Studies on molecular mechanisms of lipid accumulation in adipocytes for anti-metabolic syndrome

Ryuichiro Sato Ph. D.

(The University of Tokyo, Department of Applied Biological Chemistry, Professor)

【Outline of survey】

It is hoped to curb growth in medical spending in Japan that is becoming an aging society with a falling birthrate. Increasing in the number of patients afflicted with lifestyle-related diseases should be inevitable as the population ages. Emphasis is, therefore, on approaches for public health maintenance utilizing food functions. Based on these circumstances, the current project is aiming at basic researches for prevention of metabolic syndrome.

As obesity triggers metabolic syndrome, it is needed to study life phenomena occurring in adipocytes in greater details. Although it is well known that obesity is caused by excess accumulation of lipids in lipid droplets in adipocytes, the precise mechanism how lipid droplet formation is accelerated remains unclear. Also detailed studies on biological changes brought about by the lipid droplet formation in adipocytes have not yet been done. We attempt to study molecular mechanisms underlying the lipid droplet formation and thereby develop applied researches for evaluating food functions for anti-metabolic syndrome. At the same time, new findings produced by the current study will be utilized for drag development.

【Expected results】

Adipocytes supply energy by degrading lipids when energy is required as well as accumulate lipids in lipid droplets when oversupplied. It can be expected that targets for anti-obesity are clearly identified by elucidating the molecular mechanism by which formation and degradation of lipid droplets in adipocytes occur. Some food factors and drags affecting functions of targets newly identified should be potent candidates for anti-metabolic syndrome.

【References by the principal investigator】

・Kanayama, , T., Arito, M., So, K., Hachimura, S., Inoue, J. and Sato, R. (2007) Interaction between sterol regulatory element-binding proteins and liver receptor homolog-1 reciprocally suppresses their transcriptional activities. *J. Biol. Chem.* 10290-10298.

・Arimura, N., Horiba, T., Imagawa, M., Shimizu, M. and Sato, R. (2004) The peroxisome proliferator-activated receptor g regulates expression of the perilipin gene in adipocytes. *J. Biol. Chem*. 279, 10070-10076.

【Term of project】FY2007－2011	**【Budget allocation】 20,700,000 yen** (2007 direct cost)

【Homepage address】 http://park.itc.u-tokyo.ac.jp/food-biochem/