

INTERNATIONAL JOURNAL OF RESEARCHES IN BIOSCIENCES, AGRICULTURE & TECHNOLOGY © VISHWASHANTI MULTIPURPOSE SOCIETY (Global Peace Multipurpose Society) R. No. MH-659/13(N)

www.vmsindia.org

STUDY OF SPORTS SCIENCE IN PHYSICAL EDUCATION

Gaidhane M.K.

Dept. of Chemistry Shri Lemdeo Patil Mahavidyalaya, Mandhal, Kuhi, Nagpur (M.S.) India Email: drkgpatil@gmail.com

Abstract: Sports science (also sport science) is a discipline that studies how the healthy human body works during exercise, and how sport and physical activity promote health from cellular to whole body perspectives. The study of sports science traditionally incorporates areas of physiology (exercise physiology), psychology (sport psychology), anatomy, biomechanics, biochemistry and biokinetics. Sports scientists and performance consultants are growing in demand and employment numbers, with the ever-increasing focus within the sporting world on achieving the best results possible. Through the study of science and sport, researchers have developed a greater understanding on how the human body reacts to exercise, training, different environments and many other stimuli.

Introduction:

Research interest into the effects of dietary nitrate on their responses to exercise has increased exponentially since the seminal works of Larsen and colleagues. Recent studies have demonstrated that dietary supplementation nitrate can improve tolerance to and performance of shortmoderate-intensity duration, aerobic exercise. In addition, there is also compelling evidence that dietary nitrate supplementation can improve repeated high-intensity and supra maximal exercise performance. However, while these effects on acute bouts of exercise have been widely investigated, it is less clear how nitrate supplementation may affect chronic exercise training, with only one study to date investigating the supplement in this context [1]

Energy Expenditure:

Humans have a high capacity to expend energy for many hours during sustained exertion. For example, one individual cycling at a speed of 26.4 km/h (16.4 mph) through 8,204 km (5,098 mi) over 50 consecutive days expended a total of 1,145 MJ (273,850 kcal; 273,850 dieter calories) with an average power output of 182.5 W. [2]

Skeletal muscle burns 90 mg (0.5 mmol) of glucose each minute during continuous activity (such as when repetitively extending the human knee), [3] generating \approx 24 W of mechanical energy, and since muscle energy conversion is only 22–26% efficient, [4] \approx 76 W of heat energy. Resting skeletal muscle has a basal metabolic rate (resting energy consumption) of 0.63 W/kg [5] making a 160 fold difference between the energy consumption of inactive and active muscles. For short duration muscular exertion, energy expenditure can be far greater: an adult human male when jumping up from a squat can mechanically generate 314 W/kg. Such rapid movement can generate twice this amount in nonhuman animals such as bonobos, [6] and in some small lizards. [7]

This energy expenditure is very large compared to the basal resting metabolic rate of the adult human body. This rate varies somewhat with size, gender and age but is typically between 45 W and 85 W. [8] [9] Total energy expenditure (TEE) due to muscular expended energy is much higher and depends upon the average level of physical work and exercise done during a day. [10] Thus exercise, particularly if sustained for very long periods, dominates the energy metabolism of the body. Physical activity energy expenditure correlates strongly with the gender, age, weight, heart rate, and VO2 max of an individual, during physical activity. [11]

Rapid Energy Sources:

Energy needed to perform short lasting, high intensity bursts of activity is derived from anaerobic metabolism within the cytosol of muscle cells, as opposed to aerobic respiration which utilizes oxygen, is sustainable, and occurs in the mitochondria. The quick energy sources consist of the phosphocreatine (PCr) system, fast glycolysis, and adenylate kinase. All of these systems re-synthesize adenosine triphosphate (ATP), which is the universal energy source in all cells. The most rapid source, but the most readily depleted of the above sources is the PCr system which utilizes the enzyme creatine kinase. This enzyme catalyzes a reaction that combines phosphocreatine and adenosine diphosphate (ADP) into ATP and creatine. This resource is short lasting because oxygen is required for the resynthesis of phosphocreatine via mitochondrial creatine kinase. Therefore. under anaerobic conditions, this substrate is finite and only lasts between approximately 10 to 30 seconds of high intensity work. Fast glycolysis, however, can function for approximately 2 minutes prior to fatigue, and predominately uses intracellular glycogen as a substrate. Glycogen is broken down rapidly via glycogen phosphorylase into individual glucose units during intense exercise. Glucose is then oxidized to pyruvate and under anaerobic condition is reduced to lactic acid. This reaction oxidizes NADH to NAD, thereby releasing a hydrogen ion, promoting acidosis. For this reason, fast glycolysis can not be sustained for long periods of time. Lastly, adenylate kinase catalyzes a reaction by which 2 ADP are combined to form ATP and adenosine monophosphate (AMP). This reaction takes place during low energy situations such as extreme exercise or conditions of hypoxia, but is not a significant source of energy. The creation of AMP resulting from this reaction stimulates AMP-activated protein kinase (AMP kinase) which is the energy sensor of the cell. After sensing low energy conditions, AMP kinase stimulates various other intracellular enzymes geared towards increasing energy supply and decreasing all anabolic, or energy requiring, cell functions.

