

(or *sequelae* in medical language). The usual short-term consequences of musculoskeletal injuries will include the following limitations to physical function, irrespective of the part of the anatomy to which the injury was sustained:

- Decreased physical strength: Muscular ability will begin to decline after 24 hours of inactivity.
- Nerve impulses slow: At optimal health, the nervous system can transmit some nerve impulses, such as those crucial to coordination and reaction, at speeds of over 300 ft (100 m) per second. Inactivity through injury will reduce the overall ability of the nervous system to stimulate movement.
- Circulation and metabolic rates will slow.
- Bone mineral density decreases: Injury tends to slow the rate at which calcium and vitamin D operate in union to produce new bone cells. Collagen, the cellular protein material that provides bone with the elasticity to absorb forces directed into the otherwise hard mineral surface, is not generated at pre-injury levels.
- Collagen level decreases in the connective tissues, primarily tendons and ligaments, making these structures stiffer, less elastic, less responsive to movement, and more vulnerable to injury.
- Reduced cardiorespiratory function: The ability of the body to process oxygen, described $VO_2\text{max}$, will decline by a limited amount in the first few days of inactivity due to injury, with pronounced declines exceeding 10% of peak oxygen uptake after 15 days.
- Reduced glycogen storage: Both the musculoskeletal muscles and the liver, the primary storage sites within the body for glycogen, will not maintain peak storage levels absent muscular activity that places demands upon the body's ability to utilize glucose, converted from glycogen.

The most common cause of musculoskeletal injury is a combination of physical overloads created by overtraining or by the repetitive use of a joint or a particular muscle group. Virtually every sport has a potential for this type of injury; these injuries are more often caused by training routines than by the stresses of a single competition. Distance running is a sport that by its nature will often create conditions for both overtraining as well as leg and foot injuries that are attributable to the repetitive strains of the activity. The injuries sustained in running are rarely connected to a single event, unlike the injuries of

many contact sports; running injuries commonly are a combination of the mileage covered by the athlete in a given training period, the pace with which the training distances are run, the nature of the terrain covered in training, as well as the unique physical characteristics of the athlete, such as structural deformities or imbalances and age. These overloads lead to micro fractures of the bone structure, muscle and tendon tears, and ischemia, the reduction of blood supply to an organ or tissue. In a cross-sectional analysis of the frequency of injury occurrence among athletes of all ages and ability levels, the greatest number of musculoskeletal injuries occur to males between the ages of 15 and 25 years.

The distribution of the frequency of the different types of musculoskeletal injuries is relatively equal between male and female athletes, although different types of specific injuries occurred more frequently due to the physiological differences between men and women. The most striking of these examples is the far greater risk that female athletes face regarding a prospective anterior cruciate ligament knee injury, due to the relatively wider pelvis in relation to femur length in the female anatomy.

Various sport and government organizations in North America and Europe have analyzed musculoskeletal injury rates. Approximately 25% of all athletes will expect to sustain a musculoskeletal injury in a 12-month period. The more fit and the more sophisticated the athlete, the more likely the risk of injury, due in part to the fact that such injuries often occur to athletes performing at a higher level with greater physical stresses and risks.

The research on such injuries also confirms that foot and ankle injuries are the most common of musculoskeletal injuries, constituting approximately 25% of these occurrences. Knee injuries of all types are the next most common, representing 22% of musculoskeletal damage. Back injuries are the next most prominent occurrence, at 11%. Injuries to the lower leg, thigh, hip, shoulders, and the hand/forearm structure each occur at frequencies of between 5% and 10%.

SEE ALSO Ankle sprains; Back injuries; Hand injuries; Knee injuries; Shoulder injuries.

Myostatin

Myostatin is a gene, one of the units of heredity consisting of a sequence of deoxyribonucleic acid (DNA) that determines the inherited characteristics

of every individual. It is a gene that contributes to the differentiation in growth factors, including physical size, and regulates muscle development. Unlike those factors that spur the growth of human structures, myostatin prevents muscles from growing too large. It is protein-produced in the skeletal muscle cells, interacting with the production of myocytes, the cells that ultimately form muscles.

Interest in myostatin is a relatively recent phenomenon. While the function of myostatin within the human body is a biological research frontier, the ability of certain cattle breeds to grow to enormous, well-muscled stature, particularly the Belgian blue, is well understood, as it is a breed that inherently possesses less of the myostatin gene.

Muscle size is both an inheritable trait as well as an attribute that may be altered through physical training, coupled with diet. A large number of proteins, referred to as growth factors (GFs), operate in different ways within the body. A GF will generally signal a cell as to its rate of growth and any differentiation from other cells. Some of these proteins, such as insulin-like growth factor-1 (IGF-1), influence cell growth throughout the entire body; myostatin has a specific impact restricted to muscle cell development.

In a healthy human, the effects of how the various GFs operate is best understood in the context of how the body recovers from a muscular injury. When a cyclist sustains a tear to the gastrocnemius, one of the two calf muscles, the repair of the torn segment commences almost immediately after the injury is sustained. IGF-1 controls the creation of the cells necessary to enable the damaged muscle fibers to be rebuilt and repaired. Depending on the nature and the extent of the damage to the muscle, the repairs triggered through the action of the IGF-1 hormone will continue over time.

It is a central principle of weight training and muscle development that the creation of tears in the fibers of the muscle are necessary to build a larger muscle. It is for this reason that weight training programs should provide for rest intervals that will allow the repairs to be affected at the cellular level and for the muscle fibers to grow. Acting alone, IGF-1 would be the facilitator of unchecked muscle growth and development. Myostatin appears to act as a counterbalance to the stimulation of muscle cell growth, as it serves to slow and ultimately limit the number of new cells created to build new muscle.

As muscle size is inheritable, there exists the potential to create a variable gene, where the

increase in muscle size in an athlete could be achieved through a decrease in the action of the myostatin. The precise details of how myostatin operates within the muscle cells are not yet known to sports science, as myostatin first became the subject of published scientific commentary in 1997. The principles of myostatin function are sufficiently understood to support the significant research undertaken to develop a myostatin inhibitor. Such research is directed not only at the athletic advantages that are believed to flow from such a product, but also to combat muscle-wasting diseases such as muscular dystrophy, various cancers, and AIDS. Research directed at both cattle and poultry, both of which have myostatin-type genes, have confirmed that, in theory, a myostatin inhibitor will permit greater muscle growth in human athletes.

Supplements known as myostatin blockers have become prominent in the weight training and bodybuilding markets. These products are widely advertised throughout the health and fitness industry, with seemingly countless variations available through the mass marketing of the Internet. The products that make the claim as possessing myostatin-blocking or inhibiting capabilities have not been the subject of scientific verification.

Further research on myostatin will focus in part on the risks of the wide-scale use of this prospective inhibitor. The impact of a myostatin blocker on the function of the heart and cardiovascular system is unknown. With greater muscle size through the administration of myostatin blockers, the risk of additional strain on tendons and bone structure through increased muscle mass must also be considered.

The prospect of genetic doping with respect to the limitation of the action of the myostatin gene is one that has been considered by international sport. The fear of agencies such as the World Anti-Doping Agency (WADA) is the development of a technology where a myostatin inhibitor could be injected into a specific tissue, permitting the enhanced development of the subject muscle.

In a more benign fashion, testing for the presence or extent of the myostatin gene in an individual has other potential applications. Testing for the extent of the myostatin gene would be useful in determining which persons would be best suited to sports involving significant muscular development.

SEE ALSO Anabolic steroids; Genetic prediction of performance; Genetics; Nandrolone.