

Effect of Dietary Sodium Oxalate on Pork Quality

Brian T. Kremer, Ph.D. candidate;
Tim S. Stahly, professor; and
Joseph G. Sebranek, professor
Department of Animal Science

ASL-1620

Summary and Implications

The effects of feeding sodium oxalate, a glycolytic inhibitor, on pork quality were investigated. Four hours pretransport, market-weight pigs (111 ± 7 kg) were allowed access to 557 grams of feed containing 0, 9, or 44 grams of sodium oxalate. Dietary sodium oxalate addition slowed the rate of pH decline in muscle postmortem and minimized percentage of water loss from pork during retail storage. Muscle pH from 22 to 180 minutes postmortem was 0.09 to 0.10 units greater in pigs fed sodium oxalate, but the ultimate pH measured at 24 hours postmortem was not affected by diet. Percentage of water loss from muscle samples stored under retail conditions for 3, 6, 9, or 12 days was 1.2 to 1.8% less in pigs fed sodium oxalate. Dietary sodium oxalate did not affect color (Hunter L* and a* scores) of the pork during retail storage. Based on these data, the dietary addition of sodium oxalate shortly before slaughter is a biologically feasible technology for improving water holding capacity of pork products.

Introduction

The quality of pork products is a pressing concern in the pork industry. Loss of water from a meat product results in lower weights of saleable product. It also results in a product that has impaired processing functionality, as well as lower consumer appeal. Pale color of pork products also lowers consumer demand, particularly for exports. These problems must be addressed in an innovative manner if the pork industry is to continue to grow.

The biological basis for these limitations in pork quality is due in part to the rate of glycolysis that takes place in muscle of animals postmortem as well as to the temperature of the muscle. Initially postmortem, the muscle continues to catabolize glycogen and glucose for energy. This catabolic process is termed glycolysis. In the absence of oxygen, glycolysis results in the accumulation of lactic acid. As lactic acid accumulates, the pH of the muscle declines. The muscle temperature rises initially postmortem due to the heat generated by anaerobic glycolysis and then begins to decline slowly. The muscle temperature declines sharply when the carcass is placed in the chiller 30 to 45 minutes postmortem.

When muscle pH declines rapidly but the muscle temperature is elevated, the rate of muscle protein denaturation increases (5). If the myofibrillar protein myosin is denatured, the muscle sarcomere loses volume,

and water is evacuated to the extracellular space from which it can be more readily lost (6). Denaturation of the sarcoplasmic protein myoglobin, which gives the meat its red color, also contributes to a change in the color of the pork. Based on the quantitative relationship of pH decline and muscle protein denaturation, factors that reduce the glycolytic rate and, thus, the pH decline by 25 and 50% during the first 60 minutes postmortem have been estimated to result in a 66 and 85% reduction in rate of protein denaturation, respectively (6). A reduced rate of protein denaturation is associated with a slower pH decline and a greater water holding capacity in muscle postmortem (7).

Oxalate, a compound present in several human foodstuffs, can effectively inhibit muscle glycolysis by inhibiting a key glycolytic enzyme, pyruvate kinase (8). An oxalate concentration of $6 \mu\text{M}$ in rabbit muscle cell culture systems inhibits pyruvate kinase (8). Buc et al. (2) have further determined that the oxalate concentration needed to inhibit pyruvate kinase in muscle is less than that which inhibits liver (rabbit) and erythrocyte (human) pyruvate kinase. The objective of this study was to determine the effect of feeding the glycolytic inhibitor sodium oxalate on the water holding capacity of pork.

