The Biological Basis of Meat Tenderness and Potential Genetic Approaches for its Control and Prediction

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INTRODUCTION

Inconsistency in beef tenderness at the consumer level has been identified as one of the major problems facing the beef industry. Solving this problem has become one of the top concerns of the meat industry.

Some have argued that eating satisfaction results from desirable flavor, juiciness and tenderness combined and, thus, argue that we should study all three components. We recognize that palatability consists of these components and that it is a combination of these eating attributes that determines the degree of eating satisfaction. However, at the U.S. Meat Animal Research Center, we have determined that our research efforts should be focused on meat tenderness because: 1) consumers consider tenderness to be the single most important component of meat quality (Miller, 1992); 2) consumers can discern between tenderness levels and are wiling to pay a premium for tender meat (Boleman et al., 1995); 3) the coefficient of variation for sensory tenderness is twice that of juiciness and flavor (Figure 1; Koohmaraie et al., 1994; Shackelford et al., 1995; Wheeler et al., 1995a); and 4) Savell and Shackelford (1992) provided several lines of evidence indicating that beef subprimals and retail cuts are priced according to their expected tenderness. The higher palatability of a tenderloin steak relative to a round steak is due to higher tenderness and not juiciness or flavor. In fact, among 10 major beef muscles, tenderloin, which is the most highly-valued cut of beef, was one of the least juicy and least flavorful cuts (Shackelford et al., 1995). Thus, inadequate or inconsistent beef tenderness is the most likely cause of consumer dissatisfaction, and improvement in tenderness would result in higher product value.

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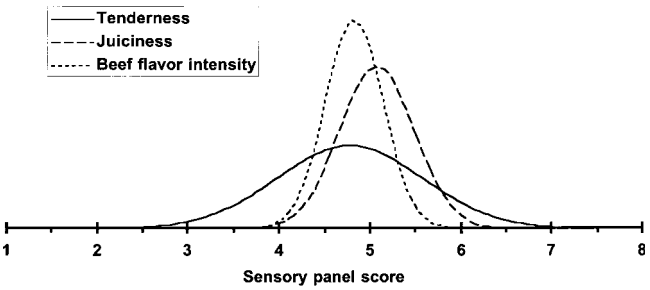
Inconsistency in meat tenderness is due to a combination of our inability to routinely produce tender meat and, perhaps more importantly, our inability to identify carcasses producing tough meat and classify them accordingly. To solve this problem, we must develop methodologies for tenderness-based classification of beef. The beef industry relies on the USDA quality grading system to segment carcasses into groups based on varying levels of expected meat palatability. However, numerous investigations of the relationship between marbling and beef palatability have shown that, although there is a positive relationship between marbling degree and tenderness, this relationship is weak at best (reviewed by Parrish, 1974). Wheeler et al. (1994a) reported that marbling explained about 5% of the variation in palatability traits and that there was both tough and tender meat within each marbling degree. Thus, there are far too many carcasses with tender meat that are discounted and far too many with tough meat that are not discounted under the current USDA quality grading system (Wheeler et al., 1994a). Any new classification system should be based on meat tenderness itself or direct predictor(s) of tenderness. Should it become necessary to use predictors of meat tenderness, the predictor(s) should explain most of the observed variation in meat tenderness as opposed to the current system in which marbling explains only 5% of the variation in beef tenderness. The objectives of this presentation are to briefly discuss the biological basis for meat tenderness and to discuss potential genetic approaches for controlling and predicting the observed variation in meat tenderness.

