**Interaction of Porcine Somatotropin Administration to Growing Pigs and Frozen Storage of Carcass on Lipids and Quality Characteristics of Roasts**

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***Note: Figures may be missing from this format of the document***

**Abstract:**
The purpose of this study was to evaluate the effect of porcine somatotropin (pST) treatment on the lipid content and fatty acid profile of cooked pork roasts and to determine if differences were exacerbated by extended frozen storage. Pigs were injected with 3 mg porcine somatotropin (pST) or a placebo daily. At slaughter, roasts were cut from the second to tenth ribs, frozen (-20°C) for 2.5 weeks or 4 months, then cooked and evaluated. Roasts from pST-treated pigs had a lower content of lipids than did the placebo-treated pigs, with the saturated fatty acids decreasing about 40-1%. They also demonstrated greater Warner-Bratzler shear values. Cholesterol content of the roasts stored frozen for 4 months decreased slightly. Four-month storage slightly decreased cooking time, cooking losses, and expressible moisture index and increased moisture, typical flavor, and juiciness of the roasts. These data suggest that pST treatment altered fat favorably when viewed in light of current dietary recommendations but had less effect on meat quality than did extended frozen storage.

**Article:**
**INTRODUCTION**
The consumption of dietary fat, especially saturated fat and cholesterol, has been implicated as contributing to the development of cardiovascular disease. Current dietary recommendations suggest that the American public reduce its intake of fat from the current value of 36% of the total calories to 30% (NCEP, 1990; NRC, 1989). In addition, it is suggested that the intake of saturated fat and cholesterol be decreased while that of polyunsaturated fat remain unchanged. This has led to decreased consumption of red meats by the health-conscious consumer because they consider them to be high in total and saturated fat and cholesterol (Sweeten et al., 1990).

As a consequence the meat industry is evaluating techniques to produce meat and meat products with altered lipid concentration and fatty acid profiles. One effective tool for altering tissue fatty acid content in nonruminant animals is by modifying diet (Villegas et al., 1973; Skelley et al., 1975; St. John et al., 1987; Miller et al., 1990). Although dietary modifications may alter fatty acid profile and cause quality changes, these changes are produced without a concomitant lowering of total lipid content. Another method being tried is the administration of recombinant porcine somatotropin (pST). With the use of existing recombinant DNA techniques, pST can be
mass-produced, thus enhancing its potential usefulness to the pork industry. It is currently approved by the FDA for use as an experimental drug. Its use has been shown to decrease carcass fat and increase lean body tissue in swine (Machlin, 1972; Campbell et al., 1989; Chung et al., 1985; Etherton et al., 1987; Evock et al., 1988; Gardner et al., 1989). Few investigations have addressed its potential to change the fatty acid profile. Prusa et al. (1989a) showed an increase in the level of the sum of polyunsaturated fatty acids (PUFA) but did not report changes in individual fatty acids, while Lonergan et al. (1992) showed no changes in fatty acid profile from seven carcass locations. As it becomes increasingly clear that each fatty acid has unique physiological properties (Grundy and Denke, 1990), such information becomes more valuable.

However, as the lipid profile of pork is altered, quality characteristics and frozen storage shelf life may also be affected. Although it is generally recognized that greater flavor is found in pork with a higher fat content (Martel et al., 1988), the impact of modification of its fatty acid profile on acceptability is contradictory. St. John et al. (1987) and Skelley et al. (1975) found feeding soybeans or canola oil to swine produced no differences in sensory characteristics of muscle. On the other hand, Shackelford et al. (1990a,b,c) and Miller et al. (1990) observed that diets high in monounsaturated fatty acids, especially canola oil, sometimes produced less acceptable pork and pork products when tasted by a trained sensory panel. Storage of highly unsaturated tissue is characterized by increased oxidative rancidity (German, 1985).

The effect of frozen storage on quality characteristics is also contradictory. Oxidized lipids cause protein polymerization, insolubilization, polypeptide chain scission, amino acid destruction, and formation of addition products (Kanner and Karel, 1976; Funes et al., 1982). These lipid-protein interactions may, in turn, alter the functional properties of meat and cause deleterious changes in final product quality. However, West and Myer (1987) found when fat in swine was altered by the addition of peanuts to the diet, there was no effect on sensory evaluation or values for thiobarbituric acid reactive substances after 4 months of frozen storage.

