Objectives

AMPK-activated protein kinase (AMPK) is a heterotrimeric enzyme that participates in the regulation of cellular energy homeostasis and is comprised of α, β, and γ subunit. In pigs, 2 known genetic mutations in the skeletal muscle specific γ3 regulatory subunit alter fresh pork quality. The first mutation, AMPKγ3 R200Q (Rendement Napole mutation), arises from an arginine to glutamine substitution and makes AMPK constitutively active. Animals with this mutation produce meat with an abnormally low ultimate pH known as acid meat. The second mutation, AMPKγ3 V199I, arises from a valine to isoleucine substitution making AMPK unresponsive to AMP. While the AMPKγ3 V199I mutation has been associated with improved pork quality, little information is available elucidating the biochemical mechanism responsible. Therefore, our objective was to investigate the role of the AMPKγ3 V199I mutation in determining fresh meat quality during the conversion of muscle to meat.

Materials and Methods

We investigated this mutation using Berkshire pigs, a breed known to carry the AMPKγ3 V199I mutation with a high frequency (80 to 90% homozygous mutants). Homozygous mutant (n = 12) and heterozygous Berkshire pigs (n = 9) were harvested at The Ohio State University’s Meat Laboratory. Muscle samples were collected from the longissimus lumborum et thoracis at 0, 30, 60, 120, 240, and 1440 min postmortem and frozen in liquid nitrogen to measure pH decline. To determine the rate of pH decline, a single exponential decay curve was fit to the data to derive a rate constant for each pig. After 1440 min, carcass data were measured for dressing percentage, backfat thickness, loin eye area, and Minolta color (L*, a*, and b*). Data were analyzed with a mixed model in JMP. The LSMeans were compared using a Student’s t test and considered significant at P ≤ 0.05.

Results

No differences were detected in any carcass measurements. The homozygous mutant pigs exhibited a decreased rate of postmortem pH decline (P = 0.0325). This difference primarily arose from the elevated muscle pH of homozygous mutant muscle at 30 min (P = 0.0036) and 60 min (P = 0.0391) postmortem compared to the heterozygous pigs. The slower postmortem pH decline may explain the superior meat quality associated with Berkshire pigs and may be due to a decreased glycolytic capacity in the homozygous pigs.

Conclusion

The AMPKγ3 V199I mutation results in a slower pH decline in homozygous mutants compared to heterozygotes. The data also suggest that the AMPKγ3 V199I mutation may be a recessive mutation compared to the dominant AMPKγ3 R200Q mutation.