BIOLOGICAL BASIS OF MEAT TENDERNESS

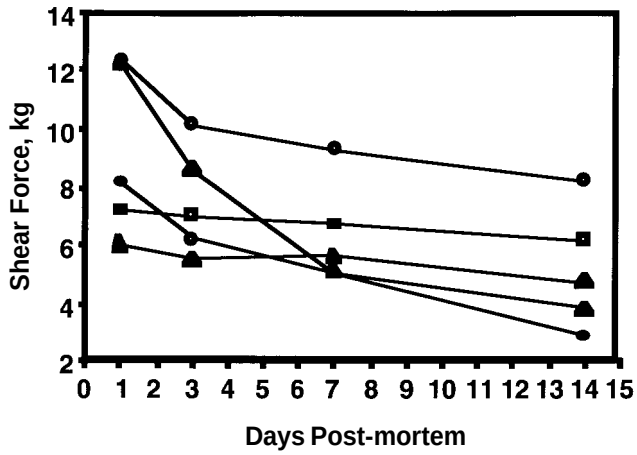
Our years of observation have indicated that there is a rather large amount of variation in extent of tenderization (shear force) after 1 day of post-mortem storage (Figure 2, as an example), which was the earliest time we had measured shear force. We had indications that these differences were created during the first 24 hr post-mortem (Koohmaraie et al., 1987) and suggested that all animals probably had the

FIGURE 1.



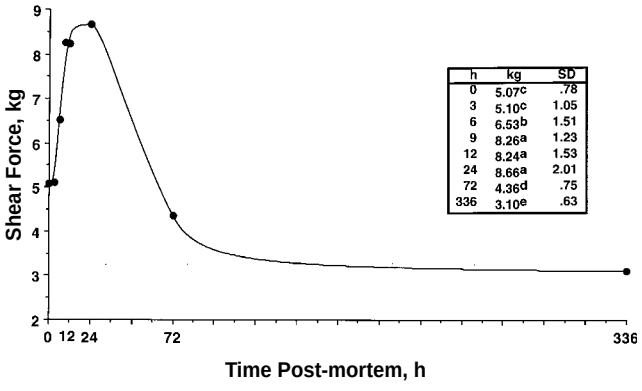
Variation in palatability attributes of beef longissimus muscle after seven days of post-mortem storage (Adapted from Koohmaraie et al., 1994).

FIGURE 2.



Effect of post-mortem storage on Warner-Bratzler shear force of longissimus muscle of five beef carcasses selected to represent different rates and extent of post-mortem tenderization (from Koohmaraie et al., 1994).

FIGURE 3.



Warner-Bratzler shear force of lamb longissimus at various post-mortem times. Means without a common superscript differ (P < .05; from Wheeler and Koohmaraie, 1994).

same tenderness level when slaughtered. To test this hypothesis, we conducted an experiment to determine the at-death shear force value of lamb in the absence of muscle shortening (Wheeler and Koohmaraie, 1994). At the time of slaughter, *longissimus* muscle has intermediate shear force. In the next several hours (i.e., rigor development), there is a large decrease in sarcomere length (from 2.24 to 1.69 μm), which is associated with a large (from 5.07 to 8.66 kg) increase in shear force (i.e., toughness). An opposite phenomenon (i.e., tenderization) also begins either at slaughter or shortly after slaughter, which results from weakening of the myofibrils caused by proteolysis of proteins responsible for maintaining structural integrity of the myofibrils (Figure 3; Wheeler and Koohmaraie, 1994). Thus, we demonstrated that there are minor inherent differences in meat tenderness at the time of slaughter and that, indeed, the observed differences in 1-day shear force are generated during the first 24 hr post-slaughter. Thus there are some animals that go through the tenderization process rapidly and could be consumed after 1 day, whereas others could be consumed after 3, 7 or 14 days, and still others would not be acceptable even after extended post-mortem storage (Figure 2). I have suggested that differences in the rate and extent of post-mortem tenderization are the cause of variation observed in meat tenderness after post-mortem storage (Figure 2; Koohmaraie 1992a, b, 1994).