Although considerable information about quality characteristics of swine given somatotropin has been generated over the past year (Prusa et al., 1989a,b, 1990; Halloran et al., 1991; Hagen et al., 1991; Kanis et al., 1990; Boles et al., 1991), limited research has been conducted on the possible interaction of storage and pST administration on these variables. Consequently, the purpose of this study was to determine the effect of the daily administration of recombinant pST to barrows on total lipid and cholesterol content, fatty acid profile, and quality characteristics of cooked loin roasts after short (2.5 weeks) and lengthy (4 months) frozen storage (-20°C).

MATERIALS AND METHODS

**Animals**

Sixteen commercial barrows of a meat-type crossbreed (exact genetic background unknown) were obtained (1989-1990) from Bischof Pig Farm (Sherwood, OR). The pigs were randomly assigned to one of two groups, treatment or control. The pigs were housed so that two pens contained control animals and two contained treated animals. The initial weight of the control group was 54 ± 2.2 kg; that of the treatment group was 54 ± 1.4 kg. Both groups received daily injections. The treated pigs were injected with 1 ml of 3 mg recombinant pST in buffer (0.025 M NaHCO₃, 0.025 M Na₂CO₃; Pitman-Moore, Terre Haute, IN). Control pigs were injected with 1 ml buffer alone. The injection site was the subcutaneous fat posterior to the base of the ear,
alternating from left to right sides daily. Approval to use swine for this study was obtained from Oregon State University's Animal Use and Care Committee. Normal husbandry practices with minimum pain to the animals were employed.

All animals were fed a corn—soybean meal-based diet formulated to contain 15.13% moisture, 17.65% crude protein, 1.15% lysine, 4.05% fat, 0.63% calcium, 0.84% phosphorous, and 0.71% potassium. Since an increase in lean tissue deposition was expected, the diet was formulated to contain lysine at 0.6% excess of the requirements for pigs (NRC, 1988).

**Sample Preparation**

Pigs were slaughtered when they reached market weight. This value averaged 106 kg, ranging from 96 to 112 kg. Loin roasts, trimmed to 0.64 cm fat cover, were cut between the second and tenth rib 48 hr postmortem, individually wrapped in film-lined freezer paper, placed in freezer bags, and immediately stored at —20°C until used. Roast weights ranged from 1.67 to 2.70 kg. Samples from the right side were stored frozen no longer than 2.5 weeks before analysis. Samples from the left side were stored for 4 months before analysis. The frozen storage was designed to mimic conditions similar to those a consumer would use.

At the time of analysis, roasts were defrosted at 3°C for 48 hr. The roasts were then roasted at 191°C in an electric oven (Kenmore Self-Cleaning Oven, Series 64491, Sears Roebuck and Co., Chicago, IL) to a final internal temperature of 85.0°C. Internal temperatures were monitored at the center of the longissimus dorsi muscle with a Leeds and Northrup W12 Temperature Recorder (Leeds and Northrup, Portland, OR). Total cooking time was measured and heating rates (°C/g) were calculated. Total and drip cooking losses were calculated using cooking weight loss and drip weight for each pork loin roast. Triplicate core samples of approximately 20 g were removed from a 2.50-cm center slice of a cooked roast which had been cooled to room temperature (2°C/min), frozen in liquid N2, and powdered in a kitchen blender (Zondagh et al., 1986). Powdered samples were stored in glass jars at —40°C until further analysis.

**Moisture**

Total moisture in the samples was determined on duplicate 5 g cooked powdered samples prepared from the core samples according to the AOAC (1990) vacuum-oven method 950.46. The method of Wierbicki and Deatherage (1958) was used to determine the water-holding capacity (expressible moisture index, EMI) of cooked meat. EMI values were calculated as the ratio of the mean pressed meat and mean juice areas.

