Loss of social interaction induces obesity in mice

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It has been reported that daily exposure to social and psychological stress is associated with life-style related diseases such as hyperglycemia, cardiovascular diseases and cancer, as well as mental disorders. The stress is considered to link to obesity. In the previous study, we evaluated changes in the hepatic gene expression profile using a DNA microarray in mice exposed to isolation stress for consecutive 30 days and found that consecutive social stress remarkably down-regulated the lipid metabolism-related pathway through peroxisome proliferator activated receptor alpha (PPARα), while the lipid biosynthesis pathway controlled by sterol regulatory element binding factor 1(SREBP1) was significantly up-regulated (Motoyama et al., 2009). These results suggested that a mild and consecutive social stress affects the systems for lipid metabolism. Therefore, we investigated whether isolation stress induces obesity or not.

Male BALB/c mice (4 weeks old, Japan SLC, Shizuoka, Japan) were housed at 5 mice per cage. After acclimatization for 10 days, the mice were exposed to isolation stress for 3 months with normal diets. Their body weights were began to increase after 9 weeks of the exposure compared with non-stressed controls, and this difference was significant at 13 weeks with accumulation of visceral fat and also hepatomegaly. Significantly higher levels of glucose and total cholesterol, and conversely lower levels of adiponectin, triglyceride, free fatty acid, and creatine kinase were observed in the stressed mice. The analysis of gene expression showed that isolation stress for 3 months toward to the energy consumption by both reduction of fatty acid synthesis pathway(*Ppara*, *Acox1*, *Ehhadh*) and enhancement of the fatty acid degradation pathway(*Srebf1*, *Fasn*, *Elovl6*).

These results suggest that prolonged daily exposure to social stress at adolescent stage induces obesity

Reference: Motoyama, K. et al. *Physiol. Genomics*. 37, 79-87 (2009)