The development of a method of predicting meat tenderness requires a sound knowledge of the mechanisms that regulate meat tenderness. Since the turn of the century, mechanisms of post-mortem meat tenderization have been the subject of many studies and many laboratories have contributed to our collective knowledge (for review, see Goll et al., 1983, 1991; Koohmaraie, 1988, 1992a,b, 1994; Koohmaraie et al., 1994; Ouali, 1990, 1992; Robson et al., 1991). Current evidence suggests that calpain-mediated proteolysis of key myofibrillar proteins is responsible for improvement in meat tenderness during post-mortem storage of carcasses or cuts of meat at refrigerated temperatures. These proteins (more than likely not all have been identified) are involved in inter- (e.g., desmin and vinculin) and intra-myofibril linkages (e.g., titin, nebulin, and possibly troponin-T). Although the list of the proteins involved could change over the years, I believe that the principle will stand the test of time; that is, proteolysis of key myofibrillar proteins (proteins whose function is to maintain structural integrity of myofibrils) is responsible for post-mortem tenderization. As new proteins are discovered (e.g., fulcin, Terasaki et al., 1995), their potential role in post-mortem tenderization will systematically be determined. If the differences in the rate and extent of proteolysis of these specific proteins account for differences in the rate and the extent of tenderization, then the variation in the tenderness of meat at the consumer level could be controlled by controlling these events. There is much evidence to support the above hypothesis (i.e., differences in post-mortem proteolysis are responsible for meat tenderization), some of which includes:

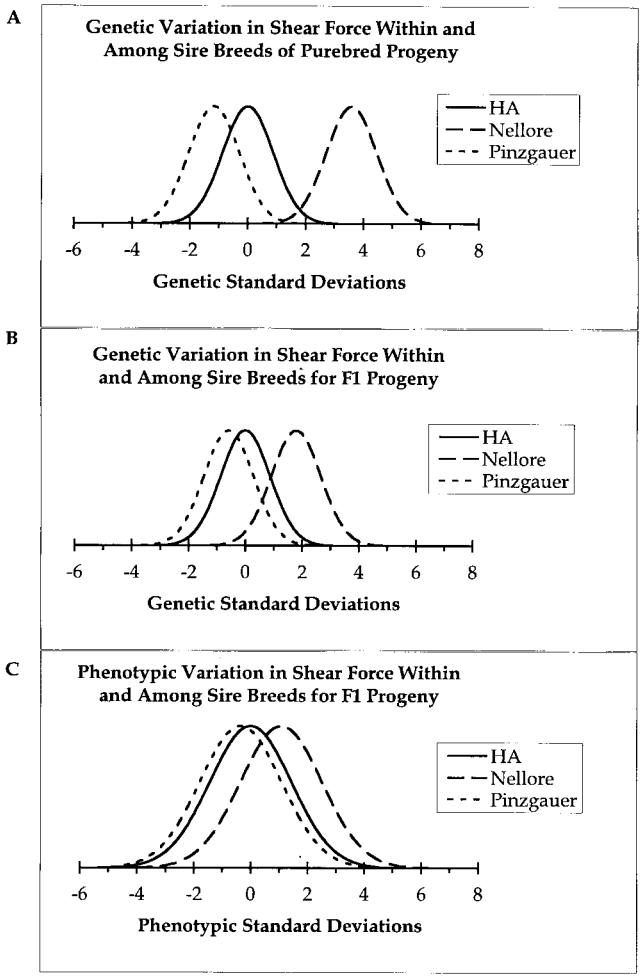
1) dietary administration of some beta-adrenergic agonists to lamb and beef reduces tenderization and proteolysis (such as L644,969 and cimaterol; for review, see Koohmaraie et al., 1991a); 2) infusion of carcasses with calcium chloride accelerates/enhances both tenderization and proteolysis (Koohmaraie et al., 1988) and zinc chloride prevents post-mortem proteolysis and tenderization (Koohmaraie, 1990); 3) animals that do not go through post-mortem proteolysis also do not go through tenderization (Koohmaraie et al., 1995a; Taylor et al., 1995). Some of our strategies on the development of methods to predict meat tenderness are based on these biochemical processes and are summarized elsewhere (Koohmaraie et al., 1994, 1995d).

