

Physiology of Marbling in Beef Cattle

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Introduction

Marbling or intramuscular fat deposition in beef is a major determinant of carcass quality and value in the US. According to the National Beef Quality Audits (NBQA; Lorenzen et al., 1993; McKenna et al., 2000; Moore et al., 2012; McKenna et al., 2000) from 1993 to 2016, marbling and quality grades have increased during this time period (Fig 1). The percentage of carcasses grading Prime has increased by 64% and upper Choice (CAB) by 75%. The percentage of carcasses grading Select or Standard is 35% or 86% lower. These changes are likely related to two major factors: 1) carcass discounts and premiums based on quality grades and 2) availability of genetic information to improve carcass traits.

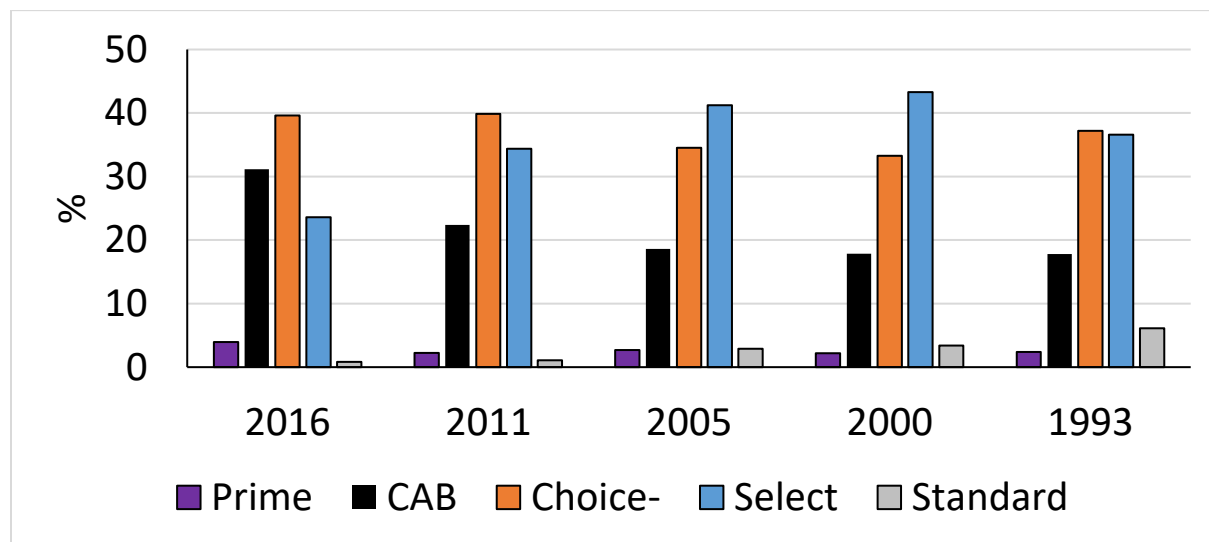


Figure 1. Percentage of Prime, Certified Angus Beef (CAB), Choice-, Select and Standard carcasses in US fed beef carcasses according to the National Beef Quality Audits (NBQA) from 1993-2016.

The Choice-Select spread can range from \$2 to \$25/cwt depending on the time of year. Premiums for Certified Angus Beef (CAB; Upper Choice) range from \$4 to \$14/cwt and for Prime range from \$9-26/cwt depending on time of year. Traditionally, the largest differentials between Choice and Select and premiums reach a peak in summer months (June/July) and again at the end of the year (Dec.). These discounts and premiums can have a significant impact on value of beef carcasses and provide incentives for improving genetic potential and altering finishing systems for greater marbling deposition.

Carcass traits like marbling are highly heritable ($h^2 = 0.48$; Angus Assoc., 2019), which means that genetic improvement can be made. Indeed if we look at the genetic change in marbling EPDs for Angus bulls, you can see the increase that has occurred during the last 20 years. Marbling score is determined on the carcass longissimus muscle at the 12/13th rib and has a strong correlation ($r = 0.79$; Wilson et al.,

1998) with chemical total fat measures. The use of real-time ultrasound has been instrumental in assisting with identification of bulls and their progeny with superior abilities to deposit intramuscular fat. Currently the average marbling EPD in the Angus breed is + 0.41 and has a range from -0.77 to +2.15. Today we have cattle with superior genetic ability to deposit marbling but we may need to rethink our traditional approach to finishing beef systems.

