INFLUENCE OF INDUCED ADRENAL INSUFFICIENCY AND STRESS ON PORCINE PLASMA AND MUSCLE CHARACTERISTICS

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Research on the response of pigs to various stressors has led to the use of the terms "stressor-susceptible" and "stressor-resistant." (Judge, Briskey and Meyer, 1966; Judge, Cassens and Briskey, 1967; Judge et al., 1968; Topel et al., 1968). An acute, shock-like, often fatal condition in stress-prone swine has been described by Topel et al. (1968) and was attributed to an acidosis condition. Similar responses to mild stressors have been described by Ludvigsen (1953, 1957) who attributed the shock conditions to circulatory insufficiency and hyperthermia. These stress-prone animals, suggested to possess insufficient levels of adrenal glucocorticoids often yield carcasses exhibiting pale, soft, exudative (PSE) musculature (Ludvigsen, 1957; Henry, Romani and Jouhert, 1958; Judge et al., 1966). Such carcasses have been shown to have high levels of muscle lactic acid at death and to undergo a rapid rate of postmortem anaerobic glycolysis (Forrest et al., 1968).

This study was designed to (1) observe the influence of induced adrenal glucocorticoid insufficiency on the ability of pigs to withstand short term physical stress and (2) to determine the interrelationships of plasma 17-hydroxycorticosteroids, plasma and muscle lactic acid and muscle glycogen content on pork muscle quality.

Materials and Methods

Twenty-four barrows and gilts (81 to 107 kg.) of Yorkshire and Yorkshire x Yorkshire hybrid breeding were randomly assigned to one of three groups. The group I pigs (controls) received no injections, while pigs of groups II and III (adrenal insufficient, non-stressed and stressed, respectively) received 100 mg. of prednisolone (i.m.) daily for 10 days and were slaughtered 48 hr. after the last injection. The group III pigs were physically exercised for 5 min. before exsanguination. Animals in group III that collapsed before the 5 min. exercise period ended were sacrificed immediately. Pigs in groups I and II were not physically exercised before slaughter but were subjected to the normal stress conditions usually encountered in slaughter.

Immediately after sticking, blood samples were collected in 250 ml. glass centrifuge tubes containing ammonium heparin and were immediately centrifuged at 8,000 rpm for 20 min. in a refrigerated centrifuge. The plasma was decanted into polyethylene bottles, sealed and stored at -12°C until assayed. Muscle samples for initial pH and color of the M. longissimus were removed from the right fifth or sixth lumbar region of the unscaled carcass immediately after the animal ceased struggling, frozen in a dry ice-acetone bath and stored at -12°C. A similar sample of the M. longissimus was obtained 24 hr. postmortem.

Adrenal glands were isolated and weighed, and the M. longissimus pH was determined on serial samples taken at death and every hour for the next 4 hr. by the method reported by Topel et al. (1966). The pH of the homogenate was determined with a Beckman Zeromatic pH meter with glass electrodes. Objective measurement of muscle color intensity was determined by using a Photovolt photoelectric reflection meter, Model 610 standardized against magnesium oxide. Muscle and plasma lactate were determined according to the method of Hohorst (1963). Plasma pyruvate was determined as described by Sigma Chemical Co. (1966). Muscle glycogen was evaluated using the method of DuBois et al. (1956) and plasma 17-OHCS levels were examined by using the procedure of Peterson, Karrer and Guerra (1957) as modified by Topel et al. (1966).

Blood pH was determined on approximately a 10 ml. aliquot of whole blood taken at the time of exsanguination and measured by using a Beckman Expanded Scale pH meter with glass electrodes. Precautions to prevent pH
TABLE 1. EFFECT OF STRESS AND PREDNISOLONE TREATMENT ON THE ADRENAL GLAND AND BLOOD CHARACTERISTICS

