99.10 **THE EFFECT OF CREATING SUPPLEMENTATION ON GLUCOSE TRANSPORT IN RAT SKELETAL MUSCLE**

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It is well established that glucose transport in skeletal muscle is stimulated by contraction and by substrates associated with a decrease in muscle phosphorylation. However, the effect of a raised phosphorylase concentration on glucose transport is not clear. Since dietary supplementation of creatine has been shown to increase phosphorylase in skeletal muscle, sprague-dawley rats were administered creatine monohydrate (100 mg/kg) daily for 7 days and then stimulated with the epinephrine muscle removed from such fasted and incubated by the method of Baron (Life Sci 61: 335, 1997). One muscle was incubated in the absence of insulin and the other was exposed to a maximally stimulating concentration of insulin (20 mU/m). Glucose transport was measured by 2-deoxy-D-glucose uptake. In a separate study, urinary creatine excretion, an indicator that an upper limit of total muscle creatine concentration has been reached, was increased by 30% in creatine-supplemented rats (1.87±0.1 vs 1.25±0.07 mg/24 hr, P<0.05). Insulin-stimulated 2-D-glucose transport in epinephrine was significantly increased from between muscles from creatine-supplemented and control rats, 8.4±2.04 to 9.7±1.63 and 2.5±2.0 to 5.0±1.1 mmol/g/min for creative supplemented and control muscles, respectively (n=4 each). Three results suggest that in increasing phosphorylase above normal levels has no effect on basal glucose transport or insulin responsiveness in skeletal muscle.

99.12 **PLASMA INSULIN RESPONSES FOLLOWING THE INGESTION OF VARIOUS AMINO ACID/PROTEIN-CARBOHYDRATE MIXTURES**

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The combined intake of protein and carbohydrate results in an increased insulin response. Increases in plasma insulin levels have also been reported following meals high in protein. In order to define a mixture with strong insulotropic properties when added to a carbohydrate solution we studied plasma insulin responses following the ingestion of carbohydrates, in combination with free amino acids (valine, phenylalanine, arginine and/or glutamine), protein hydrolysates (with or without free amino acids) and an intact protein (casein). Right after healthy, non-obese subjects had fasted overnight, an overnight fast, on 10 in a consistent with for 6 hours. Subjects ingested a beverage containing 3.5 mg/kg every 30 min leading to an intake of 0.8 g/kg glucose and 0.4 g/kg amino acid and protein (hydrolysate-mixture). All trials resulted in a strong increase in plasma glucose and insulin levels during the first 30 min, after which large differences between trials became apparent. After expressing the insulin response as area under the curve during the 2nd hour the ingestion of the drinks containing free leucine, phenylalanine and arginine and the drinks containing free leucine, phenylalanine and leucine protein hydrolysate were followed by the insulin response (201 and 209%, respectively; P<0.05). Compared to the carbohydrate-only drink, insulin responses strongly correlated with increases in plasma leucine (P<0.003), phenylalanine (P<0.01) and tyrosine (P<0.001) levels.

99.14 **EFFECTS OF CLONIDINE AND PALMITATE ON GLUCOSE INDUCTION INSULIN SECRETION FROM ISOLATED ILETS IN CHLORIDE FREE MEDIA**

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Considerable evidence has been accumulated that supports the importance of chloride for the stimulation of insulin release from islets in response to glucose and other nutrients. The effects of altered chloride concentrations on insulin secretion are believed to be mediated by changes in beta cell membrane potential, direct inhibition of calcium channels or disruption of chemical processes leading to insulin exocytosis. We investigated the potential role that chloride may play in mediating the effects of AAAR inhibition and fatty acid potentiation of glucose stimulated insulin secretion from isolated rat islets. Islets were incubated in Krebs-Ringer bicarbonate buffer containing 0.2 mmol/L chloride for the remaining 55 minutes. Blood samples were removed from the reservoir at min 15, 30, 45, and 60. Linear relationships were generated comparing HGO to 1000 mg of glucose per hour. There were no significant differences in slope between MC, FC, or ME. However, the slope was significantly lower for PE corresponding to 100% reduction of IGU in the chronologically fed female islets compared to the other groups. Consistent with the decrease in HGO, PE animals remained 20 percent less lactate from the reservoir. The results indicate that female animals chemically consuming an alcohol, a deterrent in hepatic glucagonic capacity. Funded by NIH grant R03-AA11259-02

99.13 **EFECTS OF CLONIDINE AND PALMITATE ON GLUCOSE INDUCTION INSULIN SECRETION FROM ISOLATED ILETS IN CHLORIDE FREE MEDIA**

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Previous studies showed starch induced 3-OH-3-3-Palmitoylglucose uptake was decreased in skeletal muscle and that glucagon uptake after a meal consumed a high-sodium oil (HF)-based diet relative to rats on a low-fat (LF) diet for 3-4 weeks. When a high mixed diet (HF- MIX)-based diet was compared only glucose transport (in muscle) but not glucose uptake (in adipocytes) decreased [Wilke et al., 1998]. We hypothesized that there would be up-regulation of non-insulin-mediated glucose (basal) uptake in HF-sal and HF-fats muscle and that HF-sal adipose tissue to help compensate for insulin resistance. In adipocytes, basal glucose uptake was similar in HF-sal, HF-mix, and LF (1.3 ± 0.1 mmol/L 1.8 ± 0.5 min, 1.5 ± 0.3 mmol/L and 1.8 ± 0.5 mmol/L, respectively). In contrast, basal 3-3-Palmitoylglucose uptake in soleus strips was significantly higher (+428%) in HF-sal (4.5 ± 0.4 mmol 3.0 min, 4.0 ± 0.4 mmol 3.0 min, and 4.5 ± 0.4 mmol 3.0 min, respectively) vs LF (1.5 ± 0.2 mmol 3.0 min, P<0.05). GLUT-1 levels in soleus muscle from HF-sal (312 ± 225 ± 221) and HF-sal (218 ± 22) were significantly higher (+78%) compared to GLUT-1 in soleus muscle from LF (170 ± 334). Conclusions: Soleus muscle from rats that were fed diets high in unsaturated fat have elevated basal glucose uptake compared with controls. The increased GLUT-1 expression may compensate for insulin resistance by elevating basal glucose uptake.