Accelerating Marbling Deposition

Early research (Moody and Cassens, 1968; Hood and Allen, 1973; Cianzio et al., 1982, 1985) described intramuscular fat deposition as a late developing depot compared to other adipose depots. Moody and Cassens (1968) traced marbling depots and concluded that both hyperplasia and hypertrophy were important to marbling score. Hood and Allen (1973) evaluated changes in adipocyte cell number and size in perirenal, subcutaneous, and intramuscular depots in Angus-Hereford and Holstein steers at 14 mo of age and Hereford steers at 8 mo of age. Subcutaneous and perirenal adipocytes increased in size indicating the hypertrophy was primarily responsible; however, intramuscular adipocytes were smaller in size and number of cells was positively correlated to intramuscular lipid content within and across the muscles. Cianzio et al. (1985) compared adipose cellularity and lipid concentration from 11 to 19 mo of age in steers fed a grower diet. They reported that adipocyte diameter and adipose tissue amounts in the carcasses both increased with advanced animal age indicating that adipocyte hypertrophy was primarily involved in adipose tissue deposition. However, marbling score was positively correlated to both cell diameter and total cell number suggesting that both hyperplasia and hypertrophy was important for intramuscular lipid deposition. Most of us have learned in the classroom that marbling is deposited last and the first to be mobilized in times of energy need.

Current research examining fetal programming is increasing our knowledge and understanding of muscle and adipose tissue development. Muscle and adipose tissues originate from the same mesodermal germ layer of the embryo. Muscle development begins early with primary myogenesis and followed by secondary myogenesis. In cattle, it is estimated that primary myogenesis is complete by 100 d of gestation and secondary myogenesis is complete by 180 d of gestation (Albrecht et al, 2013). Therefore, muscle fiber number is set before birth and postnatal growth is the result of hypertrophy or enlargement of existing muscle fibers. Adipose development is also occurring during this early time period but less is known regarding timing and whether hyperplasia is ever really complete. Robelin (1981) has shown that the fattening process occurred with an estimated 70% attributed to hypertrophy and 30% to hyperplasia in various adipose depots. Robelin (1981) reported that when subcutaneous adipocytes were filled with lipids that this stimulated another wave of hyperplasia to provide more adipocytes for additional lipid filling. Du et al. (2015) hypothesized that a critical window between late gestation and 250 d of age occurs for hyperplasia of intramuscular adipocytes which is followed by hypertrophy. If there are important stages of development for adipocytes, then our early management systems for calves need further examination.

Deutscher and Slyter (1978) reported higher quality grades for calves that had access to creep feed (50-50% corn and oats) in the drylot cow-calf system and then finished on concentrates. Faulkner et al. (1994) showed linear increases in quality grade of calves that received unlimited or limited creep feed prior to weaning when cows were grazing endophyte-infected fescue. In addition, Faulkner et al. (1994) found that quality grades were higher when calves were offered creep feed containing corn compared with soybean hulls in either limited or unlimited amounts prior to weaning. In contrast, Myers et al. (1999) found no difference in marbling scores or percentage grading Choice, upper Choice, or Prime when normal weaned calves were creep fed ground corn ad libitum for 55 d with dams grazing endophyte-infected tall fescue and red clover, and then finished on concentrates for 213 d.

Many researchers have evaluated early weaning (63-150 d of age) as a strategy to improve overall herd performance and potentially alter marbling deposition in calves. Early weaning of calves followed by feeding of high concentrate diets increased marbling scores and external fat thickness compared to normal weaned calves (Myer et al., 1999; Story et al., 2000; Shike et al. 2007; Meteer et al., 2013; Scheffler et al., 2014). In contrast, others reported no change in marbling score or fat thickness when early weaned calves grazed forage with supplement (1% BW; Arthington et al., 2005), fed transition diets (75% whole corn; Fluharty et al., 2000) or drylot period followed by grazing (Wiseman et al., 2019) after early weaning before entering the feedlot. Duckett et al. (2007) found that higher stocker growth rates increased marbling deposition in steers finished on concentrates. These results suggest that exposure to high concentrate diets after early or normal weaning may have the most impact on marbling deposition.

