

Improvement of meat quality in pigs by beta-adrenergic blockade

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Introduction

The porcine stress syndrome, which often leads to the development of pale, soft exudative (PSE) muscle at slaughter, and drug-induced malignant hyperthermia (MH) are characterised by similar metabolic changes and MH has been suggested as a useful model to study the mechanism of stress-susceptibility in pigs (Lister, Lucke and Hall, 1977). Both phenomena involve severe muscle stimulation and high concentrations of catecholamines are found in the blood. The metabolic effects of catecholamines in skeletal muscle are mainly concerned with carbohydrate metabolism and are mediated through beta-adrenergic receptors (Brody and McNeill, 1970). Beta-adrenergic blocking drugs, however, are not effective in the prevention or treatment of MH (Lister, Hall and Lucke, 1976). On the other hand, massive alpha-blockade prevented the death of stress-susceptible pigs subjected to suxamethonium-induced MH (Lister, Hall & Lucke, 1976) and alpha agonists will stimulate MH in these animals (Hall, Lucke and Lister, 1977). It is not certain that these are direct effects of alpha-adrenergic agonists or secondary effects caused by vasoconstriction in skin or muscle (Gronert, Milde and Taylor, 1980). Topel, Wilson, Weiss and Christian (1973) could find no effect on muscle colour or ultimate pH in pigs in which either partial alpha or beta-blockade was produced.

A new beta-adrenergic blocker, carazolol (1-(4-carbazolyloxy)-3-isopropyl-amino-2-propanol) has recently been introduced with a potency *in vivo* approximately one hundred times greater than propranolol (Innis, Correa and Snyder 1979). It has been proposed that carazolol could be used in the alleviation of stress-related disorders in pigs (Fiebiger *et al*, 1978). The present experiments were conducted to examine whether carazolol would improve meat quality in stress-sensitive and stress-resistant pigs.

Materials and Methods

Twenty Large White and 18 Pietrain pigs weighing about 70 kg were used in the experiments. Pigs of each breed were randomly allocated to the control and treated groups in equal numbers. Treated pigs were injected intramuscularly behind the right ear with the beta-blocking drug carazolol (1-(4-carbazolyloxy)-3-isopropyl-amino-2-propanol, SUACRON<sup>R</sup> Praemix Wirkstoff GmbH, Mannheim, Federal Republic of Germany) at a dose of 1 mg/100 kg live weight 0.5h before being loaded onto a standard commercial animal transporter and transported to the Meat Research Institute slaughterhouse. This journey took 4h and covered about 150 km. On arrival, the pigs were rested in lairage for 1h before being electrically stunned (90V, 50 Hz) and exsanguinated.

At slaughter a sample of blood was collected into heparin (25 U .ml<sup>-1</sup>) and the plasma analysed for glucose (Boehringer, Kit No. 124036), lactate (Boehringer, Kit No. 124168) and free fatty acids (FFA). FFA were

extracted according to Dole and Meinertz (1960) and measured using the method of Duncombe (1963).

At 15 minutes *post mortem* samples of liver and *m. longissimus dorsi* (LD) in the region of the last rib were collected into liquid nitrogen for measurement of glycogen (Sugden, Sharples and Randle, 1976) and lactate. Temperature was recorded in the LD. Forty five minutes *post mortem* the pH was measured (pH<sub>4.5</sub>) on an homogenate of a sample of LD in 5 mM sodium iodoacetate, 150 mM potassium chloride pH 7.0, and a Fibre optic probe measurement (FOP) made (MacDougall and Jones, 1975). The FOP value is a measure of the light-scattering properties of the muscle; higher values indicate paler, more opaque muscles.

Twenty-four h *post mortem*, water holding capacity (WHC) was measured in the LD (Grau and Hamm, 1953) and expressed as the ratio of the outer fluid region to the area of muscle sample (Briskey *et al*, 1959). Drip loss (% drip) from the LD was estimated on slices 1-1.5 cm thick cut across the long axis of the muscle at the last rib and hung individually in plastic netting bags inside inflated polythene bags for 72 h at +10°C.

Results are expressed as means ± standard errors. The significance of differences between means was tested using Student's *t* and between variances using the *F*-ratio.

Results

The effects of beta-blockade on meat quality measurements are given in Table 1. In the Large White pigs beta-blockade had no significant effect on meat quality which was very good in both control and treated groups. In the Pietrains beta-blockade reduced muscle temperature 15 minutes *post mortem* ( $P < 0.001$ ), raised pH<sub>4.5</sub> (reduced FOP value ( $P < 0.01$ ) and improved WHC ( $P < 0.001$ )). This resulted in significantly ( $P < 0.01$ ) smaller weights of drip lost from the stored muscle samples. Beta-blockade also reduced the variation in the FOP value in the Pietrains. Based on measurements of pH<sub>4.5</sub> and FOP, seven out of the nine control Pietrains would be considered to exhibit the PSE condition (pH<sub>4.5</sub> < 5.9, FOP > 30) while none of the treated group would.

