Sports Exerc*. 1997 Mar;29(3):390-5.
42. Pascoe DD, Gladden LB. Muscle glycogen resynthesis after short term, high intensity exercise and resistance exercise. *Sports Med*. 1996 Feb;21(2):98-118.
43. Hermansen L, Vaage O. Lactate disappearance and glycogen synthesis in human muscle after maximal exercise. *Am J Physiol*. 1977 Nov;233(5):E422-9.
44. Bangsbo J, Johansen L, Saltin B. The effects of severe exercise on fatigue

- and anaerobic energy production during subsequent intense exercise: the importance of active recovery. In: Reilly T, Clarys J, Stibbe A (eds): *Science and Football II*. London: E&FN Spon, 1993, pp. 107-113.
45. Blom P, Vollestad NK, Costill DL. Factors affecting changes in muscle glycogen concentration during and after prolonged exercise. *Acta Physiol Scand*. 1986;125(Suppl):67-74.
 46. Ivy JL. Muscle glycogen synthesis before and after exercise. *Sports Med*. 1991 Jan;11(1):6-19.
 47. Price TB, Rothman DL, Taylor R, et al. Human muscle glycogen resynthesis after exercise: Insulin-dependent and -independent phases. *J Appl Physiol*. 1994 Jan;76(1):104-11.
 48. Noakes T, St Clair Gibson A, Lambert E. From catastrophe to complexity: A novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. *Br J Sports Med*. 2005 Feb;39(2):120-4.
 49. Westerblad H, Bruton J, Lannergren J. The effect of intracellular pH on contractile function of intact single fibres of mouse muscle declines with increasing temperature. *J Physiol*. 1997 Apr 1;500 (Pt 1):193-204.
 50. Wiseman R, TW B, Chase P. Effect of intracellular pH on force development depends on temperature in intact skeletal muscle from mouse. *Am J Physiol*. 1996 Sep;271(3 Pt 1):C878-86.
 51. Sgherza A, Axen K, Fain R, et al. Effect of naloxone on perceived exertion and exercise capacity during maximal cycle ergometry. *J Appl Physiol*. 2002 Dec;93(6):2023-8.
 52. Noakes T, St Clair Gibson A. Logical limitations to the "Catastrophe" models of fatigue during exercise in humans. *Br J Sports Med*. 2004 Oct;38(5):648-9.
 53. Kent-Braun J. Central and peripheral contributions to muscle fatigue in humans during sustained maximal effort. *Eur J Appl Occup Physiol*. 1999 Jun;80(1):57-63.
 54. Koutedakis Y, Pacy PJ, Carson RJ, Dick F. Health and fitness in professional dancers. *Med Probl Perform Art*. 1997;12(1):23-7.
 55. Gandevia S. Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev*. 2001 Oct;81(4):1725-89.
 56. Noakes T. Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. *Scand J Med Sci Sports*. 2000 Jun;10(3):123-45.
 57. Lepers R, Maffiuletti N, Rochette L, et al. Neuromuscular fatigue during a long-duration cycling exercise. *J Appl Physiol*. 2002 Apr;92(4):1487-93.
 58. Millet G, Martin V, Lattier G, Balalay Y. Mechanisms contributing to knee extensors strength loss after prolonged running exercise. *J Appl Physiol*. 2003 Jan;94(1):193-8.
 59. Kay D, Marino F, Cannon J, et al. Evidence for neuromuscular fatigue during high-intensity cycling in warm, humid conditions. *Eur J Appl Physiol*. 2001 Jan-Feb;84(1-2):115-21.
 60. St Clair Gibson A, Schabert E, Noakes T. Reduced neuromuscular activity and force generation during prolonged cycling. *Am J Physiol Regul Integr Comp Physiol*. 2001 Jul;281(1):R187-96.
 61. Marcora S. Counterpoint: Afferent feedback from fatigued locomotor muscles is not an important determinant of endurance exercise performance. *J Appl Physiol*. 2010 Feb;108(2):454-6; discussion 456-7.
 62. MacIntosh B, Shahi M. A peripheral governor regulates muscle contraction. *Appl Physiol Nutr Metab*. 2011 Feb;36(1):1-11.
 63. Laffargue G, Paillard J, Lamarre Y, Sirigu A. Production and perception of grip force without proprioception: is there a sense of effort in deafferented subjects. *Eur J Neurosci*. 2003 Jun;17(12):2741-9.
