The modifying effects of oxytocin on stress-induced changes in post-mortem muscle glycolysis in pigs

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Summary. The effect of ante-mortem i.m. administration of oxytocin on post-mortem muscle metabolism in Pietrain pigs was examined. Treatment with oxytocin significantly retarded post-mortem glycolysis and stopped the production of pale, soft, exudative meat.

Catecholamines, such as adrenaline and noradrenaline, are the principal stress hormones in many species of animals. The release and actions of catecholamines may be influenced by a number of factors, and it was to investigate the interaction between endogenously produced catecholamines and oxytocin that this study was undertaken.

Oxytocin is synthesized in the supraoptic and paraventricular nuclei of the hypothalamus and released from the posterior lobe of the pituitary body. Release of oxytocin is under the control of the central nervous system and is effected by catecholamines. One of the most important situations in which oxytocin is secreted is during suckling[1] and it is well established that oxytocin release[2] and milk ejection[3] are not observed under stressful conditions. Furthermore, it is also known that the effects of exogenous oxytocin are markedly reduced by physiological levels of adrenaline such as are found in stress[4]. There is, therefore, some circumstantial evidence of an interrelationship between oxytocin, catecholamines and stress. The effects of oxytocin on the actions of stress-induced catecholamines were, therefore, studied in pigs.

A well-known effect of pre-slaughter stress in pigs is the occurrence of pale, soft, exudative (PSE) meat in the carcases of the pigs post-mortem. This condition is more prevalent in the carcases of certain breeds of pig[5] and these are often called stress-susceptible breeds. Pigs of the Pietrain breed tend to be of the stress-susceptible type and produce PSE meat. This is believed to be a result of an enhanced sensitivity of the autonomic nervous system, producing a higher potential noradrenaline output to stimulation of the sympathetic nervous system[6].

In this study the effect of administration of oxytocin on the post-mortem metabolism and ultimate meat quality of Pietrain pigs was examined.

Materials and methods. 22 pork weight Pietrain pigs (68 ± 10 kg live weight) were used. 10 pigs (6 males and 4 females) were given oxytocin (Sigma) i.m. 5 min before slaughter and 12 pigs (7 males and 5 females) received saline i.m. The oxytocin-treated pigs received approximately 2.5 units/kg, (150–200 units) of oxytocin in a volume of 0.5 ml. The control pigs received a similar volume of saline. 5 min after treatment the pigs were electrically stunned and exsanguinated under normal commercial slaughter conditions. Samples of muscle were taken from M. longissimus dorsi at 5 min, 45 min and 24 h after death and tested for pH, glycogen and lactate levels. A sample was taken at 24 h after death for estimation of drip loss.

The muscle pH was determined by macerating approximately 1 g of muscle in 10 ml 5 mM sodium iodoacetate (pH 7.0) containing 0.15 M KCl. The pH of the resultant homogenate was measured using a Radiometer pH meter. Glycogen was measured by a modification of the caustic digestion and alcoholic precipitation method[7]. Approximately 0.5 g of weighed muscle were macerated in 5 ml of 3% perchloric acid and centrifuged. The lactate levels in the resultant supernatants were determined using the method of Gutmann and Wahlefeld[8]. Drip loss was measured from slices of longissimus dorsi muscle removed 24 h after death, according to the procedure described by Taylor and Dant[9], and plasma catecholamines were determined fluorimetrically[10].

Results and discussion. The accumulation of lactic acid in the muscle of the oxytocin-treated pigs was less than in the saline treated pigs (table), and the final concentration of lactate in the muscle at 24 h post-mortem was significantly higher in the saline treated controls ($p < 0.01$). This difference in acidification produced significant changes in the pH of the muscles at both 45 min ($p < 0.01$) and 24 h ($p < 0.01$) post-mortem; the pH being higher in the oxytocin-treated pigs. A more direct measurement of post-mortem muscle glycolysis, i.e. muscle glycogen content, showed no significant differences between the 2 groups; however, the rate of glycogen loss was significantly reduced in the oxytocin-treated pigs ($p < 0.001$).

As a direct measurement of meat quality, the loss of water as drip was measured. The drip loss was also significantly reduced in the oxytocin-treated pigs ($p < 0.05$). There was no difference in any measured parameter between males and females in either the treated or control groups.

The results of this experiment show that treatment with oxytocin (albeit in high doses) can significantly slow the rate of post-mortem glycolysis in Pietrain pigs. The effect was sufficient that none of the carcases in the oxytocin-treated group exhibited PSE meat. Preliminary studies in our laboratory however, indicated that lower doses of oxytocin ($\leqslant 50$ units) had less effect on the slowing of post-mortem metabolism.