GENETIC APPROACHES TO PREDICTING MEAT TENDERNESS

Many scientists and producers have suggested that controlling the genetics of the slaughter cattle population would entirely solve the beef industry's tenderness problem. I agree that genetics makes a significant contribution to the total variation in tenderness as tenderness varies among and within breeds (Figure 4; Wheeler et al., 1995a). However, analyses indicate that genetic and environmental factors make about an equal contribution to variation in tenderness. The best estimates indicate that, within a single breed, genetics controls about 30% of the variation in beef tenderness. This 30% represents the heritability (additive gene effects) of tenderness (Koch et al., 1982) within a breed. Therefore, within a breed, 70% of the variation is explained by environmental and non-additive gene effects. Between-breed variation is about equal to or less than variation within breeds. Therefore, among cattle of all breeds, approximately 46% of the variation in tenderness is genetic and 54% is environmental. Thus, significant improvement in tenderness can be made by controlling those factors responsible for the environmental effects such as time on feed (high-energy diet), stress, carcass chilling, post-mortem aging time (Figure 2), cooking method and end-point temperature, a well as through selection of breeds or genetic selection within breed.

Traditional animal breeding theory indicates that, within a breed, the most effective genetic selection is made through progeny testing. Progeny testing would not be a practical method to improve tenderness due to the time and expense required to develop and evaluate progeny. The rate of genetic improvement in a given trait is a function of the heritability of the trait, the generation interval, and the selection differential. If we make the following assumptions: use 13 sires, hold inbreeding to less than 1%, 100 cow herd size, heritability estimates of 0.30 for shear force and 0.42 for marbling, the genetic correlation 0.25 between shear force and marbling (Koch et al., 1982, and the references therein), standard deviation of 1.0 kg for shear force, then it would take 12.0 years or 40.7 years to improve shear force by 1.0 kg by selection for shear force or for marbling, respectively. If we increase the size of the cow herd to 500, the above estimates would be 6.8 and 23.1 years, respectively. Obviously, a significant change in the above parameters will affect these estimates. There is evidence to suggest that significant improvement in shear force measurement can be made (Koohmaraie et al., 1995c; Wheeler et al., 1994b; Wheeler et al., 1995b) to improve its accuracy, which may change the heritability estimate for shear force and, thus, the time required to make improvement through selection.

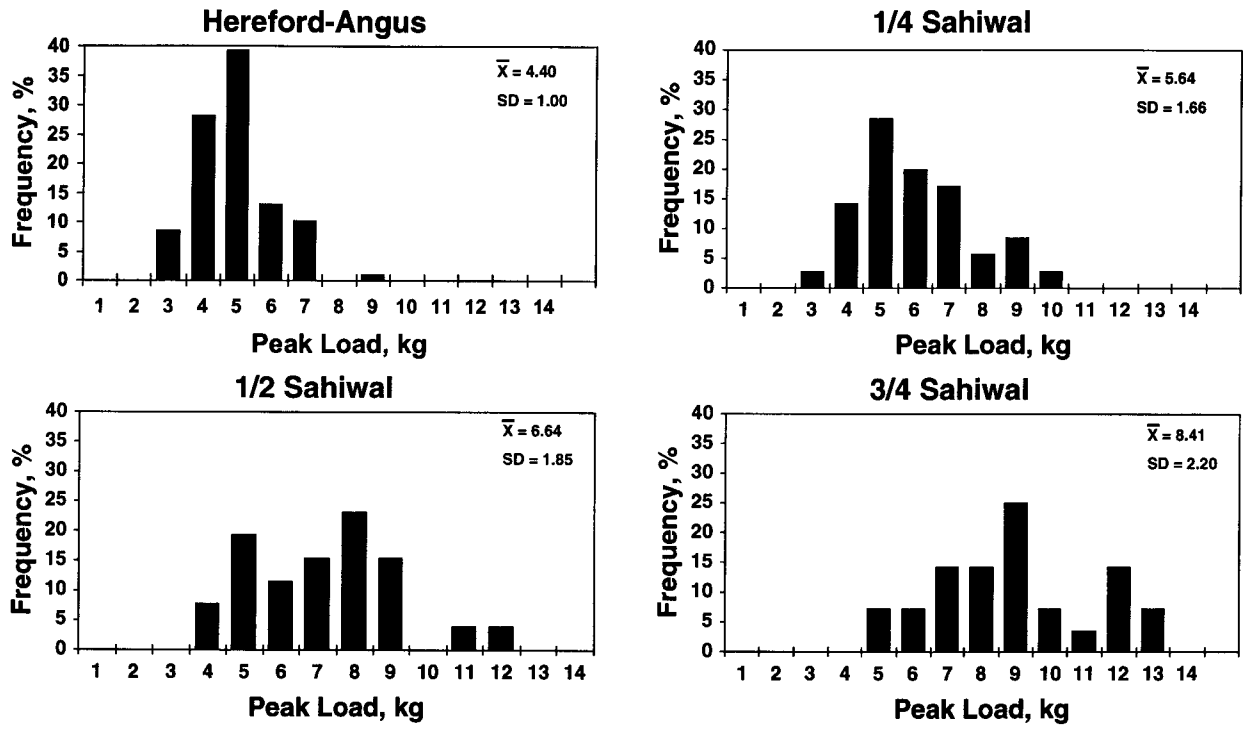
FIGURE 4.