<table>
<thead>
<tr>
<th>Observations</th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenal gland, gm.</td>
<td>Mean SE</td>
<td>Mean SE</td>
<td>Mean SE</td>
</tr>
<tr>
<td>left</td>
<td>2.10 ± 0.44</td>
<td>1.70 ± 0.23</td>
<td>1.65 ± 0.19</td>
</tr>
<tr>
<td>right</td>
<td>1.96 ± 0.43</td>
<td>1.54 ± 0.26</td>
<td>1.53 ± 0.16</td>
</tr>
<tr>
<td>Plasma 17-OHCS, μg/100 ml.</td>
<td>12.31 ± 5.75</td>
<td>6.06 ± 3.54</td>
<td>6.41 ± 4.12</td>
</tr>
<tr>
<td>Plasma lactate, umoles/ml.</td>
<td>13.36 ± 3.10</td>
<td>15.52 ± 4.98</td>
<td>27.17 ± 6.46</td>
</tr>
<tr>
<td>Blood pH</td>
<td>7.41 ± 0.11</td>
<td>7.43 ± 0.07</td>
<td>6.97 ± 0.06</td>
</tr>
</tbody>
</table>

a, b Means for each characteristic with different superscripts changes as a result of exposure to air and temperature were taken.

Analysis of variance was determined on the data, and if treatment effects were significantly different, orthogonal comparisons were calculated. Within and pooled treatment correlation coefficients were determined as described by Snedecor and Cochran (1967).

Results and Discussion

Four of the eight animals in group III (stressed, adrenal insufficient) succumbed to the exercise and produced PSE carcasses. During exercise, the pigs showed increasing signs of dyspnea and developed marked cyanotic blotches in the flank, side, shoulder and base of ham regions indicating a circulatory abnormality within the dermal tissue.

A significant (P<.05) reduction in weight was noted for left and right adrenal glands of the experimental groups from the control pigs (table 1). The reduction in adrenal weight due to the injection of prednisolone indicated that adrenal atrophy was induced, which is in agreement with the work of Topel and Merkel (1967) and Nichols and Tyler (1967). Also plasma 17-OHCS levels were significantly lower among the groups II and III pigs than in the controls, indicating further that partial adrenal insufficiency was induced (table 1).

Plasma lactate values at death were significantly (P<.01) higher in the group III pigs (table 1). The comparison of plasma lactate values between groups I and II revealed no significant prednisolone treatment differences, and the high level of the group III individuals may be attributed to the combined effect of physical exercise and low plasma 17-OHCS levels.

Plasma pyruvate levels were not significantly different between treatments, although a slightly higher level was noted for the pigs in group III (table 1), which coincides with the increased plasma lactate levels noted.

The blood pH was significantly (P<.01) lower for the group III than for groups I or II (table 1). This effect may be due to the pre-slaughter exercise and induced adrenal insufficiency and could be explained by impaired

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Figure 1. M. longissimus lactate and glycogen levels.
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TABLE 2. RELATIONSHIP BETWEEN SOME CHEMICAL AND PHYSICAL CHARACTERISTICS OF PORCINE PLASMA AND MUSCLE

<table>
<thead>
<tr>
<th>Observation</th>
<th>Sample</th>
<th>Group I</th>
<th>Group II</th>
<th>Group III</th>
<th>Pooled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle color</td>
<td>Initial</td>
<td>0.62</td>
<td>-.44</td>
<td>0.25</td>
<td>0.03</td>
</tr>
<tr>
<td></td>
<td>1 hr.</td>
<td>-.77*</td>
<td>-.79*</td>
<td>-.38</td>
<td>-.67*</td>
</tr>
<tr>
<td>M. longissimus pH</td>
<td>24 hr.</td>
<td>-.50</td>
<td>-.22</td>
<td>-.64</td>
<td>-.47*</td>
</tr>
<tr>
<td>Plasma lactate vs. Plasma Pyruvate</td>
<td>Initial</td>
<td>0.85**</td>
<td>-.09</td>
<td>-.04</td>
<td>0.27</td>
</tr>
<tr>
<td>M. longissimus lactate vs. M. longissimus pH</td>
<td>Initial</td>
<td>-.13</td>
<td>-.85*</td>
<td>-.67</td>
<td>-.55**</td>
</tr>
<tr>
<td>M. longissimus lactate vs. M. longissimus glycogen</td>
<td>24 hr.</td>
<td>0.46</td>
<td>-.34</td>
<td>0.71*</td>
<td>0.02</td>
</tr>
</tbody>
</table>

* P<.05.  ** P<.01.

conversion of blood lactate to blood glucose or glycogen in the liver during exercise (White et al., 1959).