Plasma Glucose:

Plasma glucose is said to be maintained when there is an equal rate of glucose appearance (entry into the blood) and glucose disposal (removal from the blood). In the healthy individual, the rates of appearance and disposal are essentially equal during exercise of moderate intensity and duration; however, prolonged exercise or sufficiently intense exercise can result in

an imbalance leaning towards a higher rate of disposal than appearance, at which point glucose levels fall producing the onset of fatigue. Rate of glucose appearance is dictated by the amount of glucose being absorbed at the gut as well as liver (hepatic) glucose glucose output. Although absorption from the gut is not typically a source of glucose appearance during exercise, the liver is capable of catabolizing stored glycogen (glycogenolysis) as well as synthesizing new glucose from specific reduced carbon molecules (glycerol. pyruvate, and lactate) in a process called gluconeogenesis. The ability of the liver to release glucose into the blood from glycogenolysis is unique, since skeletal muscle, the other major glycogen reservoir, is incapable of doing so. Unlike skeletal muscle, liver cells contain the enzyme glycogen phosphatase, which removes a phosphate group from glucose-6-P to release free glucose. In order for glucose to exit a cell membrane, the removal of this phosphate group is essential. Although gluconeogenesis is an important component of hepatic glucose output, it alone cannot sustain exercise. For this reason, when glycogen stores are depleted during exercise, glucose levels fall and fatigue sets in. Glucose disposal, the other side of the equation, is controlled by uptake of glucose at the working skeletal muscles. During exercise. despite decreased insulin concentrations, muscle increases GLUT4 translocation of and glucose uptake. The mechanism for increased GLUT4 translocation is an area of ongoing research.

Glucose Control:

As mentioned above, insulin secretion is reduced during exercise, and does not play a major role in maintaining normal blood glucose concentration during exercise, but its counter-regulatory hormones appear in increasing concentrations. Principle among these are glucagon, epinephrine, and growth hormone. All of these hormones stimulate liver (hepatic) glucose output, among other functions. For instance, both epinephrine and growth hormone also stimulate adipocyte lipase, which increases nonesterified fatty acid (NEFA) release. By oxidizing fatty acids, this spares glucose

utilization and helps to maintain blood sugar level during exercise.

Exercise For Diabetes :

Exercise is a particularly potent tool for glucose control in those who have diabetes mellitus. In a situation of elevated blood glucose (hyperglycemia), moderate exercise can induce greater glucose disposal than appearance, thereby decreasing total plasma glucose concentrations. As stated above, the mechanism for this glucose disposal is independent of insulin, which makes it particularly well-suited for people with diabetes. In addition, there appears to be an increase in sensitivity to insulin for approximately 12-24 hours post-exercise. This is particularly useful for those who have type II diabetes and are producing sufficient insulin but demonstrate peripheral resistance to insulin signaling. However, during extreme hyperglycemic episodes, people with diabetes should avoid exercise due to potential complications associated with ketoacidosis. Exercise could exacerbate ketoacidosis by increasing ketone synthesis in response to increased circulating NEFA's.

Type II diabetes is also intricately linked to obesity, and there may be a connection between type II diabetes and how fat is stored within pancreatic, muscle, and liver cells. Likely due to this connection, weight loss from both exercise and diet tends to increase insulin sensitivity in the majority of people. In some people, this effect can be particularly potent and can result in normal glucose control. Although nobody is technically cured of diabetes, individuals can live normal lives without the fear of diabetic complications; however, regain of weight would assuredly result in diabetes signs and symptoms.

Oxygen :

Vigorous physical activity (such as exercise or hard labor) increases the body's demand for oxygen. The first-line physiologic response to this demand is an increase in heart rate, breathing rate, and depth of breathing.