**Lipids**

Fat was extracted from cooked powdered samples prepared from the core samples following the method of Bligh and Dyer (1959) using methanol and chloroform. Total lipids were measured gravimetrically on an aliquot of this extraction. A second aliquot of the fat extract was used to determine the fatty acid profile according to previously reported procedures (Song and Wander, 1991). Heptadecanoic acid was used as an internal standard so that data could be expressed in gravimetric units. The data were also given on a weight-percentage basis to allow comparison to similar values in the literature.
Cholesterol content was determined on powdered muscle samples. The samples were saponified in alcoholic potassium hydroxide according to the procedure of Kovacs et al. (1979) and extracted with hexane. The samples were then derivatized with 0.20 ml bis(trimethylsilyl)trifluoracetamide (BSTFA) plus 1% trimethylchlorosilane (TMCS) in pyridine. Derivatization occurred immediately at room temperature. The derivatized samples were analyzed using a Hewlett—Packard 5890 gas chromatograph equipped with a 30 m X 0.245 mm i.d., 0.15-μm film thickness DB-17 column (J and W Scientific, with a 30 m X 0.245 mm i.d., 0.15-μm film thickness DB-17 column (J and W Scientific, Folsom, CA). Helium was the carrier gas at a flow rate of 2 ml/min and a split ratio of 1:55. Hydrogen and air flow rates were 35 ml/min and 400 ml/min, respectively. The injector and detector temperatures were 245 and 250°C, respectively. The column oven was operated isothermally at 240°C. For quantitation of cholesterol, 5a-cholestane was added as an internal standard prior to saponification. Sterols were identified by comparing to authentic sterol standards (brassicasterol, stigmasterol, campesterol, sitosterol, and cholesterol). Cholesterol was the only sterol found. Recovery of cholesterol and its esters was determined by adding cholesterol and cholesteryl palmitate to samples prior to saponification. Recovery for free cholesterol and cholesteryl palmitate was 98 and 95%, respectively. BSTFA plus 1% TMCS was obtained from Regis Chemical Co. (Morton Grove, IL). The authentic sterol standards were obtained from Supelco, Inc. (Bellefonte, PA) and NuChek Prep, Inc. (Elysian, MN). Cholesteryl palmitate was obtained from NuChek Prep. The 5a-cholestane was obtained from Matreya, Inc. (Pleasant Gap, PA). All other chemicals were reagent grade.

Physical Testing
Triplicate core samples running parallel to the muscle fiber direction and 1.27 cm in diameter were taken from the center of a 2.54-cm slice of each longissimus dorsi muscle from each roast. Shear force (kg/1.27 cm) was determined on a WarnerBratzler shear apparatus (25 kg X 50 g dynamometer scale, G. R. Electric Mfg. Co., Manhattan, KS). The color of an inner slice of cooked muscle was measured with a Hunter LabScan Reflectance Spectrophotometer (HunterLab, Reston, VA). For each pork roast, a 1.27-cm thick slice from the cross-sectional center of the longissimus dorsi muscle was covered with plastic wrap, placed over the sensing lamp, and covered with a black, nonreflective cloth. Duplicate readings of L (luminescence), a (redness), and b (yellowness) values were taken on each sample.

Sensory Evaluation
Sensory evaluation of pork samples was conducted by a panel of nine volunteer faculty, staff, and students from the College of Home Economics, Oregon State University. Experienced, trained panelists were selected on the basis of participation in previous meat evaluation studies, availability, and demonstrated ability to perceive differences in color, texture, flavor, and off-flavor of meats. All sensory evaluations were accomplished in an open fluorescent lit laboratory area at room temperature (27°C). Panelists evaluated each treatment for typical pork flavor, off-flavor, juiciness, and tenderness using a nine-point intensity scale (1, none; 9, extreme). Randomly coded samples presented for sensory evaluation were 1.27-cm cubes, taken from the longissimus dorsi muscle and served in a pyrex covered container. Panelists received two coded cubes for each of the two treatments, with two sessions at each sampling period. Panelists viewed, scored, and covered one treatment before viewing the next. Water was available to the panelists for rinsing their mouths after sampling. After sensory evaluation was completed, color
was evaluated using samples covered with clear plastic wrap and viewed under a MacBeth light simulating a sunny day at high noon.

**Statistical Analysis**

Summary statistics (mean and standard error of the mean) were calculated for each dependent variable. A 2 X 2 factorial design with repeated measures (storage time) on each pig was used to analyze all dependent variables except the sensory data (Milliken and Johnson, 1984). There were two levels for each factor: placebo or pST treatment; and fresh or stored samples. The sources of variation were treatment, pig within treatment, storage time, and the interaction of treatment and storage time. The sensory data also were analyzed with a two-way design with repeated measures on two factors: panelist and storage time (Milliken and Johnson, 1984). With this design each panelist received samples from each pig at each time. This design had the following sources of variation: panelist, treatment, pig within treatments, storage time, and interaction of treatment and storage time. Data were analyzed for statistical significance using SAS (SAS Institute Inc., 1985). Statistical significance was defined as $P < 0.05$.

