



Applications of genetic modification technology : How do you see the sessions of heavy training loads for long hours in light of the genetic activation of muscles?

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Athletes travel to Athens to take part in a demonstration that began in Greece more than 2,000 years ago. While the most ingenious specimens test the extremes of human strength, speed and agility, some of the participants may be entangled in a more modern and less inspiring Olympic tradition: the use of performance-enhancing drugs. Despite the frequent scandals, muscle activation has become - for many athletes - an irresistible phenomenon. At least, to keep up with competitors who do so!. Since winning is paramount, athletes seize every opportunity to gain in terms of speed by fractions of a second, or to increase their stability even by a small amount.

Sports authorities fear that it will not be possible to detect new forms of muscle activation, and preventing them will become more difficult. Therapies for muscle regeneration, strengthening, and maintenance will soon enter the stage of human clinical trials for muscular dystrophy disorders. Among these treatments is giving patients synthetic (artificial) genes, the effect of which lasts for years, and produces large amounts of chemicals from which muscles are built in the normal state.

This type of gene therapy can greatly change the lives of the elderly and those with muscular dystrophy. Unfortunately, this dream treatment turned out to be a reality for an athlete who tends to stimulate muscle. As these chemicals are indistinguishable from their natural counterparts, they are generated locally within muscle tissue. Nothing enters the blood circulation, so there is nothing officially detectable in blood or urine tests. Indeed, the World Anti-Doping Agency (WADA) asked scientists to help it find ways to prevent gene therapy from becoming the latest method for muscle activation. But as these treatments enter the clinical trial stage and eventually become widespread, preventing athletes from accessing them will become impossible.

The skeletal muscles form the largest tissues in the body, and it is this type, especially the strongest of those

known as fast fibers, that declines with age. With this loss of strength, losing one's balance becomes more likely; and restoring this balance before the fall becomes more difficult. Skeletal muscle loss occurs with age in all mammals, and is likely to result from a cumulative failure to repair damage from normal use. Intriguingly, aging-related skeletal muscle changes parallel the functional and physical changes of a group of diseases known as Increasing muscle loads activates a number of signaling pathways, which leads to the addition of new cellular components within individual muscle fibers and to changes in the muscle fiber pattern. In extreme conditions, new muscle fibers are added.

In order to influence muscle growth, scientists combine the molecular details with each other in order to know how fibers muscle are built in the normal state, and how they are lost. Unlike a typical cell whose membrane is surrounded by liquid cytoplasm and a single nucleus, the muscle cell is actually a long cylinder multinucleated, and in which the cytoplasm consists of long, more precise fibers, known as myofibrils and these muscle fibers, in turn, consist of from stacks of contractile units (2), they are known as sarcomeres. It's shortening results in muscle contraction. However, the force generated by it can damage the muscle fiber if it is not directed outward. Dystrophin (the missing protein in Duchene muscular dystrophy) transmits this kinetic energy across the fibrous membrane, thus protecting the fibrous tissue. But even with this protective action generated by dystrophin, proper use also damages muscle fibers. In fact, this particular injury is thought to be one of the ways exercise builds muscle mass and strength. Microscopic ruptures of the fibers resulting from the effort create a chemical alarm, which triggers tissue regeneration, which in relation to the muscles does not mean the production of new muscle fibers, but rather the restoration of the outer membrane of the existing fibers and the repair of what is inside these fibers by adding new myofibrils. Making this new fibrous protein requires activation of related genes within the muscle

cell's nuclei. When the demand for myofibrils becomes great, the mobilization of additional nuclei becomes necessary to enhance the manufacturing capacity of the muscle cell. Unlike a typical cell whose membrane is surrounded by liquid cytoplasm and a single nucleus, the muscle cell is actually a long cylinder. A Myostatin appears to limit muscle growth during fetal development and adult life. It acts as an inhibitor that slows down normal muscle growth, possibly as an inducer of muscular dystrophy when the functional need for muscle action decreases. Experiments in genetically engineered mice indicate that the absence of this anti-growth factor leads to the formation of muscles of apparent bulkiness, due to muscle fiber hypertrophy and hyperplasia; Any significant increase in the number of muscle fibers.

Nature has provided examples of the effects of myostatin blockage, including the Belgian breeds of cattle, known as blue bulls and Piedmonts bulls. Both clones inherited a genetic mutation that produces a truncated, inactive version of the myostatin molecule. These cows are often called double-muscled animals (6). And their exaggerated muscular systems are more impressive, because the absence of myostatin also hinders the

accumulation of fat, so their bodies take on a smooth, non-sagging sculptural appearance. Myostatin-related muscle hypertrophy is a rare genetic condition characterized by reduced body fat and increased skeletal muscle size. Affected individuals have up to twice the usual amount of muscle mass in their bodies, but increases in muscle strength are not usually congruent.

Scientists themselves, as well as bodies including the World Anti-Doping Agency (WADA), the International Olympic Committee, and the American Association for the Advancement of Science, started discussing the risk of gene doping in 2001, and by 2003 WADA had added gene doping to the list of banned doping practices, and shortly thereafter began funding research on methods to detect gene doping.

Genetic enhancement includes manipulation of genes or gene transfer by healthy athletes for the purpose of physically improving their performance. Genetic enhancement includes gene doping and has potential for abuse among athletes, all while opening the door to political and ethical controversy.

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