RESPONSES TO HOMEOSTATIC SIGNALS IN RACTOPAMINE-TREATED PIGS

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Ractopamine (RAC) is a ß-agonist which increases lean tissue deposition in pigs, without necessarily affecting fat deposition (Dunshea 1991). The aim of this study was to determine whether this lack of effect on fat deposition was due to alterations in adipose tissue responses to homeostatic signals, such as insulin and ß-agonists, in RAC-treated pigs.

Eight female pigs (initial BW, 73 kg) prepared with jugular vein catheters were fed 3.0 kg/d (6 x 0.5 kg every 4 h) a wheat-based diet (14.5 MJ DE/kg, 0.65 g available lysine/MJ DE) containing either 0 or 20 ppm RAC for 24 d. Pigs received an i.v. challenge of insulin (1 μg/kg) on d 3, 9 and 23. Challenges of the $_2$-agonist fenoterol (2 μg/kg) were administered on d 4, 10 and 24. Blood samples were taken at -30, -20, -10, -1, 2.5, 5, 10, 20, 30, 45, 60 and 120 min relative to challenges. Plasma were analysed for glucose and non-esterified fatty acids (NEFA). In addition, insulin and fenoterol challenges were administered on d 30 and d 31 (ie. six and seven days after withdrawal of RAC), respectively. Data are expressed as area above or below basal.

	Day 3/4		Day 9/10		Day 23/24			
RAC (ppm)	0	20	0	20	0	20	sed	Significance[1,2]
Insulin								
NEFA, μM.min	-114	-304	-22	-120	-86	-74	101	R^{+}
Glucose, mM.min	-81	-102	-73	-67	-64	-85	9	D*
Fenoterol								
NEFA, μM.min	2314	1071	2206	554	2538	855	194	R***, D*
Glucose, mM.min	11	14	29	13	23	22	7	

[1]R, RAC treatment; D, day. [2]+ P<0.10, * P<0.05, *** P<0.001

Insulin was antilipolytic as evidenced by a small decrease in plasma NEFA after insulin challenge in most instances with the plasma NEFA response tending to be greater (P=0.075) in RAC-treated gilts. Insulin injection caused acute hypoglycaemia in all pigs although there was no difference between treatments. Intravenous challenge with fenoterol stimulated lipolysis as evidenced by an acute increase in plasma NEFA in all gilts. However, the lipolytic response was markedly reduced in RAC-treated gilts. Lipolytic responses returned to control values by 7 d after withdrawal of RAC (2464±817 vs 2333±606 μM.min, P=0.90). These data suggest that RAC treatment results in de-sensitisation of adipose tissue ß-adrenergic receptors. Thus, lipolytic responses to both endogenous and exogenous adrenergic stimuli would be diminished and this is reflected by minimal effects of RAC on fat deposition in gilts (Dunshea 1991).

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