the other AAs such as alanine and urea cycle AAs but only a minor part was utilized for ammonia synthesis. A significant amount of 13C and 15N in circulating AAs was also evident. However, incorporations of dietary Glu-N into circulating alanine, glutamine, glycine and BCAA were much higher than those of Glu-C. These results indicated importance of dietary Glu as N-source for intestinal AA synthesis and for maintenance of circulating AAs.

PD2-098 Suppression of muscle atrophy by oral administration of methionine

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Objectives: Loss of muscle weight with undernutrition, aging and disuse is major public health concern. It is suggested that leucine stimulates muscle protein synthesis and suppresses protein degradation. The present study was performed to examine suppression of muscle protein degradation by dietary methionine (Met).

Methods: Exp. 1, Male Wistar rats (4 weeks of age) were orally administered 8mg, 20mg, 40mg, or 80mg /100g body weight of Met. Extensor digitorum longus (EDL) muscle was isolated after 3h of methionine administration. 3-Methylhistidine (MeHis) release from EDL muscle was measured by HPLC. Calpain activity and proteasome activities were measured. LC-I and LC-2, markers of autophagic activity were determined by Western blotting. Atrogin-1 and MuRF-1 expression were measured by Northern blotting. Exp 2, Male Wistar rats (4 wk) were fed 20% casein, 5% casein or 0.35% Met-5% casein diet for 6 days and then muscle weight was measured.

Results: MeHis release from the muscle, an index of myofibrillar protein degradation was dose dependently decreased. Calpain and proteasome activity were not changed with dose. Expressions of atrogin-1 and MuRF-1 were not different among dose. However, LC-II and LC-I ratio was significantly decreased in rats administrated 80mg Met. Dietary supplementation of methionine for 6 days suppressed loss of EDL muscle weight by feeding of 5% casein diet.

Conclusions: Oral administration of Met decreased muscle protein degradation through the suppression of autophagy formation. Thus dietary supplementation of Met may have beneficial effect on muscle atrophy.

PD2-099 The impairments of glucose stimulated insulin secretion caused by dietary oxidized frying oil

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Objectives: We previously reported that, in rodents, a diet with a high oxidized frying oil (OFO) content leads to glucose intolerance associated with a reduction in insulin secretion. This study aimed at investigating the impairments of pancreatic islets caused by dietary OFO. Methods C57BL/6J mice were divided into three groups to receive a low fat basal diet containing 5 g/100 g of fresh soybean oil or a high fat diet containing 20 g/100 g of either fresh soybean oil or OFO. 13-hydroxy octadecadienoic acid (13-HODE), an oxidized fatty acid which has been demonstrated to be absorbed by the intestine and released into circulation within triacylglycerol-rich lipoproteins, was also tested on HIT-T15 β cell line. Results After feeding experimental diet for 8 weeks, mice fed OFO showed glucose intolerance and hypoinsulinemia and their islets showed decreased glucose-stimulated insulin secretion (GSIS). Significantly higher oxidative stress and lower mitochondrial membrane potential, vitE content and insulin were observed in islets from the OFO-fed mice. Immunoblots showed that the reduction in insulin levels in islets was associated with activation of c-Jun NH2-terminal kinase (JNK) and a reduction in levels of pancreatic and duodenal homeobox factor-1 (PDX1). The 13-HODE mediated effects on GSIS and JNK/PDX1/insulin signaling were also investigated in HIT-T15. Conclusions We conclude that dietary OFO-impaired glucose metabolism might be associated with 13-HODE, which compromised GSIS by activating JNK pathway in pancreatic β cells.

PD2-100 Thermic effects of animal protein diet and plant protein diet on postprandial energy expenditure(PEE) in healthy female adults

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Background: Recently, there are many efforts to increase TEF by changing nutrient source, which may be relevant for weight loss program.

Objective: The effects of animal protein diet and plant protein diet on PEE were compared.

Method: Seven healthy university female students (mean age 22.3±0.5 yrs) participated in two isoenergetic diet interventions, which providing 15% of each subjectís resting metabolic rate (RMR, calculated by DRIís equation), consisting of 22% protein (chicken or tofu), 18% fat, and 60% carbohydrates. Energy expenditure was measured in resting and postprandial state (every 30-min during 4 hours) using an indirect calorimeter.

Result: There were no significant differences in PEEs between chicken and tofu diet. PEEs of chicken diet group increased more rapidly (peak on 30 min) than those of tofu diet (peak on 120 min). However, PEEs of tofu diet decreased relatively faster after peak time.
Total thermogenesis of animal protein diet was 16.8 kcal/4h, higher than plant protein dietís (13.7 kcal/4h), but not significantly different. 8.68% of energy intake in chicken diet and 6.94% in tofu diet were oxidized as thermic effects for the digestion and absorption of the diets.

Conclusions: Further studies that using higher energy content in test meals should be continued to find the adequate protein source for increasing TEF.