AN ABSTRACT OF THE THESIS OF

Hajime Tanida for the degree of Doctor of Philosophy in Animal Science presented on April 28, 1987.

Title: The Inheritance of Longevity Traits in Beef Cattle

Abstract approved: Redacted for Privacy
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Thirty years and 23 years of life history data from a Hereford herd in Arizona and an Angus herd in Wyoming were analyzed. The Hereford herd averaged 325 ± 20 cows and 98 ± 6 replacement heifers per year, while the Angus herd averaged 672 ± 21 cows and 110 ± 9 replacement heifers. Longevity traits averaged 4.21 ± 0.06 for years from first calving to disposal (FST), 7.40 ± 0.06 for years from birth to disposal (AGE) and 3.46 ± 0.06 for lifetime number of calves weaned (NUM) in the Hereford herd and 4.49 ± 0.13 (FST), 6.68 ± 0.12 (AGE) and 3.66 ± 0.11 (NUM) in the Angus herd. Heritabilities of longevity traits and genetic correlations among them were estimated from daughter-dam regression and paternal half-sib analysis, in which longevity traits were adjusted for significant environmental sources of variation. In the Hereford herd, heritability estimates for longevity traits ranged from 0.16 to 0.26. In the Angus herd, heritability

estimates of longevity traits from daughter-dam regression ranged from 0.03 to 0.05, whereas those from paternal half-sib analyses were from 0.62 to 0.69. The paternal half-sib estimates were unreliable because sire and birth year effects largely were confounded. In the Hereford herd, genetic correlations between either birth weight or weaning weight and longevity traits from daughter-dam regression were negative and generally of low magnitude, whereas the estimated genetic correlations between weaning condition score and longevity were positive and moderate. Analogous estimates from paternal half-sib analyses all were positive and moderate to high. In both herds, phenotypic correlations between early life traits and longevity traits all were low, ranging from -0.04 to 0.16. In the Angus herd, age-specific survivorship (Lx) and age-specific survival rates (Px) were calculated for several sires' daughters. The Lx and Px curves varied markedly among sires. The differences were largely expressed before daughters were 6 years of age. This study suggested the existence of moderate genetic variation for longevity traits in beef cattle.

The Inheritance of Longevity Traits in Beef Cattle

by

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A THESIS

submitted to

Oregon State University

in partial fulfillment of
the requirements for the
degree of

Doctor of Philosophy

Completed April 28, 1987

Commencement June 1987

APPROVED:

Redacted for Privacy

Professor of Animal Science in charge of major

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Date thesis is presented April 28, 1987

ACKNOWLEDGEMENTS

Thank you very much.

Special thanks to Dr. W. D. Hohenboken (or Bill) and Yumiko.

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CHAPTER 1

INTRODUCTION

Longevity of beef cows can be defined as reproductive lifespan or length of time that a cow remains productive (O'Mary and Dyer, 1978). Average longevity of a herd influences economic returns by affecting the proportion of female offspring that must be retained as replacements (and therefore the proportion available for sale) and the annual depreciation cost per cow. Herd age structure, dependent upon longevity, also will influence average calf weaning weights. Finally, longevity determines generation interval and therefore influences the rate of genetic change possible in the herd.

Compared with the dairy industry, beef herds often lack complete production records and pedigree information to make longevity studies possible, and estimates of genetic parameters for longevity traits in beef cattle have not been reported. According to dairy studies, genetic variation for longevity exists but heritability generally is low, in the proximity of 0.1 to 0.2.

This thesis starts with a literature review which covers biological theories of aging and longevity, then studies on the inheritance of longevity in dairy cattle, beef cattle and sheep.

Objectives of research reported herein were 1) to characterize two beef cattle populations for certain traits indicative of longevity or herd life, 2) to estimate heritabilities of the traits, 3) to estimate genetic and phenotypic correlations among the longevity traits and between longevity and traits measured early in a cow's life and 4) to compare survival patterns of paternal half sib groups of cows by graphical examination of life history statistics (Schons et al., 1985).