Materials and Methods

Nine sets of three littermate pigs from a high lean, halothane-negative genetic strain were penned individually in a thermal neutral environment and self-fed a basal corn-soy diet. At a body weight of 111 ± 7 kg, three pigs within each litter were randomly assigned to one of three dietary regimens. The regimens consisted of the basal diet supplemented with 0, 9, and 44 grams of oxalate per 454 grams of feed. These regimens were estimated to provide 0, 2, and 10 times K_i of muscle pyruvate kinase. The K_i for an enzyme is the amount of an inhibitor required to reduce the activity of that enzyme by half. The amount of sodium oxalate required to provide $2\times$ and $10\times$ K_i was based on the following assumptions: oxalate digestibility of 1%, equal distribution of absorbed sodium oxalate in the body water pool, and body water equal to 52% of the body weight. Each pig was allowed access to 557 grams of feed for a 4-hour period prior to loading and transport to the ISU Meat Laboratory.

The pigs were transported (2.5 miles) and killed within littermate groups by the method of Bertram et al. (1). The pigs were killed by exsanguination within 7 seconds of stunning (280 V for 7 seconds), bled for 5 minutes, and placed in a scalding tank (64°C) at 7 minutes postmortem. The pigs were removed from the scalding tank, the carcass was eviscerated, and the body was split at 15, 20, and 25 minutes postmortem, respectively.

At 45 minutes postmortem, the right carcass half was chilled at 0°C . The left side was maintained at 18.3°C until 180 minutes postmortem at which time it was chilled at 0°C . These conditions simulated a rapid and slow chilling

environment, respectively (4, 3). Two muscles in each carcass side were monitored (longissimus and semimembranosus). These two muscles have a predominance of glycolytic (white) fibers and are of economic importance to the packer. At 24 hours postmortem, the longissimus and semimembranosus muscles were removed from the carcass and cut into 2.5-cm thick chops and trimmed of subcutaneous fat and bone and stored.

Muscle pH and temperature were measured at 0, 22, 45, 90, and 180 minutes postmortem in both muscles and both chilling environments by using an ISFET pH probe (accurate to 0.02 units) and thermister (accurate to 0.1°C). Muscle ultimate pH also was determined at 24 hours postmortem. Muscle drip loss and color were measured on day 0, 3, 6, 9, and 12 of retail storage, according to the methods of Bertram et al. (1) and Stahly et al. (9); however, the storage temperature was 5.6°C in muscles from both chilling regimens. Muscle drip loss was determined as the weight loss of the muscle expressed as a percentage of the initial weight. Color scores were assessed by Hunter L*a*b* analysis. Pork cooking loss and tenderness were estimated on the samples on day 12 of retail storage. Cooking loss was determined by heating a chop to an internal temperature of 68°C, then reweighing the sample. Pork tenderness was estimated via the Star Probe technique by using an Instron Universal Testing Machine.

The data were analyzed using the GLM and Mixed procedures of SAS. Initial chop weight was used as a covariate in the analysis of data for the drip loss. Responses over time were analyzed as repeated measures.

Results

During the 4-hour pretransport feeding period, pigs voluntarily consumed 340, 358, and 100 grams of feed in the 0, 2×, and 10× sodium oxalate treatment groups, respectively, resulting in sodium oxalate intakes of 0, 70, and 97 mg, respectively (Table 1). The main effects of diet pooled across the two muscles (longissimus and semimembranosus), chilling temperatures (slow and quick), and monitoring times on pork quality are reported in Table 1 and are discussed below. Dietary sodium oxalate increased muscle pH (5.88, 5.97, and 5.98) but did not alter temperature or ultimate pH at 24 hours postmortem (5.54, 5.55, and 5.63). This observation supported our hypothesis that a glycolytic inhibitor would reduce the rate of pH decline in muscle postmortem but would not change the ultimate pH. Dietary sodium oxalate addition also lowered the percentage of water loss during the retail storage (11.3, 10.1, and 9.5%). These observations lead us to conclude that slowing the rate of pH decline but not altering ultimate pH can improve the water holding capacity of fresh pork muscle. Dietary sodium oxalate addition did not alter Hunter L* (paleness) or Hunter a* (redness) scores of the muscles.