Fluorimetric analysis of slaughter blood for catecholamines showed no difference in the levels of either adrenaline (14.1 ± 2.5 µg/l and 16.1 ± 4.2 µg/l) or noradrenaline

The pH and lactate levels at 5 min, 45 min and 24 h post-mortem, the changes in glycogen between 5 and 45 min post-mortem and the drip loss in longissimus dorsi of oxytocin-treated and control pigs expressed as the mean ± SEM

	pH			Δ glycogen (mg/g)	Lactate (µmole/g)			Drip
	5 min	45 min	24 h	5–45 min	5 min	45 min	24 h	(%)
Oxytocin	6.68 ± 0.08	6.39 ± 0.11	5.79 ± 0.02	0.275 ± 0.37	39.93 ± 2.93	64.62 ± 8.19	84.97 ± 3.75	10.66 ± 0.99
Control	6.57 ± 0.06	5.96 ± 0.07	5.61 ± 0.05	2.171 ± 0.12	50.46 ± 3.43	88.33 ± 5.22	106.62 ± 3.93	13.96 ± 0.90
n	22	22	19	22	17	18	19	17
p	n.s.	**	**	***	*	*	**	*

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ by Student's t-test.

(28.8±5.2 μg/l and 28.6±8.5 μg/l) between the oxytocin and saline-treated pigs respectively. Thus, if PSE meat is the result of a stress-induced catecholamine release, this finding suggests that oxytocin modifies the action of the catecholamines rather than their release.

Malignant hyperthermia is a well-documented reaction which occurs in stress-susceptible pigs following exposure to halothane anaesthesia. It has been proposed that the mechanisms which produce malignant hyperthermia (irreversible lacticacidosis) are similar to those which bring about PSE meat[11]. In 2 pigs which were given oxytocin during an established hyperthermic reaction (that is, the lacticacidosis was sufficient that the blood pH was less than 7.00) the reaction continued its usual fatal course. However, in 1 pig which was given oxytocin after halothane, but before the blood pH reached the critical level of 7.00, the reaction was halted (personal observations).

The physiological implications of these findings require further investigations of the effects of oxytocin and catecholamines on receptors in skeletal muscle, but may provide a useful tool for the investigation of the PSE condition in pig meat.

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Effect of continuous light on the blood and urinary sugar levels in alloxan diabetic rats

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Summary. Continuous light decreases the rise in blood glucose and the excretion of urinary glucose, along with increased urine volume, in alloxan diabetic rats.

It is now well known that adrenocortical hypersecretion[2-4] and hyperglycemia[5] commonly accompany the onset of alloxan diabetes. Exton et al.[6] have noted that adrenalectomy reduces, and glucocorticoid treatment restores, glucose output from liver in alloxan diabetic rats. Since continuous exposure to light inhibits normal secretion of adrenocortical hormones[7] and appears to counteract the adrenocortical hyperactivity in the alloxan diabetic rat[8,9] the present experiment has been undertaken to determine whether constant illumination can prevent hyperglycemia in alloxan diabetes.

Materials and methods. 24 adult female rats maintained at 34 °C with 10 h illumination daily were used. The rats were fed a diet of 'Hindlever Rat Chow' and water ad libitum. Samples of urine were collected every 24 h in flasks from all the rats with 0.5 ml of a mixture containing 1.2% each of penicillin and streptomycin, saturated with thymol to avoid bacterial contamination of the urine. Urine volumes were measured at the end of the 24-h period. 18 rats on 48 h fasting were given a single i.m. injection of alloxan (BDH, London) at a dose level of 7 mg/100 g b.wt, and the remaining 6 control rats received a measured amount of saline. After 3 days blood was obtained from all the rats by cardiac puncture under anesthesia. Blood and urinary glucose content were estimated by the methods of Folin and Wu[10], and Benedict[11] respectively. 12 alloxanized rats with exhibited a blood sugar level within the range of 450–500 mg/100 ml were divided equally into 2 groups. 6 alloxan treated rats were then exposed to continuous light. Illumination (40 W) was provided by an overhead fluorescent bulb (cool white). 6 other treated animals, and 6 controls, were kept in LD (light-darkness) cycles of 10:14 h. After 14

Table 1. Effect of continuous light on the blood glucose of alloxan diabetic rats

Treatment	Blood glucose after 3 days mg%	Blood glucose after 17 days mg%	Adrenal wt mg/100 g body weight	Initial body weight	Body weight after 17 days
Control	101.02±1.61*	98.45±0.67	15.81±0.32	147.51±0.80	169.04±0.61
Diabetic	457.23±1.68	664.18±3.91	28.97±0.58	147.42±0.42	133.98±0.51
Diabetic+light	462.51±2.10	349.30±2.51	18.49±0.36	147.33±0.58	153.63±0.35
p-value:					
Control vs diabetes	<0.001	<0.001	<0.001	NS	<0.001
Control vs diabetes+light	<0.001	<0.001	<0.01	NS	<0.001
Diabetes vs diabetes+light	NS	<0.001	<0.001	NS	<0.001

* Mean±SE; NS, indicates statistically not significant.