**RESULTS AND DISCUSSION**

Growth performance and carcass parameters are presented in a subsequent publication in detail (Clark et al., 1992). Briefly, the data suggested that the pST-treated pigs had a greater average-daily gain than the control pigs but that feed efficiency was not affected. There was also a small decrease in backfat thickness and an increase in the calculated percent of lean tissue.

Moisture in the cooked loin roast was not changed by pST treatment but was increased slightly (3.5%, $P < 0.05$) after storage (Table 1). Prusa et al. (1989b) also saw no change in the moisture content of raw and broiled rib chops from pigs treated with pST. However, other investigators using similar pST treatments (Halloran et al., 1991; Boles et al., 1991) have shown that moisture increased slightly in pST-treated pigs. There was a small decrease (5.6%, $P < 0.05$) in the EMI of the pST-treated animals (Table 1) and a decrease (18.4%, $P < 0.01$) in the stored samples. These data are in contrast to those observed by Prusa et al. (1989a) and Fabry et al. (1991), both of whom noted that pST treatment did not affect EMI. They are in keeping with the effect of frozen storage on water-holding capacity (Fennema, 1985).

The total lipid content of the loin roasts was altered by pST treatment. When expressed on a wet weight basis, the total lipid content from treated animals decreased 33% ($5.5 \pm 0.65$ vs $3.4 \pm 0.30$ g/100 g wet wt, $P < 0.01$; Table 1), a loss of 2.1 g/100 g. The impact of the pST treatment on the total lipid content remained when the data were expressed on a dry weight basis (Table 1). On a dry weight basis, pST treatment caused a 32% decrease in total lipid content ($15.4 \pm 1.51$ vs $10.4 \pm 0.79$ g/100 g dry wt, $P < 0.01$). A decrease in the amount of fat in tissues of pigs treated with pST has been observed often (Etherton et al., 1987; Evock et al., 1988; Campbell et al., 1989). Prusa et al. (1989a) observed a decrease from 4.3 to 2.4% fat in broiled rib chops. However, it should be noted that the fat content measured in our study in both control and treated pigs as well as by Prusa et al. (1989a) was low in comparison to that given in the USDA (1983) Handbook 8-10 where total lipids of roasted pork loin, center rib, separable lean are listed at 13.80 g/100 g wet wt but in keeping with the values
found by Buege et al. (1991). In the current study, there was no effect of storage time on the total lipid content of loin roasts.

The cholesterol content of the cooked loin roasts was not affected by pST treatment but was affected by storage (P < 0.01; Table 1). Before storage, cholesterol content was 78.2 mg per 100 g wet wt. After storage, it had decreased 19%. In 100 g of tissue this represented a loss of 14.9 mg of cholesterol. When expressed on a dry weight basis, the decrease in cholesterol remained significant (14%, P < 0.01), indicating that the loss of cholesterol with freezer storage was not due to a change in moisture. The complication of significant differences in cooking loss in stored samples could be a partial explanation. Additionally, a loss of cholesterol with freezer storage has been reported previously (Kregel et al., 1986). Little is known about the breakdown of cholesterol in muscle foods, although it has been suggested that conditions present during cooking and storage may be conducive to cholesterol oxidation in muscle foods (Kregel et al., 1986). In view of the fact that cholesterol oxidation products have been implicated in the process of atherosclerosis (Addis and Park, 1989), such changes need to be investigated further.