 64. Koutedakis Y, Myszkewycz L, Soulas D, et al. The effects of rest and subsequent training on selected physiological parameters in professional female classical dancers. *Int J Sports Med*. 1999 Aug;20(6):379-83.
 65. Koutedakis Y, Sharp NC. Seasonal variations of injury and overtraining in elite athletes. *Clin J Sport Med*. 1998 Jan;8(1):18-21.
 66. Brinson P, Dick F. *Fit to Dance?* London: Calouste Gulbenkian Foundation 1996.
 67. Laws H. *Fit to Dance 2: Report of the Second National Inquiry into Dancers' Health and Injury in the UK*. London: Newgate Press, 2005.
 68. Allen N, Nevill A, Brooks J, Wyon M. The impact of a performance profiling system using a three-year prospective injury study. In: Solomon R, Solomon J (eds): *Proceedings of the 20th Annual Meeting of the International Association for Dance Medicine and Science*. Birmingham, UK: University of Wolverhampton, 2010, pp. 9-10.
 69. Angioi M, Metsios GS, Koutedakis Y, Wyon MA. Fitness in contemporary dance: a systematic review. *Int J Sports Med*. 2009 Jul;30(7):475-84.
 70. Twitchett EA, Koutedakis Y, Wyon MA. Physiological fitness and professional classical ballet performance: a brief review. *J Strength Cond Res*. 2009 Dec;23(9):2732-40.
 71. Koutedakis Y, Dick F, Pacy PJ. Aspects of health and fitness in professional dancers. *Med Probl Perform Art*. 1997;12(1):23-7.
 72. Koutedakis Y, Frischknecht R, Murthy M. Knee flexion to extension peak torque ratios and low-back injuries in highly active individuals. *Int J Sports Med*. 1997 May;18(4):290-5.
 73. Angioi M, Metsios G, Koutedakis Y, et al. Physical fitness and severity of injuries in contemporary dance. *Med Probl Perform Art*. 2009;24(1):26-9.
 74. Koutedakis Y, Clarke F, Wyon M, et al. Muscular strength: applications for dancers. *Med Probl Perform Art*. 2009;24(4):157-65.
 75. Koutedakis Y, Stavropoulos-Kalinooglou A, Metsios G. The significance of muscular strength in dance. *J Dance Med Sci*. 2005;9(1):29-34.
 76. Twitchett E, Brodrick A, Nevill AM, et al. Does physical fitness affect injury occurrence and time loss due to injury in elite vocational ballet students? *J Dance Med Sci*. 2010;14(1):26-31.
 77. Takekura H, Fujinami N, Nishizawa T, et al. Eccentric exercise-induced morphological changes in the membrane systems involved in excitation-contraction coupling in rat skeletal muscle. *J Physiol*. 2001 Jun 1;533(Pt 2):571-83.
 78. Bangsbo J, Saltin B. Recovery of muscle from exercise - its importance for subsequent performance. In: Macleod DAD, Maughan RJ, Williams C, Madeley C, Sharp J, Nutton R (eds): *Intermittent High Intensity Exercise: Preparation, Stresses and Damage Limitations*. London: E&FN SPON, 1993, pp. 49-69.
 79. Wyon M, Deighan M, Nevill A, et al. The cardiorespiratory, anthropometric and performance characteristics

- of an international/national touring ballet company. *J Strength Cond Res*. 2007 May;21(2):389-93.
80. Angioi M, Metsios G, Twitchett E, et al. Effects of supplemental training on fitness and aesthetic competence parameters in contemporary dance: a randomised controlled trial. *Med Probl Perform Art*. 2012 Mar;27(1):3-8.
81. Brown A, Wells T, Schade M, et al. The effects of plyometric training versus traditional weight training on strength, power and aesthetic jumping ability in female collegiate dancers. *J Dance Med Sci*. 2007;11(2):38-44.
82. Koutedakis Y, Cross V, Sharp NCC. Strength training in male ballet dancers. *Impulse*. 1996;4(3):210-9.
83. Deighan M. Flexibility in dance. *J Dance Med Sci*. 2005;9(1):13-7.
84. Twitchett E, Brodrick A, Nevill A, et al. Does physical fitness affect injury occurrence and time-loss through injury in classical ballet dancers? *J Dance Med Sci*. 2010;14(1):26-31.

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