Beta-blockade reduced initial (15 minutes *post mortem*) liver glycogen significantly in both Large White pigs ( $P < 0.01$ ) and Pietrains ( $P < 0.001$ ) (Table 2) and, in the Pietrains, variation in glycogen concentration was also reduced ( $P < 0.01$ ). Beta-blockade increased initial muscle glycogen concentrations ( $P < 0.05$ ) and reduced initial muscle lactate levels ( $P < 0.01$ ) in Pietrains but had no effect in the Large Whites.

Table 1. Meat quality measurements in the LD of control and beta-blocked pigs of Large White (n = 10 per group) and Pietrain (n = 9 per group) breeds (means  $\pm$  SE)

	Breed	Control	$\beta$ -block	Significance of difference between means
T <sub>OC</sub> 15 LD	LW	36.8 $\pm$ 0.3	36.1 $\pm$ 0.2	NS
	P	38.2 $\pm$ 0.4	36.1 $\pm$ 0.2	P < 0.001
pH LD	LW	6.82 $\pm$ 0.09	6.89 $\pm$ 0.08	NS
	P	5.86 $\pm$ 0.16	6.57 $\pm$ 0.07	P < 0.01
FOP LD	LW	14.0 $\pm$ 0.4	13.4 $\pm$ 0.4	NS
	P	31.2 $\pm$ 4.7	14.8 $\pm$ 0.5	P < 0.01
WHC LD	LW	1.76 $\pm$ 0.15	1.75 $\pm$ 0.09	NS
	P	2.78 $\pm$ 0.16	1.73 $\pm$ 0.11	P < 0.001
% drip (Chop)	LW	6.8 $\pm$ 1.0	6.0 $\pm$ 0.8	NS
		13.3 $\pm$ 0.8	10.0 $\pm$ 0.7	P < 0.01

Table 2. Concentrations of glycogen and lactate in the liver and LD at 15 minutes post-mortem in control and beta-blocked pigs (means  $\pm$  SE)

	Breed	Control	$\beta$ -block	Significance of difference between means
Liver glycogen (mg.g <sup>-1</sup> )	LW	12.3 $\pm$ 2.2	3.9 $\pm$ 1.5	P < 0.01
	P	10.8 $\pm$ 2.6	0.8 $\pm$ 0.3	P < 0.01
LD glycogen (mg.g <sup>-1</sup> )	LW	9.2 $\pm$ 0.5	9.7 $\pm$ 0.6	NS
	P	6.2 $\pm$ 1.0	8.6 $\pm$ 0.5	P < 0.05
LD lactate (mg.g <sup>-1</sup> )	LW	3.2 $\pm$ 0.3	3.1 $\pm$ 0.3	NS
	P	6.8 $\pm$ 0.6	3.9 $\pm$ 0.2	P < 0.01

The concentrations of glucose, FFA and lactate in the plasma are given in Table 3. Beta-blockade did not affect mean glucose or FFA levels in either breed but reduced variation in FFA in the Pietrains (P < 0.05). Plasma

lactate was not affected in Large Whites; in Pietrains the large increase seen in the control group at slaughter was prevented but the difference between the means just failed to reach significance although the variation was significantly (P < 0.05) reduced.

Table 3. The effect of beta-blockade on plasma glucose, FFA and lactate in Large White and Pietrain Pigs (means  $\pm$  SE)

	Breed	Control	$\beta$ -block	Significance of difference between means
Glucose (mg. 100 ml <sup>-1</sup> )	LW	105 $\pm$ 2	108 $\pm$ 2	NS
	P	102 $\pm$ 2	103 $\pm$ 2	NS
FFA (mg. 100 ml <sup>-1</sup> )	LW	10.2 $\pm$ 1.2	9.2 $\pm$ 0.8	NS
	P	10.0 $\pm$ 1.0	11.1 $\pm$ 0.4	NS
Lactate (mg. 100 ml <sup>-1</sup> )	LW	15.2 $\pm$ 1.7	16.6 $\pm$ 2.5	NS
	P	27.7 $\pm$ 5.3	16.0 $\pm$ 1.8	NS

#### Discussion

All the Large White pigs showed very good meat quality typical of a stress-resistant breed and beta-blockade had no effect. By contrast, the untreated Pietrains had very poor quality meat typical of a stress-susceptible breed and beta-blockade significantly improved it, based on lower temperature and higher pH<sub>45</sub> in the muscles which were less pale and had improved WHC leading to less drip loss in storage. Most British slaughter pigs would be expected to have meat quality between these two extremes exemplified by the Large White and Pietrain breeds and any improvement in meat quality by beta-blockade would therefore be expected to be related to the degree of stress-susceptibility exhibited by the genotype of pig under consideration.