Our findings that pST treatment (3 mg per day) had no influence on the cholesterol content of cooked loin roasts have also been observed by Prusa et al. (1989a) with doses of 4 mg pST per day. However, they found that 8 mg pST per day increased cholesterol content slightly. Collectively these two studies suggest that tissue cholesterol levels are dependent on the dose of pST and that a dose of 3 mg/day (as was used in the current study) is too low to cause changes. The value of 78.2 mg cholesterol per 100 g cooked loin roast is comparable to 79 mg/100 g given in USDA Handbook 810 values (USDA, 1983).
The fatty acid profile of the loin roast changed with pST treatment (Table 2). On a wet weight basis, 18:1(n — 7) decreased 33% (P < 0.05), 14:0, 16:0, 16:1(n — 7), 18:0, 18:1(n — 9)c, 20:0, and 20:1(n — 9) all decreased nearly 41% (P < 0.05), and 18:3(n — 3) decreased 50% (P < 0.05) with pST treatment. For many of these fatty acids, the decrease represented only a small change to the lipid content of the tissue. For instance, changes of less than 0.05 g per 100 g of tissue were observed for 14:0, 18:3(n — 3), 20:0, and 20:1(n — 9). Total saturated fatty acids (SFA) and monounsaturated fatty acids (MUFA) both decreased 39% (P < 0.05) with treatment losses of 0.84 g and 1.06 g, respectively. Total polyunsaturated fatty acids (PUFA) were unchanged with treatment. Expressing fatty acids on a dry weight basis did not alter these effects (data not shown). The fatty acid composition of cooked loin roasts as weight percentages is given in Table 3. Reported in this manner, there was a decrease in 18:1(n — 9)c (6.2%, P < 0.01) and increases in 18:2(n — 6) (34.4%, P < 0.05), 20:3(n — 6) (57.1%, P < 0.01), and 20:4(n — 6) (58.3%, P < 0.05) with pST treatment. Overall, there was a 4.5% decrease in the percentage of MUFA (P < 0.05) and a 35.4% increase in the percentage of PUFA (P < 0.05). Data expressed in this manner are less useful to the consumer (Kinsella et al., 1975) who is ultimately interested in intake of each of the fatty acids, not the intake relative to the other fatty acids.

The effect of storage on the fatty acid composition of cooked loin roast on a wet weight basis is given in Table 2. After storage, 18:1(n — 7) (P < 0.01), 18:2(n — 6) (P < 0.05), 20:3(n — 6) (P < 0.01), 20:4(n — 6) (P < 0.01), and ΣPUFA 0.01), and ΣPUFA (P < 0.05)

**TABLE 2**

**EFFECT OF pST AND STORAGE ON THE FATTY ACID COMPOSITION OF COOKED PORK LOIN ROAST (mg/100 g WET WT)**

<table>
<thead>
<tr>
<th>Fatty Acid⁶</th>
<th>Control³</th>
<th>pST³</th>
<th>Control⁷</th>
<th>pST⁷</th>
<th>Trt</th>
<th>Stor</th>
<th>Trt x Stor</th>
</tr>
</thead>
<tbody>
<tr>
<td>14:0</td>
<td>72±8.3</td>
<td>42±4.8</td>
<td>65±9.9</td>
<td>42±6.6</td>
<td>&lt;0.05 NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>16:0</td>
<td>1500±150</td>
<td>900±70</td>
<td>1300±190</td>
<td>800±110</td>
<td>&lt;0.05 NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>16:1(n — 7)</td>
<td>160±17</td>
<td>100±10</td>
<td>150±18</td>
<td>100±16</td>
<td>&lt;0.05 NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>18:0</td>
<td>70±8.2</td>
<td>400±83</td>
<td>660±105</td>
<td>400±57</td>
<td>&lt;0.05 NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>18:1(n — 9)c</td>
<td>13±1.4</td>
<td>7±0.8</td>
<td>15±2.7</td>
<td>12±2.0</td>
<td>NS NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>18:1(9c)</td>
<td>2300±240</td>
<td>1300±100</td>
<td>2200±120</td>
<td>1400±200</td>
<td>&lt;0.05 NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>18:2(n — 6)</td>
<td>300±31</td>
<td>190±12</td>
<td>240±33</td>
<td>170±22</td>
<td>&lt;0.05 &lt;0.01</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>18:2(6)</td>
<td>460±32</td>
<td>380±18</td>
<td>390±40</td>
<td>350±33</td>
<td>&lt;0.05 &lt;0.05</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>18:3(n — 3)</td>
<td>16±1.7</td>
<td>11±1.2</td>
<td>15±1.9</td>
<td>11±1.8</td>
<td>&lt;0.05 NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>20:0</td>
<td>10±1.3</td>
<td>6±1.2</td>
<td>12±1.1</td>
<td>8±1.2</td>
<td>&lt;0.05 &lt;0.01</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>20:1(n — 9)</td>
<td>43±5.4</td>
<td>25±3.0</td>
<td>45±7.8</td>
<td>30±4.0</td>
<td>&lt;0.05 NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>20:2(n — 6)</td>
<td>17±2.0</td>
<td>13±1.3</td>
<td>18±2.3</td>
<td>13±1.5</td>
<td>NS NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>20:3(n — 6)</td>
<td>6±0.4</td>
<td>7±0.7</td>
<td>5±0.4</td>
<td>5±0.4</td>
<td>NS &lt;0.01</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>20:4(n — 6)</td>
<td>51±4.0</td>
<td>51±2.1</td>
<td>42±4.1</td>
<td>43±2.6</td>
<td>NS &lt;0.01</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>ΣSFA⁸</td>
<td>2300±240</td>
<td>1300±120</td>
<td>2000±100</td>
<td>1300±180</td>
<td>&lt;0.05 NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>ΣMUFA⁹</td>
<td>2800±290</td>
<td>1700±120</td>
<td>2600±180</td>
<td>1700±240</td>
<td>&lt;0.05 NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>ΣPUFA⁹</td>
<td>550±39</td>
<td>460±22</td>
<td>460±48</td>
<td>420±38</td>
<td>NS &lt;0.05</td>
<td>NS</td>
<td></td>
</tr>
</tbody>
</table>

