

higher (at least 4–10-fold) in oxido-glycolytic bovine skeletal muscles than in oxidative muscles or heart. On the contrary, it has been previously shown in the rat that heart and oxidative muscles contain higher amounts of GLUT4 than glycolytic muscles. This species-specificity of GLUT4 expression may favour short-chain fatty acids and ketone body utilization by oxidative muscles in bovine and glucose utilization by oxidative muscles in the rat.

Differences in food digestion and metabolism between ruminant and monogastric species are well known. However, these results demonstrated large differences in the basic biological mechanisms of molecular regulation of muscle metabolism in ruminant and monogastric animals. These differences affect energy metabolism and particularly glucose transporters.

D-Glucose has an immediate and specific sensitizing effect on chicken pancreatic β cells. N Rideau¹, C Saulnier² (¹ INRA, Station de Recherches Avicoles, 37380 Nouzilly; ² CNRS URA 307, Laboratoire de Physiologie du Développement, Université Paris VII, 2, place Jussieu, 75251 Paris cedex 05, France)

The perfused isolated chicken pancreas is insensitive to both D-glucose and other known 'initiators' of insulin release when used alone. For example the total cumulative insulin output in response to 10, 20 or 40 mM α -ketoisocaproic acid (KIC) perfused alone was low, 7 ± 3 , 1 ± 1 , 2 ± 1 ng/30 min (mean \pm SEM, $n = 4$, $p > 0.05$) respectively. The potentiation of α -KIC (10 mM) was studied by associating α -KIC with various fuel nutrients perfused at non-insulinotropic concentration in the chicken. The total cumulative insulin output (ng/30 min, mean \pm SEM, $n = 4$) was :

1. α -KIC (10 mM) in the presence of carbohydrates:

alone	+ D-glucose 14 mM	+ 3-O-methyl-D-glucose 14 mM
7 ± 3	212 ± 49	11 ± 6
	+ D-glyceraldehyde 5 mM	+ D-mannose 50 mM
	156 ± 49	16 ± 13

2. α -KIC (10 mM) in the presence of amino acids:

alone	+ L-asparagine 10 ± 10	+ L-glutamine 24 ± 10
	+ L-asparagine 17 mM + L-glutamine 17 mM 127 ± 31	

α -KIC was significantly ($P < 0.05$) potentiated by D-glucose (14 mM), D-glyceraldehyde (5 mM) or L-asparagine (17 mM) + L-glutamine (17 mM). The response with the last 2 nutrients was however delayed (7–70 min) as compared to that observed in the presence of D-glucose. These results suggest that D-glucose at non-insulinotropic concentrations exerts an immediate specific sensitizing effect on the chicken pancreatic β cell. A sensitizing effect of glucose was also observed with D-glyceraldehyde (poorly insulinotropic alone) or D-mannose (non-insulinotropic alone). Thus the metabolic step initiating insulin release is only found in the chicken. Before testing this hypothesis, it is necessary to isolate and characterize the A and B islets of Langerhans in the chicken. An immunohistochemical identification (immuno-peroxidase) method of isolated chicken islets is in progress.

Insulin resistance induced by post-weaning high fructose diet in Wistar rats: reversal by metformin. S Halimi¹, E Rosini¹, PY Benhamou¹, P Faure¹, P André² (¹ Laboratoire de Recherches Métaboliques GREPO, Université et CHU de Grenoble; ² Groupe Liphia, France)

The fructose intake in the western diet is increasing and can sometimes reach 30% of the total carbohydrate intake. Fructose is known to have smaller hyperglycemic and insulinosecretory effects than glucose. Fructose has been recommended as sweetener of natural foods for both diabetic patients and normal subjects, including children. Some deleterious effects have been described, such as hypertriglyceridemia, hyperuricemia and hepatic insulin resistance in some circumstances or subjects. The aim of this work was to study hepatic and peripheral insulin sensitivity in post-weaning male Wistar rats fed a high fructose diet (58% of total carbohydrates, Fr58) for 4 weeks. They were compared with control animals fed a standard chow (Cer) and with another group of animals fed a high fructose diet