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Effects of short-chain fructo-oligosaccharides on insulin sensitivity and gene expression of fat tissue in obese dogs

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Canine obesity is strongly associated with hyperinsulinaemia and insulin resistance, both primary risk factors for type 2 diabetes in human subjects. Dietary fibres may modulate some variables associated with insulin resistance and glucose homeostasis. Their efficacy differs, however, according to their origin, physical properties and fermentability in the large bowel. The objective of the study was to evaluate the effects of fructo-oligosaccharides (FOS) on insulin sensitivity.

Eight healthy Beagle dogs (body condition score (BCS) 3/5; body weight (BW) 14.1 (SE 1.3) kg) were fed once daily a regular commercial diet (17 489 kJ (4180 kcal), 170 diethyl ether extract and 270 g crude protein/kg) until they became obese (BCS >6/9; BW 19.2 (SE 2.3) kg). They were then maintained obese and included in a cross-over designed study as obese–control or as obese–FOS (10 g FOS/kg DM included in the diet). The euglycaemic–hyperinsulinaemic clamp technique was performed before and after fattening and at the end of each 6-week period of the cross-over study. Fat-tissue biopsies were taken before and 1 h after the meal in order to measure the mRNA abundance of genes involved in fatty acid or glucose metabolism or inflammation. Data were analysed using Proc Mixed (SAS institute Inc., Cary, NC, USA) and were considered significantly different at $P<0.05$.

The energy allowances were 1079 kJ (258 kcal)/kg $BW^{0.75}$ and 782 kJ (187 kcal)/kg $BW^{0.75}$ respectively during fattening and stable obesity. Insulin resistance appeared progressively with fattening and the rate of glucose infusion during euglycaemic clamp was lower ($P<0.05$) in obese dogs than in lean dogs. Fasting plasma insulin and TAG concentrations were increased ($P<0.05$) in obese dogs compared with lean dogs, while fasting plasma glucose, insulin, TAG and cholesterol concentrations were not altered by FOS. By contrast, obese dogs were less resistant to insulin with FOS supplementation than with the control diet, as shown by the infusion rate of glucose during the euglycaemic clamp (6.6 (SE 1.9) *v.* 3.8 (SE 1.3) respectively). While FOS did not alter fasted mRNA levels, in the fed state there was a tendency towards increased carnitine palmitoyl transferase 1 ($P=0.05$) and uncoupling protein 2 ($P>0.10$) mRNA transcription in subcutaneous fat tissues.

Adding FOS (10 g/kg DM) to the diet of obese dogs decreases insulin resistance, although there is no effect on fasting blood variables. FOS may also modulate transcription of genes involved in fatty acid or glucose metabolism. The mechanisms of action of FOS on insulin resistance in dogs remain unknown.