⁶Expressed as the weight of the fatty acid methyl esters per 100 g wet weight of tissue; values are means ± SEM
⁷Fatty acids are represented in the following manner: the number before the colon is the number of carbon atoms; the number after the colon is the number of double bonds; n is the number of carbon atoms from the methyl end of the molecule to the first double bond; r is a trans-isomer and c is a cis-isomer.

ⁿ=6

¹ⁿ = 5

³NS = not significant at p < 0.05; Trt = pST treatment; Stor = frozen storage
⁴ΣSFA = 14:0 + 16:0 + 18:0 + 20:0
⁵ΣMUFA = 16:1(n — 7) + 18:1(n — 9)c + 18:1(n — 9c) + 18:1(n — 7) + 20:1(n — 9)
⁶ΣPUFA = 18:2(n — 6) + 18:3(n — 3) + 20:2(n — 6) + 20:3(n — 6) + 20:4(n — 6)
decreased. On a dry weight basis the decrease in 18:1(n — 7), 20:3(n — 6), and 20:4(n — 6) remained significant whereas the decrease in 18:2(n — 6) and PUFA did not (data not shown). In addition, there was an increase in 20:0 which was not seen when fatty acids were expressed by wet weight. However, this change was less than 0.01 g per 100 g of tissue. Overall, these effects are small.

The effect of storage on the weight percentage of the fatty acids in cooked loin roast is given in Table 3. An interaction between storage and treatment was seen for one fatty acid, 18:1(n — 9)t. With storage, the percentage of this fatty acid increased 27% in the control samples but the increase was greater for pST-treated roasts (65%). Increases in 18:0 (1.5%, P < 0.05), 18:1(n — 9)c (3.9%, P < 0.01), 20:0 (39.4%, P < 0.01), 20:1(n — 9) (16.6%, P < 0.01), 20:2(n — 6) (4.0%, P < 0.01), and ΣMUFA (2.4%, P < 0.01) and decreases in 16:0 (2.7%, P < 0.01), 18:1(n — 7) (11.1%, P < 0.01), 20:3(n — 6) (25.8%, P < 0.01), and ΣSFA (0.9%, P < 0.05) were also seen with storage.

Limited information is available in the literature with which to compare the changes produced by pST and storage on fatty acid profile of cooked loin roasts. Prusa et al. (1989a), expressing data as relative weight percentages, saw no change in the content of SFA, MUFA, or PUFA of intramuscular fat from broiled, boneless rib chops from pigs treated with 4 mg/day of pST. Lonergan et al. (1992) found no differences in the fatty acid profile of adipose tissue at seven carcass locations when data were expressed as weight percentages. In this study, the most important changes seen were the decreases in the amounts (milligrams per 100 g wet tissue) of 16:0, 18:0, and 18:1(n — 9)c, since these three fatty acids account for nearly 75% of the total