These effects of dietary sodium oxalate on muscle pH and percentage of water loss were observed in both the semimembranosus (SM) and the longissimus muscles (LD) and in both chilling temperature environments (rapid vs. slow). Thus, it is hypothesized that dietary sodium oxalate

addition would help minimize the water loss in pork muscles processed under good as well as suboptimum chilling conditions.

Dietary sodium oxalate did not alter percentage of cooking loss of the pork muscles, even though the chops from pigs fed oxalate retained more water during retail storage. Dietary sodium oxalate also did not alter the penetration resistance of the muscle, indicating that the dietary treatments did not alter the ultimate structure of the pork product. Based on these data, the short-term feeding of sodium oxalate results in improved pork quality as measured by the amount of water retained by the meat.

References

1. Bertram, M.J., T.S. Stahly, and R.C. Ewan. 1995. ISU Swine Research Report, ASLR1262.
2. Buc, H., F. Demaugre, and J.P. Leroux. 1978. *Biochem. Biophys. Res. Comm.* 85:744–779.
3. Kauffman, R.G., W. Sybesma, F.J.M. Smulders, G. Eikelenboom, B. Engel, R.L.M.J. van Laack, A.H. Hoving-Bolink, P. Sterrenburg, E.V. Nordheim, P. Walstra, and P.G. van der Wal. 1993. *Meat Sci.* 34:283–300.
4. Landes, D.R., L.E. Daeson, and J.F. Price. 1971. *J. Food Sci.* 36:122–124.
5. Lawrie, R.A. 1985. *Meat Science* 4th ed. Pergamon Press, Inc., Maxwell House, Fairview Park, Elmsford, NY.
6. Offer, G., and P. Knight. 1988. Structural basis of water-holding in meat. In: *Developments in Meat Science*, pp. 63–173.
7. Penny, I.F. 1969. *J. Food Technol.* 4:269–273.
8. Reed, G.H., and S.D. Morgan. 1974. *Biochem.* 13:3537–3541.
9. Stahly, T.S., N.H. Williams, M.J. Bertram, D.R. Cook, and D. Gerrard. 1997. *Am. Soc. Anim. Sci. Midwest Mtg. Abstr.*, p. 64.

Table 1. Main effect of dietary sodium oxalate on biological and pork quality criteria. (Data pooled across chill temperature [rapid vs. slow] and muscle type [LD vs. SM] and time postmortem.)

Criteria	Dietary sodium oxalate, K_i		
	0	2X	10X
Feed consumption (4 hours prior to transport)			
Feed allocation (g/pig)	545	568	557
Feed intake (g/pig)	342	360	100
Oxalate intake (g/pig)	0	7.0	9.7
Muscle traits postmortem ^b			
pH ^a	5.88	5.97	5.98
Temperature (°C)	31.4	31.2	31.0
Pork retail traits during storage ^c			
Water loss (%) ^d	11.3	10.1	9.5
Color L*	52.9	53.0	52.7
Color a*	5.6	5.5	5.3
Pork traits after storage ^e			
Cooking loss (%)	29.5	29.6	28.4
Penetration resistance (kg)	3.00	2.97	3.00

^aEffect of dietary sodium oxalate 0 vs. 2 & 10x, $P < .06$.

^bData pooled across 22, 45, 90, and 180 minutes postmortem.

^cData pooled across 0, 3, 6, 9, and 12 days of retail storage.

^dEffect of dietary sodium oxalate 0 vs. 2 & 10x, $P < .05$.

^eData from day 12 of retail storage.