Serial slaughter studies indicate that marbling deposition occurs after 120 d on a high concentrate based diet (Greene et al., 1989; Duckett et al., 1993; Bruns et al. 2004, 2005). Early weaning and exposure to a high concentrate diet has been reported to increase marbling deposition in finished cattle (Scheffler et al., 2014); whereas carcass weights are often decreased in turn. Koch et al. (2018) reported that exposure to high concentrates for 110 d post normal weaning followed by forage finishing produced carcasses with similar marbling scores to steers fed high concentrates early and late in the finishing phase. High concentrate, corn based diets up-regulate expression of lipogenic enzymes and down-regulate expression of lipolytic genes in subcutaneous tissue (Duckett et al., 2009; Key et al., 2013; Koch et al., 2018). In contrast, corn supplementation (< 1% of body weight) on pasture (Pavan and Duckett, 2008; Greenwood et al., 2015; Wright et al. 2015) does not appear to alter marbling deposition. Researchers (McGilchrist et al., 2011; Fitzsimmons et al., 2014) have suggested that marbling deposition may be related to development of insulin resistance. Kitessa and Abeywardena (2016) reported that localization of glucose transporter (GLUT4) and fatty acid translocase (CD36) to the plasma membrane are altered in high plasma insulin states during insulin resistance. During insulin resistance, the fatty acid translocase moves to the plasma membrane creating an open gate that allows fatty acid to enter the cell; whereas glucose transporter remains inside the cytosol to limit glucose uptake into the cell and thereby elevating plasma glucose levels. A consequence of type II diabetes is heightened accumulation of intramyocellular fat in skeletal muscle of humans (Gemmick et al., 2017). More research is needed to develop a better understanding of how intramuscular lipid deposition is regulated in skeletal muscle in order to stimulate deposition earlier at the expense of excess subcutaneous fat.

Literature Cited

- Albrecht et al. 2013. *J. Anim. Sci.* 91:3666
- Angus Association. 2019. <https://www.angus.org/Nce/Heritabilities.aspx> and <https://www.angus.org/Nce/Averages.aspx>
- Arthington et al. 2005. *J. Anim. Sci.* 83:933-939.
- Boykin et al. 2017. *J. Anim. Sci.* 95(7):2993-3002.
- Bruns et al. 2004. *J. Anim. Sci.* 82:1315-1322.
- Bruns et al. 2005. *J. Anim. Sci.* 83:108-116.
- Cianzio et al. 1982. *J. Anim. Sci.* 55:305-312.
- Cianzio et al. 1985. *J. Anim. Sci.* 60:970-976.
- Deutsch and Slyter. 1978. *J. Anim. Sci.* 47:19-28.
- Du et al. 2015. *Meat Sci.* 109:40-47.

Duckett et al. 1993. J. Anim. Sci. 71:2079 – 2088.

Duckett et al. 2007. J. Anim. Sci. 85:2691-2698.

Duckett et al. 2009. J. Anim. Sci. 87:1120-1128.

Faulkner et al. 1994. J. Anim. Sci. 72:470-477.

Fitzsimmons et al. 2014. J. Anim. Sci 92:4616-4631.

Fluharty et al. 2000. J. Anim. Sci. 78:1759-1767.

Garcia et al. 2008. J. Anim. Sci. 86(12):3533-3543.

Gemmink et al. 2017. Biochim. Biophys. Acta Mol Cell Biol Lipids 1862(10 Pt B):1242-1249.

Greene et al. 1989. J. Anim. Sci. 67:711.

Greenwood et al. 2015. J. Anim. Sci. 93(8):4132-4143.

Hood and Allen. 1973. J. Lipid Res. 14:605–610.

Key et al. 2013. J. Anim. Sci. 91:2616-2627.

Kitessa and Abeuwardena. 2016. Nutrients 8:466.

Kochet al. 2018. Meat and Muscle Biology 2(1):1-14.

Lorenzen et al. 1993. J. Anim. Sci. 71:1495-1502.

McGilchrist et al. 2011. Animal 5:10, pp. 1579-1586.

McKenna et al. 2000. J. Anim. Sci. 20:1212-1222.

Myers et al. 1999. J. Anim. Sci. 77:300-310.

Meteer et al. 2013. Prof. Anim. Sci. 29:469-481.

Moody and Cassens, 1968. J. Food Sci. 33:47-55.

Moore et al. 2012. J. Anim. Sci. 90(13):5143-5151.

Pavan and Duckett. 2008. J. Anim. Sci. 86:3215-3223.

Robelin. 1981. J. Lipid Res. 22:452-7.

Scheffler et al. 2014. J. Anim. Sci. 92:320-324.

Shike et al. 2007. Prof. Anim. Sci. 23:325-332.

Smith. 2017. J. Anim. Sci. 95:2185-2197.

Story et al. 2000. J. Anim. Sci. 78:1403-1413.

Vasconcelos et al. 2009. J. Anim. Sci. 87:1540-47.

Wilson et al. 1998. A.S. Leaflet R1529, 1998 Beef Research Report Iowa State Univ., available at: <https://www.extension.iastate.edu/pages/ansci/beefreports/asl-1529.pdf>

Wiseman et al. 2019. J. Anim. Sci. 97(3):1198-1211.

Wright et al. 2015. J. Anim. Sci. 93:5047-5058.