CHAPTER 2

LITERATURE REVIEW

Longevity and Aging

Introduction

In the history of science, art, philosophy and religion, I believe that the secret of life and death has always been the main theme to be solved. Scientists have been trying to define life by shuffling test tubes, while alchemists have sought a key to eternal life. Artists have been expressing joy, anger, sadness, anguish and beauty of life and death by means of painting, music, novels, etc. Philosophers have kept thinking of the significance of life and trying to differentiate soul from body. Religionists have attempted to free people from the anxiety of death and have advocated eternity of the soul. Each group has been approaching one objective, "the secret

of life", but by different means. That reminds me of a game in which blindfolded people touch a huge statue with their hands and try to guess what it is. Since each person cannot touch the whole object, their answers would never be the same, but all could be right.

In the field of science, numerous studies of aging and longevity have been done. The objective of this chapter is to review some of the previous research on aging and longevity and to shed a bit of light on the statue of life and death.

Definitions of Longevity and Aging

Longevity is generally considered to be the time span from birth to death of an organism, and the process of aging is one of the factors which determines longevity.

Numerous definitions of aging have been proposed. Sharma and Talukder (1979) regard aging as occurring from fertilization onwards. It is progressive and eventually harmful, irreversible under usual conditions and accompanied by alterations in functional capacity which result in the loss of the organism's ability to repond to environmental changes. Sonneborn (1978) defined aging as

inherent, progressive, irreversible impairments of function. It occurs not only in higher organisms but also as far down the scale of beings as some but not all unicellular organisms. Medawar (1952) regarded it as those changes of the bodily faculties, sensibilities and energies which rendered the individual progressively more likely to die from accidental causes of random incidence. Comfort (1964) described it as a decrease in viability and an increase in vulnerability. Strehler (1977) defined it as the changes which occurred, generally in the post reproductive period, and which resulted in a decreased survival capacity on the part of the individual organism. He also pointed out that it was necessary to define a living system in order to define aging adequately.

Theories of Aging

Numerous theories of aging have been proposed. There is probably no single cause of or explanation for aging, and theories are not necessarily mutually exclusive. Sonneborn (1978) explained it as follows : "In the search for a general basis or cause of aging, the first thing one

is struck with is the multiplicity of theories. A recent reviewer made the comment that there are almost as many ideas and theories about aging as there are researchers in the field.--- Each researcher is a specialist, focusing his attention on a particular part of the whole subject."

In spite of this fact, most gerontologists agree on a general hypothesis that aging is associated with cellular information loss (Sharma and Talukder, 1979). If so, theories of aging could be classified according to two hypotheses: (1) It is caused by the random accumulation of chemical noise in the cellular information system, and is itself unprogrammed; (2) It is a built in consequence of differentiation, caused by the switching-off of synthetic processes which cannot be switched on again without loss of differentiation (Comfort, 1968). In other words, the major controversy in theories of aging is whether the changes in the genetic material are induced or programmed (Sharma and Talukder, 1979).

Theories of Random Accumulation of Induced Damage

The somatic mutation theory was first proposed in definite terms by Failla (1958), Curtis and Gebhard (1958)

and Szilard (1959). It postulates that the net effect of accumulated mutations in somatic cells contributes to aging deterioration. The theory was supported by experiments which showed that there is an acceleration of the aging process when an animal receives a non-lethal dose of irradiation (Curtis and Gebhard, 1958; Stevenson and Curtis, 1961; Curtis, 1963; Curtis and Crowley, 1963). However, Lints (1978) objected that if the frequency of mutations in the somatic cells is the same as it is in the germ cells, that frequency seemed inadequate to account for senescence. Failla (1958), on the other hand, assumed that in man the spontaneous gene mutation rate in somatic cells was approximately 12 times greater than that in germ cells.

Orgel (1963) proposed an error-catastrophe theory. The basic idea of this theory is that the ability of a cell to produce its complement of functional proteins depends not only on the correct genetic specification of the various polypeptide sequences, but also on the competence of the protein-synthetic apparatus. Deterioration of a cell may be through a progressive decrease in the adequacy of its transcription mechanism as well as through the accumulation of somatic mutations. The basic concept of Orgel's error-catastrophe theory, however, is the same as the mutation theory. The primary

events are different, but the end results are identical (Lints, 1978).