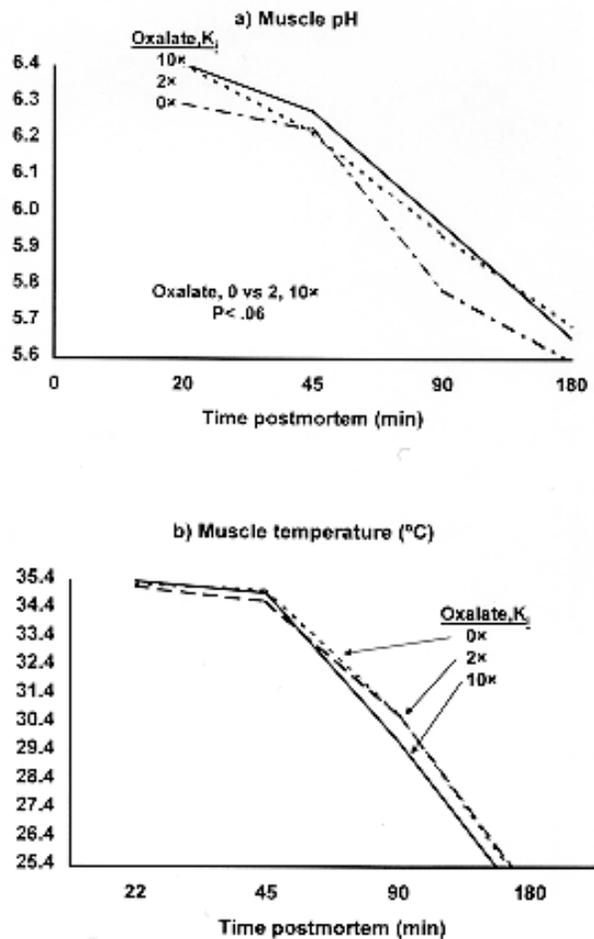


Figure 1. Effect of dietary sodium oxalate on (a) muscle pH and (b) muscle temperature postmortem. (Data pooled across chill temperature and muscle type.)

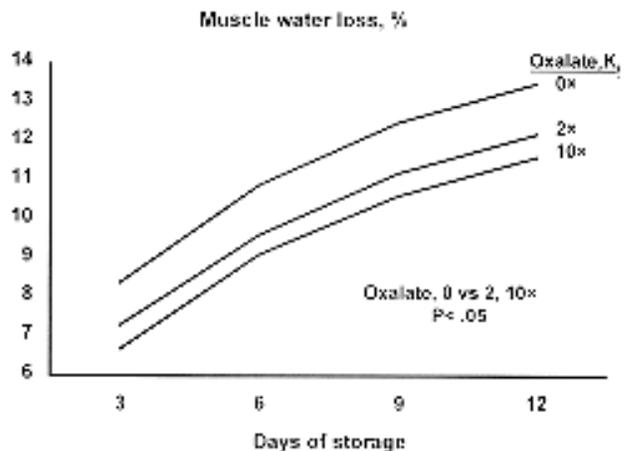


Figure 2. Effect of dietary sodium oxalate on pork water loss during retail storage. (Data pooled across chill temperature and muscle type.)

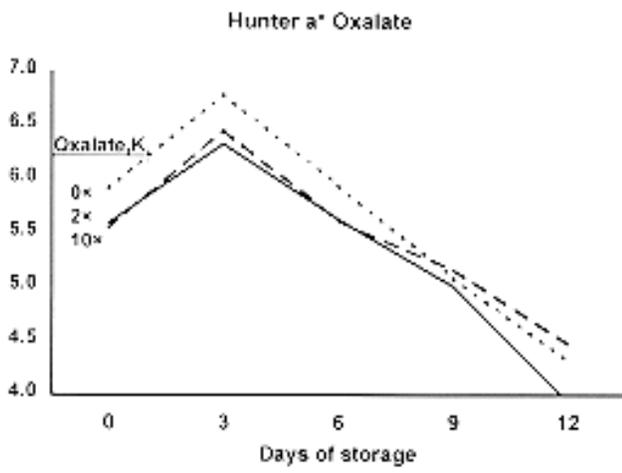


Figure 3. Effect of dietary sodium oxalate on pork color (a) Hunter L* scores and (b) Hunter a* scores. (Data pooled across chill temperature and muscle type.)