Genetic and phenotypic variation in shear force. Variation in shear force within and among sire breeds representing Hereford x Angus (HA), Nellore (least tender), and Pinzgauer (most tender) progeny. HA was set to zero. Differences are expressed in standard deviation units as deviations from HA. A) Potential genetic variation among and within purebred progeny was obtained by doubling the differences in F1 progeny, B) Genetic variation among and within sire breeds of F1 progeny, C) Phenotypic variation among and within sire breeds of F1 progeny (from Wheeler et al., 1995a).

No doubt, focusing on the genetic components of meat tenderness through selection and progeny testing is a long-term approach to the problem and we should focus our immediate attention on other factors (i.e., time on feed, stress, post-mortem aging time, cooking method and end-point temperature) to reduce the variation in meat tenderness at the consumer level. This does not mean, and should not be interpreted to mean, that the genetic contributions to tender-

FIGURE 5. Day 7 Shear Force.



Warner-Bratzler shear force after seven days of post-mortem storage distributions for longissimus muscle from Bos taurus *and* Bos indicus *cattle (adapted from Crouse et al., 1989).*

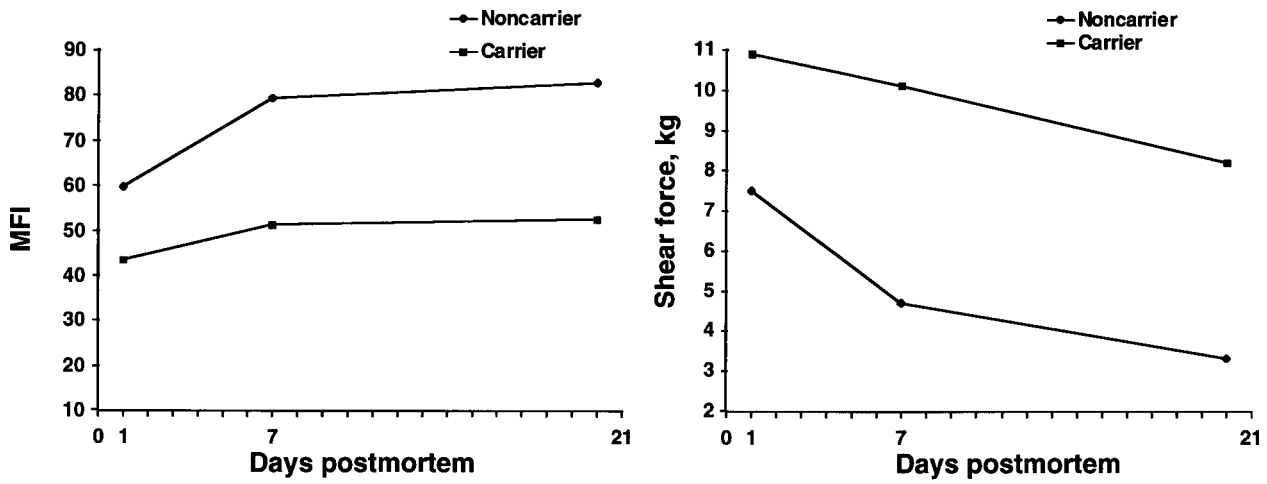
ness are not important. The major impact that genetics can have on meat tenderness is well documented. For example, it is well known that the mean shear force value and its standard deviation increases as the percentage of *Bos indicus* inheritance increases (Figure 5; Crouse et al., 1989). Another good example is the case of the callipyge phenotype in sheep. The callipyge condition is a recently identified phenotype in lamb which has a major effect on carcass composition by increasing total muscle weight by approximately 30%. However, carrier lambs produce meat that has extremely high *longissimus* muscle shear force value (248% of control), even after 21 days of post-mortem storage (Figure 6; Koohmaraie et al., 1995a,b). Thus, the application of molecular genetic approaches could hasten our ability to control the genetic aspects of meat tenderness. The genetic contribution to tenderness or any other trait can be evaluated by using the candidate gene approach and/or a whole genome approach. With the current capabilities, these two approaches are not mutually exclusive and, thus, can be pursued simultaneously.

