Myostatin-B dysfunction in serially stressed humans during lean days

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University of Tokyo Professor Kazuo Takeuchi, says he has discovered one of the strongest components of traumatic arthritis in human muscles: a protein called MUSCLSP3 that impairs synthesis of a protein required for the development of a plant-like cell type called cells called myostatin-B.

Muscles already have this protein and they control the growth and remodeling of their structures. In animals, with the exception of some neurological diseases such as Huntingtonâ€™s disease and muscular dystrophy, damage to the cells in this important role-playing network is not expected and it is suggested that some substances, such as anabolic steroids, could cause this imbalance in muscle cells.

In an experiment in which repeatedly pumping anesthetics into mature muscle cells called mouse and rat mitochondria disrupted the synthesis of this protein, the scientists show that impairment of MUSCLSP3 profoundly impairs the synthesis of myostatin-B. Isofotonine, a human substance with all-natural anti-inflammatory qualities, shows similar effects.

A key point was establishing that this process is innate and it involves the protein synthase, which is required to generate all other mammalian protein substrates including myostatin-B. Moreover, this problem is not limited to the creation of muscle cells, but includes the expression of other proteins that have important functions in the body, such as the cellâ€™s transportation system. Thus, modulation of the expression of these molecules might be considered as a potential therapeutic approach to combat or at least manage the condition of systemic traumatic arthritis.

Dr. Takeuchi believes that the next step would be to test myostatin-B in live animal experiments. If a therapeutic approach is indeed established to combat the disease, the researcher hopes it could be quickly developed into a drug, which could be used to treat those with chronic trauma-mediated inflammatory arthritis and suffer from arthritis of joints involved in aging.

Dr. Takeuchi reports on his research findings in â€œMuscle myostatin-B transcriptional machinery is observed to be dysregulated in humans with systemic toxic arthritis.â€ Current Mutations in myostatin gene, which cause myostatin-B encoded, and Ferocious C2/AURIGEN region are well understood, but the many molecular pathways that converge to cause systemic toxic arthritis are not fully understood. Media enquiries may be sent by email (aiw@fuji.edu.jp) or phone (813) 476-1658.

Full text of the research, â€œMuscle myostatin-B transcriptional machinery is observed to be dysregulated in humans with systemic toxic arthritis,â€ published in the journal Cell on 15-15-2011, is available on our website (http://www.thctr.uni.jp/en/....

A Red Fire Hydrant In The Middle Of A Field