<table>
<thead>
<tr>
<th>Fatty Acid</th>
<th>Control</th>
<th>pST*</th>
<th>Control</th>
<th>pST*</th>
<th>Trt</th>
<th>Stor</th>
<th>Trt×Stor</th>
</tr>
</thead>
<tbody>
<tr>
<td>14:0</td>
<td>1.2±0.04</td>
<td>1.2±0.00</td>
<td>1.2±0.06</td>
<td>1.2±0.06</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>16:0</td>
<td>25.5±0.37</td>
<td>24.5±0.33</td>
<td>24.8±0.33</td>
<td>24.1±0.29</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>NS</td>
</tr>
<tr>
<td>16:1(n—7)</td>
<td>2.9±0.14</td>
<td>2.9±0.24</td>
<td>2.9±0.14</td>
<td>2.9±0.21</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>18:0</td>
<td>12.1±0.51</td>
<td>11.3±0.48</td>
<td>12.2±0.53</td>
<td>11.5±0.33</td>
<td>NS</td>
<td>&lt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>18:1(n—9r)</td>
<td>0.2±0.01</td>
<td>0.2±0.01</td>
<td>0.3±0.01</td>
<td>0.3±0.02</td>
<td>NS</td>
<td>&lt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>18:1(n—9c)</td>
<td>39.8±0.32</td>
<td>37.3±0.51</td>
<td>41.3±0.44</td>
<td>38.7±0.95</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>NS</td>
</tr>
<tr>
<td>18:1(n—7)</td>
<td>5.2±0.19</td>
<td>5.4±0.32</td>
<td>4.7±0.14</td>
<td>4.7±0.13</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>NS</td>
</tr>
<tr>
<td>18:2(n—6)</td>
<td>8.2±0.67</td>
<td>10.8±0.56</td>
<td>7.7±0.69</td>
<td>10.5±0.85</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>18:3(n—3)</td>
<td>0.3±0.02</td>
<td>0.3±0.03</td>
<td>0.3±0.02</td>
<td>0.3±0.01</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>20:0</td>
<td>0.2±0.01</td>
<td>0.2±0.02</td>
<td>0.2±0.02</td>
<td>0.2±0.01</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>20:1(n—9)</td>
<td>0.7±0.05</td>
<td>0.7±0.05</td>
<td>0.8±0.05</td>
<td>0.9±0.04</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>NS</td>
</tr>
<tr>
<td>20:2(n—6)</td>
<td>0.3±0.02</td>
<td>0.3±0.02</td>
<td>0.3±0.02</td>
<td>0.4±0.02</td>
<td>NS</td>
<td>&lt;0.01</td>
<td>NS</td>
</tr>
<tr>
<td>20:3(n—6)</td>
<td>0.1±0.01</td>
<td>0.2±0.01</td>
<td>0.1±0.01</td>
<td>0.1±0.01</td>
<td>&lt;0.01</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>20:4(n—6)</td>
<td>0.9±0.13</td>
<td>1.5±0.10</td>
<td>0.9±0.14</td>
<td>1.4±0.27</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>ΣSFA</td>
<td>39.0±0.89</td>
<td>37.1±0.68</td>
<td>38.5±0.84</td>
<td>36.9±0.56</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>ΣMUFA</td>
<td>48.8±0.45</td>
<td>46.4±0.75</td>
<td>49.9±0.50</td>
<td>47.5±1.22</td>
<td>&lt;0.05</td>
<td>&lt;0.05</td>
<td>NS</td>
</tr>
<tr>
<td>ΣPUFA</td>
<td>9.8±0.83</td>
<td>13.1±0.67</td>
<td>9.3±0.87</td>
<td>12.7±1.14</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Expressed as the weight percent of the fatty acid methyl esters; values are mean ± SEM
Fatty acids are represented in the following manner: the number before the colon is the number of carbon atoms; the number after the colon is the number of double bonds; n—r is the number of carbon atoms for the methyl end of the molecule to the first double bond; r is a cis-isomer and c is a cis-isomer.

n=5

*NS = not significant at p<0.05; Trt = pST treatment; Stor = frozen storage

ΣSFA = 14:0 + 16:0 + 18:0 + 20:0
ΣMUFA = 18:1(n—7) + 18:1(n—9r) + 18:1(n—9c) + 18:1(n—7) + 20:1(n—9)
ΣPUFA = 18:2(n—6) + 18:3(n—3) + 20:2(n—6) + 20:3(n—6) + 20:4(n—6)
fatty acids. Also of interest is the fact that 18:2(n — 6), a fatty acid that accounts for about 10% of the total fatty acid, was unchanged with pST treatment.