Candidate Gene Approach

The candidate gene approach takes advantage of the existing knowledge of the biochemical basis of meat tenderness. As stated, current data indicate that calpain-mediated proteolysis of key myofibrillar proteins is responsible for post-mortem tenderization; thus, differences in the potential proteolytic activity of the calpain system result in differences in the rate and extent of post-mortem tenderization. We have collected evidence indicating that, within a species, 24 hr rather than at-death, calpastain activity is related to meat tenderness. In beef, for example, calpastatin activity at 24 hr post-mortem is highly related to beef tenderness after 14 days of post-mortem storage (for review, see Koohmaraie et al., 1995d). Among all species, at-death calpastatin activity is highly related to meat tenderness (Koohmaraie et al., 1991b; Ouali and Talment, 1990). In some special circumstances, at-death calpastatin is also related to tenderness of meat within a species, such as dietary administration of some beta-adrenergic agonists (such as L644,969 and cimaterol; for review, see Koohmaraie et al., 1991a) and expression of callipyge gene in lamb (Koohmaraie et al., 1995a,b).

The estimates for the relationship between calpastatin activity and meat tenderness vary, but up to 40% of the variation in beef tenderness is explained by calpastatin activity at 1-day post-mortem (Koohmaraie et al., 1995d). Such a high degree of association could be the justification for using calpastatin in a candidate gene approach for predicting meat tenderness. The drawbacks to the candidate gene approach are twofold. Undoubtedly, more than one gene is involved in regulation of tenderness and this approach only allows for examination of one gene at a time. Secondly, the factors affecting the expression of the gene of interest (e.g., calpastatin) could be located on an entirely different chro-

FIGURE 6.



Effect of callipyge gene on Warner-Bratzler shear force and myofibril fragmentation of lamb longissimus muscle at various post-mortem times (from Koohmaraie et al., 1995c).

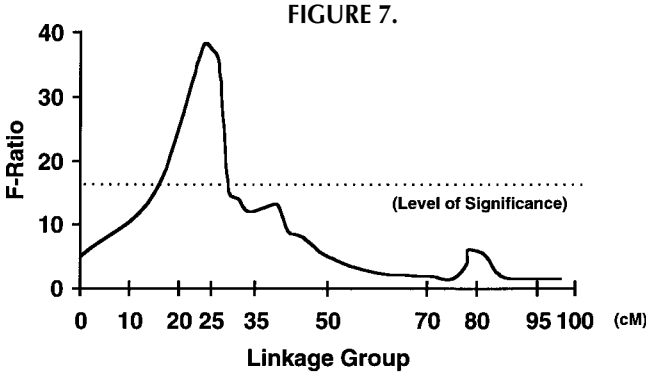
mosome; thus, such regulatory factors could not be identified in a candidate gene approach.