Oxygen consumption (VO2) during exercise is best described by the Fick Equation: VO2=Q x (a-vO2diff), which states that the amount of oxygen consumed is equal to cardiac output (Q) multiplied by the difference between arterial and venous oxygen concentrations. More simply put, oxygen consumption is dictated by the quantity of blood distributed by the heart as well as the working muscle's ability to take up the oxygen within that blood; however, this is a bit of an oversimplification. Although cardiac output is thought to be the limiting factor of this relationship in healthy individuals, it is not the only determinant of VO2 max. That is, factors such as the ability of the lung to oxygenate the blood must also be considered. Various pathologies and anomalies cause conditions such as diffusion limitation. ventilation/perfusion mismatch, and pulmonary shunts that can limit oxygenation of the blood and therefore oxygen distribution. In addition, the oxygen carrying capacity of the blood is also an important determinant of the equation. Oxygen carrying capacity is often the target of exercise (ergogenic aids) aids used in endurance sports to increase the volume percentage of red blood cells (hematocrit), such as through blood doping or the use of erythropoietin (EPO). Furthermore, peripheral oxygen uptake is reliant on a rerouting of blood flow from relatively inactive viscera to the working skeletal muscles, and within the skeletal muscle, capillary to muscle fiber ratio influences oxygen extraction.

Dehydration:

Dehydration refers both to hypohydration (dehydration induced prior to exercise) and exercise-induced dehydration to (dehydration that develops during exercise). The latter reduces aerobic endurance performance and results in increased body temperature, heart rate, perceived exertion, and possibly increased reliance on carbohydrate as a fuel source. Although the negative effects of exercise-induced dehydration on exercise performance were clearly demonstrated in the 1940s, athletes continued to believe for years thereafter that fluid intake was not beneficial. More recently, negative effects on performance have been demonstrated with modest (<2%) dehvdration. and these effects are exacerbated when the exercise is performed

in a hot environment. The effects of hypohydration may vary, depending on whether it is induced through diuretics or sauna exposure, which substantially reduce plasma volume, or prior exercise, which has much less impact on plasma volume. Hypohydration reduces aerobic endurance, but its effects on muscle strength and endurance are not consistent and require further study.[12] Intense prolonged exercise produces metabolic waste heat, and this is removed by sweat-based thermoregulation. A male marathon runner loses each hour around 0.83 L in cool weather and 1.2 L in warm (losses in females are about 68 to 73% lower).[13] People doing heavy exercise may lose two and half times as much fluid in sweat as profound urine.[14] This can have physiological effects. Cycling for 2 hours in the heat (35 °C) with minimal fluid intake causes body mass decline by 3 to 5%, blood volume likewise by 3 to 6%, body temperature to rise constantly, and in comparison with proper fluid intake, higher heart rates, lower stroke volumes and cardiac outputs, reduced skin blood flow, and higher systemic vascular resistance. These effects are largely eliminated by replacing 50 to 80% of the fluid lost in sweat.[13][15]

Other factors:

The exercise fatigue has also been suggested to be effected by:

- Brain hyperthermia[16]
- Glycogen depletion in brain cells[17]
- Reactive oxygen species impairing skeletal muscle function[18]
- Reduced level of glutamate secondary to uptake of ammonia in the brain
- Fatigue in diaphragm and abdominal respiratory muscles limiting breathing[19]
- Impaired oxygen supply to muscles[20]
- Ammonia effects upon the brain
- Serotonin pathways in the brain[21]
- Exercise Induced Asthma (EIA) narrowing of the airways in the lungs that is triggered by strenuous exercise. It causes shortness of breath, wheezing, coughing and other symptoms during or after exercise. The preferred term for this condition is exercise-induced

bronchoconstriction (brong-koh-kun-STRIK-shun). This term is more accurate because the exercise induces narrowing of airways (bronchoconstriction) but is not the root cause of asthma. Among people with asthma, exercise is likely just one of several factors that can induce breathing difficulties.For most people with exerciseinduced bronchoconstriction, treatment with common asthma medications and preventive measures enable them to exercise and remain active.[22]

Exercise-Induced Muscle Pain:

Physical exercise may cause pain both as an immediate effect that may result from stimulation of free nerve endings by low pH, as well as a delayed onset muscle soreness. The delayed soreness is fundamentally the result of ruptures within the muscle, although apparently not involving the rupture of whole muscle fibers.[23]

Muscle pain can range from a mild soreness to a debilitating injury depending on intensity of exercise, level of training, and other factors.[24]

Education In Exercise Physiology:

Accreditation programs exist with professional bodies in most developed countries, ensuring the quality and consistency of education. In Canada, one may obtain the professional certification title - Certified Exercise Physiologist for those working with clients (both clinical and non clinical) in the health and fitness industry. An exercise physiologist's area of study may include but is not limited to biochemistry, bioenergetics, cardiopulmonary function, hematology, biomechanics, skeletal muscle physiology, neuroendocrine function, and central and peripheral nervous system function. Furthermore, exercise physiologists range from basic scientists, to clinical researchers, to clinicians, to sports trainers.