In the mutation theory, the primary event is badly coded DNA and in the error-catastrophe theory it lies in the malfunctioning of the protein-synthetic apparatus, at the level either of transcription or of translation. The error-catastrophe theory was experimentally supported by Harrison and Holliday (1967), who reported a decreased adult life span for Drosophila melanogaster when fed amino acid analogs in the larval stage to induce errors in protein synthesis.

Theories of Genetically Programmed Aging

A major theory of genetically programmed aging could be stated as: aging proceeds according to a genetically determined program, which is manifested as a gradual deterioration of the organism, senescence proceeding in much the same way as growth and differentiation. Aging becomes manifested during morphogenesis (Samis, 1978). In other words, aging is an essential process in differentiation, or a built in consequence of differentiation. If this were the case, Comfort (1968)

suggested that the only likely way of prolonging vigor would be through stretching the developmental program as a whole by the elongation of immaturity. From this point of view, McCay et al. (1935, 1939) showed that dietary retardation of growth in rats lengthened their life-span. However, since their experiments were based solely on measuring the relationship between diet and aging, it could not be determined whether the slow growth rate or the low metabolic rate increased longevity of the rats.

Roberts (1961) compared a strain of mice selected for high 6-week weight (i.e. a fast growth rate or large strain) with a strain selected for low 6-week weight (i.e. a slow growth rate or small strain). The mean life span of the small strain exceeded that of the large strain by approximately 6 months. The large strain had a short reproductive life, producing only 4.5 litters, against approximately 11 in the small strain. As a consequence, the small strain females eventually weaned almost twice as many offsprings per lifetime as the large strain individuals. Eklund and Bradford's selection experiment in mice (1977), where the strain of mice with rapid 3-6 week body weight gain had a life span only 57% that of those from the control line, further supported the study of Roberts.

Rose (1984a) suggested the possibility of

antagonistic pleiotropy for the evolution of senescence. Several experiments (Rose and Charlesworth, 1980, 1981; Rose 1984b for example) showed that selection for increased later reproductive output was associated with decreased early reproductive output, lowered overall reproductive rate, and increased longevity in Drosophila melanogaster. Rose et al. (1984) attempted to postpone senescence in Drosophila by selection and found a substantial reduction in early ovary weight among flies from the population with genetically postponed senescence.

Some researchers have approached the study of senescence by the method of cell culture. Carrel (1912) demonstrated that cells derived from chick heart tissue could be propagated indefinitely in vitro. There are also well known immortal cell populations such as HeLa (derived from a human cervical carcinoma in 1951) and L cells (derived from mouse connective tissue in 1943) which are flourishing at present. However, these results do not establish the potential immortality of vertebrate cells, because the founder cells were abnormal. Most immortal cell lines lack either the exact number or the precise morphology of chromosomes characteristic of the cells composing the tissue from which they originally descended, and they are not characteristic of any cell type found in human or animal tissue (Hayflick, 1977).

It was found that cultured normal human embryonic cells had a finite lifetime in vitro, degenerating after about 50 subcultivations or one year in culture (Hayflick and Moorhead, 1961). These authors hypothesized that the phenomenon was attributable to intrinsic factors which are expressed as senescence at the cellular level. Hayflick (1965) reported that human diploid cell strains derived from adult lung had a significantly lower doubling potential in vitro than did fetal strains. When fibroblasts derived from human embryos are stored in liquid nitrogen, they exhibit a remarkable "memory". If they are frozen at the 20th population doubling and later thawed, they will undergo 30 more doublings and then stop (Hayflick, 1980).

Before the cultured cells stop dividing, functional losses already have taken place, and proliferative activity declines (Absher et al., 1974; Cristrofalo and Sharf, 1973; Macieira-Coelho et al., 1975).

Hayflick (1977) suggests that functional losses which occur in cells, prior to loss of their ability to divide, produce age changes in animals long before those normal cells have reached their maximum divisional limit, and the death of cells and the destruction of tissues and organs is a normal part of the developmental sequence in animals. Some examples are the primitive kidney of higher

vertebrates, the tail and gills of tadpoles, larval organs in insects, and in many vertebrates, the thymus.

The theories of random accumulation of induced damage and genetically programmed senescense are probably not mutually exclusive. That will become more clear in the next section on aging from an evolutionary point of view.