The differences in pH<sub>45</sub> in the control and treated Pietrain groups were reflected in the concentration of muscle metabolites. Thus, beta-blockade prevented the rapid loss of muscle glycogen and increase in lactate shown by the control Pietrains.

The effect of catecholamines on the carbohydrate metabolism of the liver cannot clearly be ascribed to alpha or beta receptors although adrenergic blockade of both types will inhibit catecholamine-induced hyperglycaemia (Hornbrook, 1970). The large reduction in liver glycogen produced by beta-blockade in both breeds is therefore difficult to explain but might be due either to an enhancement of the alpha-agonistic effects of endogenous catecholamines through blocking the beta-receptors or to an entirely secondary effect. The mobilised carbohydrate was not reflected in raised blood glucose levels which were not elevated significantly above resting

levels in any group.

FFA, produced by increased sympathetic nervous activity, catecholamine secretion and associated lipolysis, are a major source of energy for muscles during exercise (Gollnick, 1967). In this experiment, FFA concentrations were relatively low in all pigs but this could reflect increased utilisation as well as production. Surprisingly, FFA were practically unaffected by beta-blockade however the results from the FFA analysis were verified independently using a different assay procedure. The lactacidaemia seen in the control Pietrains was prevented by beta-blockade.

In conclusion, beta-blockade, induced before a preslaughter transport stress, was effective in preventing the development of PSE meat in stress-susceptible pigs. It seems likely that the pigs in these experiments were effectively beta-blocked at slaughter as well as during the preceding transport and lairage. If a longer time had elapsed between injection and slaughter, through prolonged transport or lairage, the effectiveness of the adrenergic blockade may have decreased to the extent that, in the Pietrains, the metabolic events leading to poor meat quality were still triggered at slaughter.

Whilst there is no doubting the affinity of carazolol for beta-adrenergic receptors (Innis *et al*, 1979) some of the results observed may have been attributable to properties other than beta-blockade. It is well known that many of the so-called beta-blocking effects of propranolol are due to the drug's membrane stabilising and local anaesthetic properties and this is a possible cause of the effects demonstrated here.

#### Acknowledgements:

We would like to thank Praemix Wirkstoff GmbH, Mannheim, for financial assistance and S.C. Kestin, R.D. Lovell, L.J. Wilkins and S.N. Brown for help with the experiments.

#### References:

- Briskey, E.J., Bray, R.W., Hoekstra, W.C., Phillips, P.H. and Grummer, R.H. (1959) *J. Anim. Sci.* **18**, 146
- Brody, T.M. and McNeill, J.H. (1970) *Fed. Proc.* **29**, 1375
- Dole, V.P. and Meinertz, H. (1960) *J. Biol. Chem.* **235**, 2595
- Duncombe, W.G. (1963) *Biochem. J.* **88**, 7
- Fiebiger, E., Fiebiger, K., Nitz, K.J., Vollers, K. and Bartsch, W. (1978) *Tierarztl. Umschau.* **33**, 531
- Gollnick, P.D. (1967) *Am. J. Physiol.* **213**, 734
- Grau, R. and Hamm, R. (1953) *Naturwissenschaften* **40**, 29
- Gronert, G.A., Milde, J.H. and Taylor, S.R. (1980) *J. Physiol.* **307**, 319
- Hall, G.M., Lucke, J.N. and Lister, D. (1977) *Br. J. Anaesth.* **49**, 855
- Hornbrook, K.R. (1970) *Fed. Proc.* **29**, 1381
- Innis, R.B., Correa, F.M.A. and Snyder, S.H. (1979) *Life Sci.* **24**, 2255
- Lister, D., Hall, G.M. and Lucke, J.N. (1976) *Br. J. Anaesth.* **48**, 831
- Lister, D., Lucke, J.N. and Hall, G.M. (1977) *Proc. 3rd Int. Conf. Production disease in farm animals*, Pudoc, Wageningen. p 144
- MacDougall, D.B. and Jones, S.M. (1975) *Proc. 21st European Meeting Meat Research Workers*, Berne p. 113
- Sugden, M.C., Sharples, S.C. and Randle, P.J. (1976) *Biochem. J.* **160**, 817
- Topel, D.G., Wilson, D.G., Weiss, G.M. and Christian, L.L. (1973) *J. Anim. Sci.* **36**, 1077