Colleges and universities offer exercise physiology as a program of study on various different levels, including undergraduate, graduate, and doctoral programs. The basis of Exercise Physiology as a major is to prepare students for a career in field of health sciences. A program that focuses on the scientific study of the physiological processes involved in physical or motor including sensorimotor activity, interactions, response mechanisms, and the effects of injury, disease, and disability. Includes instruction in muscular and skeletal anatomy; molecular and cellular basis of muscle contraction; fuel utilization; neurophysiology of motor mechanics; systemic physiological responses (respiration, blood flow, endocrine secretions, and others); fatigue and exhaustion; muscle and body training; of specific exercises physiology and activities; physiology of injury; and the effects of disabilities and disease. Careers available with a degree in Exercise Physiology can include: non-clinical, clientbased work; strength and conditioning specialists; cardiopulmonary treatment; and clinical-based research.[25]

In order to gauge the multiple areas of study, students are taught processes in which to follow on a client-based level. Practical and lecture teachings are instructed in the classroom and in a laboratory setting. These include:

• Health and risk assessment: In order to safely work with a client on the job, you must first be able to know the benefits and risks associated with physical activity. Examples of this include knowing specific injuries the body can experience during exercise, how to properly screen a client before their training begins, and what factors to look for that may inhibit their performance.

• Exercise testing: Coordinating exercise tests in order to measure body cardiorespiratory compositions. fitness. muscular strength/endurance, and flexibility. Functional tests are also used in order to gain understanding on a more specific part of the body. Once the information is gathered about a client, exercise physiologists must also be able to interpret the test data and decide what health-related outcomes have been discovered.

• Exercise prescription: Forming training programs that best meet an individuals health and fitness goals. Must be able to take into account different types of exercises, the reasons/goal for a clients workout, and pre-screened assessments. Knowing how to prescribe exercises for special considerations and populations is also required. These may include age differences, pregnancy, joint diseases, obesity, pulmonary disease, etc.[26]

Curriculum:

The curriculum for exercise physiology includes biology, chemistry, and applied sciences. The purpose of the classes selected for this major is to have a proficient understanding of human anatomy, human and exercise physiology. physiology, Includes instruction in muscular and skeletal anatomy; molecular and cellular basis of muscle contraction; fuel utilization; neurophysiology of motor mechanics; systemic physiological responses (respiration, blood flow, endocrine secretions, and others); fatigue and exhaustion; muscle and body training; physiology of specific exercises and activities; physiology of injury; and the effects of disabilities and disease. Not only is a full class schedule needed to complete a degree in Exercise Physiology, but a minimum amount of practicum experience required and internships is are recommended.[27]

References:

1) David J. Muggeridgea, Nicholas Sculthorpea, Philip E. Jamesb, Chris Eastona, Journal of Science and Medicine in Sport. 20 (2017) 92–97.

2) Gianetti, G; Burton, L; Donovan, R; G; Pescatello, LS (2008). Allen, "Physiologic and psychological responses of an athlete cycling 100+ miles daily for 50 consecutive days". Current Sports Medicine Reports. 7 (6): 343–7. This individual while exceptional was not physiologically extraordinary since he was described as "subelite" due to his not being "able to adjust power output to regulate.

3) energy expenditure as occurs with elite athletes during ultra-cycling events" page 347.

4) **Richter, EA; Kiens, B; Saltin, B; Christensen, NJ; Savard, G (1988).** "Skeletal muscle glucose uptake during dynamic exercise in humans: Role of muscle mass". The American journal of physiology. 254 (5 Pt 1): E555–61.PMID 3284382.

5) **Bangsbo, J (1996).** "Physiological factors associated with efficiency in high intensity exercise". Sports medicine (Auckland, N.Z.). 22 (5): 299–305. doi:10.2165/00007256-199622050-00003. PMID 8923647.

6) **Elia, M. (1992)** "Energy expenditure in the whole body". Energy metabolism. Tissue determinants and cellular corollaries. 61–79 Raven Press New York. ISBN 978-0-88167-871-0

7) Scholz, MN; d'Août, K; Bobbert, MF; Aerts, P (2006). "Vertical jumping performance of bonobo (Pan paniscus) suggests superior muscle properties". Proceedings. Biological sciences / the Royal Society. 273 (1598): 2177–84. doi:10.1098/rspb.2006.3568. PMC 1635523 . PMID 16901837.

