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Influence of Aldosterone on pale, soft, exudative (PSE) porcine muscle[1]

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Researchers have implicated various hormonal mechanisms in the physiological and biochemical etiology of PSE porcine musculature. Ludvigsen (1954) suggested, since this condition is more prevalent in breeds which have had intensive selection for muscling, perhaps there has been automatic selection for a high content of growth stimulating hormone and a lower content of thyroid stimulating and adrenocorticotropic hormone. Ludvigsen (1953), Briskey (1963) and Judge *et al.* (1965) suggested the thyroid hormone was related to post-mortem muscle properties. Topel and Merkel (1966) failed to confirm this hypothesis, but noted a decrease in adrenal gland weight. These workers concluded that the development of PSE musculature was not associated with hypothyroidism *per se*; but that it may be the indirect result from the secondary effects of methylthiouracil upon the adrenal gland.

Two different classes of hormones are produced by the adrenal gland: catecholamines by the medulla and steroids by the cortex.

Radouco-Thomas (1962) and Trautman *et al.* (1963) showed that adrenalin prevented the development of the PSE condition. Topel (1968) suggested an interaction between catecholamines and glucocorticoids on microcirculatory integrity as related to PSE muscle. Research efforts have focused on the glucocorticoids with regard to the steroid influence on PSE musculature. Topel *et al.* (1967) reported slightly lower level of plasma 17-hydroxycorticosterone in pigs with severe PSE musculature. However, Topel and Merkel (1967) failed to produce PSE muscle when using prednisolone and methylprednisolone to obtain adrenal atrophy and lower 17-hydroxycorticosterone levels. Mineralocorticoids have been given little consideration in relation to PSE muscle. Henry *et al.* (1958) reported that pigs with PSE musculature had hyperproduction of aldosterone. However, Topel and Merkel (1966) and Topel *et al.* (1967) conducting research closely allied but not directly related to this problem failed to support the findings of Henry. Upon close

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examination of the physiological role of aldosterone, it was postulated that aldosterone could be responsible for a rate limiting factor, specifically the potassium dependence of pyruvic acid kinase, resulting in predisposing factors which have been shown to be associated with PSE musculature. Therefore, studies were undertaken to determine if PSE musculature could be prevented using an aldosterone blocking agent and/or produced using aldosterone.

MATERIALS AND METHODS

In this study, eight Poland China hogs, approximately 90 kg. were used. Three were given an oral drench of 500 mg. of aldactazide, which acts as a competitive inhibitor at the site of action of aldosterone. After a 30 minute equilibration period, the hogs were stressed for 30 minutes using an electric prod, and slaughtered immediately. The one control animal received the same stress treatment 30 minutes after drenching with distilled water. Four hogs were injected intravenously with 1.5 mg. of aldosterone dissolved in 10 ml. of physiological saline. These hogs were allowed to remain undisturbed for a 1 ½ hr. equilibration period, and then slaughtered immediately. After a 24 hr. chill at 3° C, all carcasses were cut and a quality score applied according to the Wisconsin Pork Quality Standards (Anon., 1963).

RESULTS AND DISCUSSION

It was observed in carcasses from animals that received a drench of 500 mg. of aldactazide followed by 30 minutes of stress using an electric prod that the muscles of the ham were uniform in color, firm in texture and dry in appearance. The *longissimus dorsi* muscle appeared to have a slight amount of moisture on the surface, but it was not excessive. Animals stressed for 30 minutes with the electric prod after receiving a drench of distilled water produced carcasses having less desirable quality attributes as observed in the face of the ham and *longissimus dorsi*. The muscles were lighter in color

Table 1. *Means and standard deviations for subject quality evaluation.*

Item	*Aldosterone*		*Control*		*Aldactazide*	
	Mean	*S.D.*[a]	*Mean*	*S.D.*	*Mean*	*S.D.*
Wisc. Qual. Score[b]	1.38	±0.83	1.0	±0.0	3.0	±1.0

a Standard deviation.
b Wisconsin Pork Quality Score PSE=1, Normal=3.

with muscle separation being evident and the *longissimus dorsi* exhibited excessive exudation. These carcasses were therefore classified as exhibiting PSE quality. Also, animals receiving a 1.5 mg. injection of aldosterone immediately prior to slaughter produced carcasses exhibiting PSE condition in that muscles in the face of the ham lacked uniformity of color, were extremely soft, had an open structure and were very watery. The *longissimus dorsi* muscle exhibited conditions characteristic of PSE pork muscles, also.

Table 1 contains the means of the subjective quality evaluation for the various treatments. Mean score for animals receiving aldactazide was 3.00, while the score for the control animal was 1.00. Likewise, animals which received aldosterone had a mean quality score of 1.38.

When considering the possible influence of aldosterone on PSE muscle, it must be remembered that the primary function of aldosterone is to regulate sodium and potassium retention and excrertion by the renal tubules of the kidney. In the reversal of glycolysis to form glucose-6-phosphate, adenosine triphosphate (ATP), available from the aerobic oxidation of a fraction of lactic acid (via pyruvic acid and the citric acid cycle) must be provided to reverse the pyruvic acid kinase and phosphoglyceric kinase reactions. However, there is a rate limiting factor, pyruvic acid kinase is potassium dependent. The occurrence of PSE muscle is associated with a rapid rate of pH decline while the muscle is at a high temperature immediately post-mortem (Sayre and Briskey, 1963) . At this time in muscle, there is a certain amount of aerobic glycolysis occurring; therefore, oxidative phosphorylation and the production of ATP. Perhaps it is possible that in normal muscle sufficient potassium is present for the pyruvic acid kinase reaction, thereby reversing glycolysis. This allows a portion of the lactic acid from preslaughter exercise, excitement, etc. to go back into the Embdem-Meyerhoff pathway as intermediate compounds, thereby decreasing the rate of pH drop post-mortem. In PSE muscle, hyperproduction of aldosterone is believed to cause an increased excrertion of potassium, thereby inactivating the pyruvic acid kinase reaction which prevents the reversal of glycolysis. This allows the preslaughter lactic acid to remain, plus that lactic acid which normally accumulates during post-mortem glycolysis, resulting in a more rapid drop of pH at a high temperature. Marple *et al.* (1968) support this postulation by concluding that the adrenal insufficient pigs, produced by prednisolone injection, may be unable to adequately remove high levels of lactate from the blood and that low levels of plasma 17-hydroxycorticosterone *per se* do not serve to increase the rate of post-mortem anaerobic glycolysis.

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