Genome Mapping

Perhaps the best approach for determining the genetic controls of beef tenderness and, more importantly, utilizing such information, is to use the genome mapping approach. Genetic maps are rapidly being constructed as a basis for identification of markers associated with Quantitative-Trait-Loci (QTLs) for use in Marker-Assisted-Selection (MAS) in cattle breeding programs. About 1000 markers spaced evenly throughout the cattle genome have been identified, sequenced, and used to trace the inheritance of DNA segments from parent to offspring in cattle families designed for development of a linkage map (Bishop et al., 1994). A linkage map characterizing heterozygous, well-spaced markers enables efficient selection of markers for identification of QTLs segregating in cattle resource populations. Resource populations are well-defined large families of animals having traceable heritage through pedigree analysis and segregating alleles of genes affecting phenotypic characteristics of interest (i.e., meat tenderness, carcass retail yield, etc.). These resource populations may be derived from within breed, breed crosses, or interspecies crosses. However, the type of resource population used or constructed will influence the level of heterozygosity within parental genomes. Several hundred more markers must be available for parental screening for a within-breed (such as Angus or Hereford) search of QTLs than for an interspecies cross (such as Brahman x Angus) search, due to the lower level of marker heterozygosity in the purebred genome. Depending on the objective for use of the marker information, resource populations must either be created in a research setting or identified in the field from cattle populations currently in production.

Strategies for identifying loci affecting economically important traits, in the examples cited above, have relied on the concept of "whole-genome-linkage-scanning" (Figure 7; Andersson et al., 1994). This concept is contrary to the candidate gene approach in that it allows, at the DNA level, an assessment of genetic variation at multiple intervals simultaneously with phenotypic records across all regions of the genome flanked with markers. Because of their ease of use, high utility and high throughput, microsatellites are the current marker of choice in whole-genome-linkage-scanning. Microsatellites allow rapid efficient dissection of a plant or animal genome into interval parts for determining their direct contribution to variation in quantitative and disease-related traits. A method of searching for markers involves the use of a large number of half-sibs from interspecies backcrosses involving only a few sire families. To discover what region(s) of the genome contribute to meat tenderness, phenotypic observations on tenderness (i.e., shear force) will be

FIGURE 7.



Tenderness-loci mapping using the concept of whole-genome-linkage-analysis-scanning. A hypothetical graph drawn based on the concept described by Andersson et al. (1994).

collected and associated with variation at the DNA level. Once found, markers for meat tenderness can be implemented in various MAS schemes. However, these tenderness-associated markers may only be useful for MAS in the reference population in which they were identified. In order for such information to be useful for other populations, the region(s) of the genome that are contributing to variation in meat tenderness must be sequenced (positional cloning) to determine the identify of the gene in this region(s). This information can then be used for MAS in all populations.

Experiments are already underway at the MARC to identify markers for beef tenderness and other traits; however, it is important to recognize that even with the genes regulating tenderness at hand, not all the variation in meat tenderness can be controlled. The reason is that tenderness is a trait that is highly affected by factors other than additive genetics. For example, a steak could be very tough or tender depending on the time post-mortem it was cooked, degree of doneness, etc. For this reason, a comprehensive approach is needed to consistently provide consumers with an acceptable product. The approach would include the use of the best genetics, along with sound management during growth, slaughter and processing. I believe that even after all these factors are controlled, there could be a significant percentage of animals that would still produce tough meat. These are the carcasses that do not tenderize with post-mortem storage. We must develop methodologies to identify these carcasses and then process them differently. Our approaches toward controlling meat tenderness variation and development of an objective method for predicting meat tenderness are described in detail elsewhere (Koohmaraie et al., 1994, 1995d).

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