The changes in the fatty acid content in the treated animals are consistent with reports in the literature of pST treatment decreasing lipogenesis. Treating pigs with pST has been reported to decrease fatty acid synthesis 50 to 70%. In addition, the activity of several lipogenic enzymes, including fatty acid synthetase, has been shown to decrease (Boyd and Bauman, 1989). Our findings of decreases in total SFA (including 16:0 and 18:0), decreases in MUFA (including 18:1(n — 9)c), and no change in PUFA (including 18:2(n — 6)), support this mechanism. A decrease in fat synthesis would lead to a decrease in the production of the SFA and MUFA with little change in the amount of PUFA since the majority of the PUFA in pig tissues, 18:2(n — 6) and 18:3(n — 3), come from the diet. However, these data also could be explained by an increased turnover of storage lipids. The changes in the fatty acid content of the stored sample are consistent with suggestions that meat undergo some oxidation during storage at low temperature which often results in the loss of some of the PUFA (Kinsella et al., 1975).

The effects of pST and storage on the tests of quality characteristics of the cooked loin roasts are given in Table 4. There was no difference in the initial weights of the roast in each group (data not shown); pST treatment did not affect cooking time. Frozen storage, however, shortened cooking time when data were expressed as min/ g (P < 0.05). It has long been known that such factors as freezing prior to roasting of poultry can affect cooking time, although the results have not been consistent (Goertz et al., 1960; Alexander et al., 1948). A similar phenomenon may have occurred in the loin roasts. There was an interaction (P < 0.05) of pST treatment and storage on the percentage of total cooking loss. The percentage of cooking loss decreased with storage for roasts from both treated and control animals but the loss was greater for the control roasts. Other storage effects included a decrease in drip loss (P < 0.05) and an increase (P < 0.01) in the Hunter color a value (red—green hues). pST-treated pigs showed a 29% decrease in drip loss (P < 0.01) and a 30% increase in the WarnerBratzler shear value (P < 0.01). There was no change due to pST treatment in cooking time nor in Hunter color values.

The effects of pST and storage on the sensory traits of cooked loin roasts evaluated by a trained panel are given in Table 4. There was an interaction of treatment and storage on off-flavor. The off-flavor of control roasts increased with storage but pST treated roasts were unchanged. There was an increase in typical flavor and juiciness from the samples taken from the stored roasts. Perhaps the shorter cooking time of the stored roasts was responsible for the increased moisture, lower cooking losses (both as total and drip), lower EMI value, and higher typical flavor and juiciness in the stored samples. Sensory traits were not influenced by pST treatment.

Several studies have evaluated the effect on texture of various tissues from swine treated with pST using either the Warner—Bratzler measurement of shear strength (Halloran et al., 1991; Gardner et al., 1989; Evock et al., 1988; Solomon et al., 1991; Miller et al., 1991; Prusa et al., 1989a, 1990) or trained panels (Halloran et al., 1991; Hagen et al., 1991; Evock et al., 1988; Boles et al., 1991; Prusa et al., 1989a, 1990; Beerman et al., 1990). The results, however, have not been consistent. About one- half of the studies have reported that shear values and tenderness did not change; the
remainder showed an increase in shear values and decrease in tenderness. The amount of pST administered, the weight of the pig at the time of the first injection, and the length of time pST was given may explain these differences. However, as pointed out by Solomon et al. (1991), seasonal effects and genetic differences between the pigs may also play a role.

This study indicated that the daily administration of pST significantly reduced the fat content in pork. When consuming a 100 g serving of loin roast, the fat intake would be lowered by about 2 g. Effectively all of this loss in fat can be attributed to a loss in saturated and monounsaturated fatty acids for the polyunsaturated fatty acid content was virtually unchanged. These changes made little difference to the overall acceptability of the roasts. Consequently, the use of pST treatment in pork may provide a product that is more in keeping with current dietary guidelines but that does not differ extensively in quality characteristics either when consumed after short-time or after lengthy frozen storage.

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REFERENCES


