NILS SEMINAR

Anti-aging role of the mechano-sensing protein p130Cas in the maintenance of muscle tissues

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Abstract:
Muscle aging, termed sarcopenia, is characterized by progressive loss of muscle waste and consequent decline in muscular function and adaptability. Exercise is known to counteract the muscle aging effects, however, the mechanism remains largely unknown. While we previously reported that p130Cas acts as a cytoskeletal mechano-sensor, we found that phosphorylation of p130Cas was essential for the myogenic differentiation of C2C12 myoblasts involving actin-dependent nuclear accumulation of MAL. In addition, we found that p130Cas was proteolyzed in oncogenic Ras (RasV12)-senesced myoblasts that were defective in differentiation. Furthermore, electrical stimulation rescued p130Cas from proteolysis in RasV12-senesced myoblasts. Since p130Cas was also restored from proteolysis in RasV12-transformed NIH3T3 cells, electrical stimulation appeared to have en effect similar to mechanical stretching in terms of the protection of p130Cas. Based upon these observations, we postulate that senescence-induced p130Cas proteolysis might play a role in the development of sarcopenia. Furthermore, the protection of p130Cas may be involved in the mechanism underlying the anti-aging effect of physical exercise on muscle tissues. In the seminar, our recent results from analysis of muscle tissues of sarcopenia model mice will also be discussed.

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東棟6F会議室

運動器疾患研究部　池田（5501）