Dietary ractopamine decreases adipose tissue sensitivity but not responsiveness to β-adrenergic challenge in the pig

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Ractopamine (RAC) is a β-adrenergic agonist which increases lean tissue deposition in pigs, without necessarily altering fat deposition (1). It has been demonstrated that the lipolytic response to a single β-adrenergic challenge (eg fenoterol) is diminished during dietary ractopamine treatment (2). The aim of this study was to determine whether this lack of effect on fat deposition was due to alterations in adipose tissue sensitivity and/or responsiveness to β-adrenergic stimulation.

Nine male pigs with venous catheters were fed 3 kg/d of a protein and energy adequate diet containing either 0 (n = 4) or 20 (n = 5) ppm RAC. On d 34 pigs were given increasing intravenous doses of fenoterol (3, 9, 27, 81, 243 mg/kg body weight) and blood samples taken for analysis of plasma non-esterified fatty acids (NEFA). Lipolytic responses were defined as peak increase in plasma NEFA above basal after each injection and are presented as the mean ± standard error. Data from each individual pig were fitted to an exponential function and maximal response (Rmax) and the dose which gave a lipolytic response 50% that of Rmax (ED50) were estimated.

	Dose of fenoterol[1]					Estimated	
	3	9	27	81	243	R_{max}[2]	ED_{50}[1]
Control[2]	298 ± 62	693 ± 199	1203 ± 102	1563 ± 155	1835 ± 300	1739 ± 232	11.5 ± 2.3
Ractopamine[2]	100 ± 17	195 ± 24	314 ± 47	679 ± 62	1513 ± 370	1847 ± 415	107.0 ± 24.1
P-value	0.011	0.026	<0.001	<0.001	0.54	0.83	0.010

[1] μg/kg body weight; [2] Plasma NEFA response (μmol/L)

For the control pigs the lipolytic response increased with dose of fenoterol and clearly reached a plateau (Rmax) within the dose range of fenoterol used. On the other hand, the NEFA response to fenoterol in RAC-treated pigs did not appear to reach a clear plateau. However, since the responses in both the control and RAC-treated pigs were not significantly different at the highest fenoterol dose, it can be concluded that there was no apparent effect of RAC on maximum lipolytic responsiveness. This was confirmed from comparison of the Rmax estimated from the fitted curves. Estimated ED_{50} was increased almost 10-fold by RAC indicating that adipose tissue sensitivity was decreased.

Therefore, while adipose tissue responsiveness is unchanged, there is a de-sensitisation of adipose tissue β-adrenergic receptors during RAC treatment. This could explain why exogenous dietary RAC has little effect upon the rate of fat deposition (1).

Supported in part by the Australian Pig Research and Development Corporation.

1. Dunshea FR. Effect of metabolism modifiers on lipid metabolism in the pig. J Anim Sci 1993;71:1966.
2. Dunshea FR, King RH. Responses to homeostatic signals in ractopamine-treated pigs. Brit J Nutr 1995;73:809.