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Quantification of Pyruvate Dehydrogenase in Normal and PSE Turkey Breast Muscles

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Objectives

A major challenge facing the turkey industry continues to be the Pale Soft Exudative (PSE) syndrome. The PSE turkey meat problem is most evident as poor protein extractability and gelation in processed meat products. PSE development is generally thought to result from an unusually high rate of postmortem glycolysis causing a rapid drop of pH while the temperature is still warm, resulting in denaturation of meat proteins. However, the specific mechanism underlying the accelerated postmortem metabolism is still poorly understood. Recent studies from our laboratory have shown that expression of the pyruvate dehydrogenase kinase isozyme 4 (*PDK4*) gene and the PDK4 protein are dramatically downregulated in PSE turkey. PDK4 serves as a modulator of glycolytic metabolism by regulating pyruvate dehydrogenase (PDH) activity. Phosphorylation of PDH by PDK4 results in inactivation of PDH with a shift to anaerobic metabolism and lactate production. A crucial first step in defining the specific mechanism by which PDK4 expression could affect development of PSE muscle is the quantification of PDH levels in normal and PSE samples. In this study, we test the hypothesis that PDH levels are not different between meat samples characterized as normal or PSE.

Materials and Methods

Randombred Control Line 2 turkeys (n = 20), representing the turkey of the 1960s maintained without selection pressure were raised to 22 wk of age, and slaughtered and processed according to industry standards. Muscle samples from pectoralis major were collected at 5 min postmortem, cut into small pieces, snap frozen in liquid nitrogen and stored at –80°C until further use. Breast muscle samples were classified as normal or PSE based on marinade uptake at 24h postmortem, with high uptake for normal and low uptakes for PSE. To quantify PDH levels, frozen muscle samples (6 normal and 6 PSE) were pulverized and extracted with cell lysis buffer supplemented with protease and phosphatase inhibitors. Following centrifugation to remove insoluble material, proteins of the supernatants were separated by SDS-PAGE and analyzed by western blotting using a polyclonal antibody for human E1 component subunit α-PDH, and a monoclonal antibody to chicken β-actin was used as a loading control. Following immunoblotting, the membrane was analyzed using the Odyssey Imaging System (Licor) using fluorescent secondary antibodies.

Results

Imaging of the membrane revealed that there was no significant difference (P = 0.24) in PDH abundance between normal and PSE meat samples.

Conclusion

These results suggest that variation in PDH abundance does not contribute to the development of PSE meat. Taken together with the previous observation that PDK4 levels are decreased in PSE muscle, the results suggest that the phosphorylation state of PDH may be a determinant of whether a muscle is likely to become PSE.