The initial lactic acid content of the post-mortem M. longissimus (figure 1) was significantly lower (P<.05) for the group II pigs than the controls or the group III pigs. This further indicates that low levels of plasma 17-OHCS per se are not highly associated with muscle lactate content, but the plasma 17-OHCS levels appear to influence lactate metabolism under stress conditions such as the physical exercise given to the group III pigs.

The marked increase in muscle lactate in the stressed, adrenal insufficient group is postulated to be the result of an inability of the group III animals to withstand the effects of preslaughter exercise and may represent an impairment of the animal's ability to remove tissue lactate as rapidly as it is formed. Since Keul, Keppler and Doll (1967) assumed an immediate equilibration of lactate concentrations between the cytoplasm and extracellular fluid, it may be reasonable to assume that the glucocorticoid influence on reducing plasma lactate (Ramey and Goldstein, 1957; White et al., 1959) would apply to the homeostatic mechanisms within the musculature and lactate metabolism in other organs such as the liver. No significant differences in 24-hr. post-mortem muscle lactate levels were noted (figure 1), indicating that the ultimate amount of lactate produced is not dependent upon the initial lactate levels. The lactic acid levels of the M. longissimus from the initial and 24-hr. samples were significantly (P<.01) correlated (table 2) with their respective muscle pH values (r1=-.55, r24=-.58) when pooled correlation coefficients were calculated over all treatments. This is in partial agreement with the work of Gunther and Schweiger (1966) who stated that it was not always possible to detect any significant correlation between the lactic acid content and the pH of muscle samples taken at the same time.

Initial glycogen levels of the M. longissimus were significantly higher (P<.05) in the animals of groups II and III when compared to group I (figure 1) and was attributed to the prednisolone injections. No significant differences were observed between groups II and III. Similar effects of prednisolone injection were noted by Aberle and Merkel (1968). It is proposed that, during the prednisolone injections, lactate and other precursors of liver glycogen may undergo metabolic changes, resulting in increased blood glucose levels...

Figure 2. M. longissimus reflectance. Higher values denote lighter color.
As a result, increased muscle glycogen could be deposited from blood glucose, as described by Glenn *et al.* (1961). Significant treatment differences in 24-hr. muscle glycogen were noted when groups II and III were compared (figure 1). Since the 24-hr. levels of lactic acid and initial glycogen levels were not significantly different for groups II and III, the lower ultimate glycogen level for the group III individuals may indicate that the muscle glycogen has been broken down to some metabolic intermediate between glycogen and lactic acid. Also, the level of initial muscle glycogen observed in this study did not appear to influence the ultimate production of lactic acid by postmortem anaerobic glycolysis (figure 1). This is in accord with work by Sayre, Briskey and Hoekstra (1963) who could not account for all the glycogen broken down in the corresponding accumulation of lactic acid.

No significant treatment differences in muscle color or pH were noted (Figures 2 and 3). Similarly, no significant differences were noted in the rate of pH decline among treatments, although the number of carcasses developing PSE type musculature was higher in group III.

**Summary**

Injecting 100 mg. prednisolone daily for 10 days resulted in atrophy of the adrenal gland. The animals were stressed 48 hr. after receiving the last prednisolone injection and then sacrificed. Susceptibility to stress was increased in the partial adrenal insufficient pigs determined by their inability to withstand 5 min. of physical exercise before slaughter. Plasma lactate levels were increased significantly \( P < .05 \) due to the effect of the physical exercise, and blood pH at death was significantly decreased among the stressed adrenal insufficient group. Initial lactic acid content of the \( M. \ longissimus \) was significantly lower in the group two pigs when compared to group three. No significant differences were noted among 24-hr. muscle lactate levels. The glycogen content of the \( M. \ longissimus \) was significantly increased because of prednisolone injections, but the level of initial muscle glycogen did not appear to influence the ultimate amount of lactic acid produced by postmortem anaerobic glycolysis. No significant differences were noted among treatments when \( M. \ longissimus \) pH values and color scores were examined statistically. Low levels of plasma 17-OHCS per se...
did not serve to increase the rate of postmortem anaerobic glycolysis. The partial adrenal insufficient pigs used in this study may be unable to adequately remove high levels of lactic acid from the blood and muscle during exhaustive exercise.

**Literature Cited**