8) Curtin, NA; Woledge, RC; Aerts, P (2005). "Muscle directly meets the vast power demands in agile lizards". Proceedings. Biological sciences / the Royal Society. 272(1563): 581-4. doi:10.1098/rspb.2004.2982. PMC 1564073 .PMID 15817432. http://journals. royalsociety.org/content/gdgkj59wydr0vca7 /fulltext.pdf.

9) Henry, CJ (2005). "Basal metabolic rate studies in humans: Measurement and development of new equations". Public health nutrition. 8 (7A): 1133-52. doi:10.1079/phn2005801. PMID 16277825. 10) Henry 2005 provides BMR formula various ages given body weight: those for BMR aged 18-30 in MJ/day (where mass is body weight in kg) are: male BMR = 0.0669 mass + 2.28; females BMR = 0.0546 mass + 2.33; 1 MJ per day = 11.6 W. The data providing these formula hide a high variance: for men weighing 70 kg, measured BMR is between 50 and 110 W, and women weighing 60 kg, between 40 W and 90 W.

11) Torun, B (2005). "Energy requirements of children and adolescents". Public health nutrition. 8 (7A): 968–93. doi:10.1079/phn2005791. PMID 16277815.
12) Keytel, L.R. (March 2005). "Prediction of energy expenditure from heart rate monitoring during submaximal exercise."

(PDF). Journal of Sports Sciences: 10. Retrieved 16 April 2015.

13) **Barr, SI (1999).** "Effects of dehydration on exercise performance". Canadian Journal of Applied Physiology. 24 (2): 164–72. doi:10.1139/h99-014. PMID 10198142.

14) **Cheuvront SN, Haymes EM (2001).** "Thermoregulation and marathon running: biological and environmental influences". Sports Med. 31: 743–62. doi:10.2165/ 00007256-200131100-00004. Porter, AM (2001). "Why do we have apocrine and sebaceous glands?". Journal of the Royal Society of Medicine.

15) González-Alonso, J; Mora-Rodríguez, R; Below, PR; Coyle, EF (1995). "Dehydration reduces cardiac output and increases systemic and cutaneous vascular resistance during exercise". Journal of applied physiology (Bethesda, Md. : 1985). 79 (5): 1487–96. PMID 8594004.

16) **Nybo, L (2008).** "Hyperthermia and fatigue". Journal of applied physiology (Bethesda, Md. : 1985). 104 (3):

17) **Dalsgaard, MK; Secher, NH (2007).** "The brain at work: A cerebral metabolic manifestation of central fatigue?". Journal of neuroscience research. 85 (15): 3334–9. doi:10.1002/jnr.21274. PMID 17394258.

18) **Ferreira, LF; Reid, MB (2008).** "Muscle-derived ROS and thiol regulation in muscle fatigue". Journal of applied physiology (Bethesda, Md. : 1985). 104 (3): 853–60. doi:10.1152/japplphysiol. 00953.2007. PMID 18006866.

19) **Romer, LM; Polkey, MI (2008).** "Exercise-induced respiratory muscle fatigue: Implications for performance". Journal of applied physiology (Bethesda, Md. : 1985). 104 (3): 879–88. doi:10.1152/ japplphysiol.01157.2007.

20) **Amann, M; Calbet, JA (2008).** "Convective oxygen transport and fatigue". Journal of applied physiology (Bethesda, Md. : 1985). 104 (3): 861–70. doi:10.1152/ japplphysiol.01008.2007. PMID 17962570.

21) Newsholme, EA; Blomstrand, E (1995). "Tryptophan, 5-hydroxytryptamine and a possible explanation for central fatigue". Advances in experimental medicine and biology. Advances in Experimental Medicine and Biology. 384: 22) Mayo Clinic. 23) **Nosaka, Ken (2008).** "Muscle Soreness and Damage and the Repeated-Bout Effect". In Tiidus, Peter M. Skeletal muscle damage and repair. Human Kinetics. pp. 59–76. ISBN 978-0-7360-5867-4.

24) Cheung, Karoline; Hume, Patria A.; Maxwell, Linda (2012-10-23). "Delayed Onset Muscle Soreness". Sports Medicine. 33 (2): 145–164. doi:10.2165/00007256-200333020-00005. ISSN 0112-1642. PMID 12617692.

25) **Davis, Paul.** "Careers in 75. Exercise Physiology".

26) American College of Sports Medicine (2010). ACSM's guidelines for exercise testing and prescription (8th ed.). Philadelphia: Lippincott Williams & Wilkins. ISBN 978-0-7817-6903-7.

27) University, Ohio. "Class Requirements".