Significance of Aging and Death

There are many theories of aging, and they were not necessarily established from an evolutionary point of view. Rather, each was developed from the author's particular field of interest. However, aging or death ought to have an evolutionary explanation like other biological phenomena.

Dawkins (1976) postulated that the gene was the unit of selection and stated that rapid and accurate replication was necessary to assure the survival of genes throughout generations. The maintenance of accuracy can be achieved only through the expenditure of energy (Kirkwood, 1977). For instance, there are corrective mechanisms such as direct proof-reading and kinetic proof-reading of genetic codes (Hopfield, 1974). Erroneous proteins already

produced can also be removed by means of a "suicide" protein (Orgel, 1963; Holliday, 1975). From the point of view of strategies to reduce energy costs, Kirkwood (1977) stated as follows: "Accuracy in the germ line is vital for gene survival but a high level of accuracy in somatic cells may be a luxury our genes do better to forgo. Aging may, therefore, be the result of an energy-saving switching off of the mechanisms responsible for high accuracy in the translation apparatus at or around the time of differentiation of somatic cells from the germ line." Kirkwood (1977) stated further that this strategy was evolutionarily stable, as defined by Maynard-Smith and Price (1973), such that, if most of the members of a population adopt the strategy, there is no "mutant" strategy that would give higher reproductive fitness. Immortal life by maintaining high transcriptional and translational accuracy in all cells would carry the penalty of an increased energy requirement. Laughrea (1982) postulated that death would occur not because the accumulated errors were too numerous but because the errors were numerous enough such that too much energy would be needed to degrade faulty proteins, leaving little for proper functioning of cells. By these theories, aging or death is presumed to be an energy efficient mechanism to transmit genetic information (DNA) to the next

generations, and this would explain its evolution. Organisms which happened to have a death mechanism possibly had selective advantages over ones which did not.

Longevity and DNA

When does life of an organism begin? Is it at fertilization? Is it at the time when the embryo is formed? The statement of Curtis and Barnes (1985) is quoted here. "In the evolutionary sense, however, none of these events marks the beginning of life. Life began more than 3 billion years ago and has been passed on since that time from organism to organism, generation after generation, to the present, and stretches on into the future, further than the mind's eye can see. Each new organism is thus merely a temporary participant in the continuum of life." It is sometimes said that a hen is only an egg's way of making another egg.

It is analogous to a car-car owner relationship (Fig.1). A car represents a living organism and the car owner its DNA. Let us suppose that one person buys a car (organism) which has a 3 year guarantee period. Another person also buys the same model. The first person sells

the car and then buys a new car of the same model every 3 years. The second keeps the same car for 10 years without buying a new one. The car owner who kept his car for 10 years owns an old style car and possibly spent more money maintaining and repairing the car than the cost of buying two additional brand new cars. In addition, the car can only be sold at a giveaway price. On the other hand, the car owner who exchanged his car every 3 years spent little for repairs and as a bonus, he had a car with newer design, better equipment and quality (from genetic variation and natural selection), and less depreciation.

Longevity and aging probably matter to sentient human beings and create anxiety among us, but not to DNA (genetic material) itself. Each living organism, including a human being, can be thought of as merely a protective shell or a dust cover for DNA which is itself a dictator or boss. Skeletons of animals and deserted shells of organisms remind me of cars scraped by their drivers.

Differences in Longevity among Living Organisms

Aging and longevity are dependent upon differences in survival strategies among and within species, in

surrounding environments and habitats and in interactions among them. Sacher (1959) documented a linear relationship between the logarithm of life span and the logarithms of brain weight and of body weight, and showed that this regression accounted for 85 percent of the variance in total life span among 63 species. Based on various results, Sacher (1978) concluded that longevity of mammals was a constitutional characteristic. Over the course of evolution of many mammalian taxa, there has been a trend toward increased body size and brain size and decreased reproductive rate. This has been accompanied by an increase of life span over the course of evolution. Strehler (1977) suggested that some groups of plants and animals have persisted evolutionarily by increasing the durability of the individual carriers of the genetic material rather than by merely increasing their number (e.g., high rate of reproduction). Their adaptations were frequently the production of more resistant physical structures (e.g., the thick bark on long lived sequoias, the heavy shells on tortoises). These adaptational stratgies indirectly increased life span of the organims.

Each type of DNA wears a different type of coat, varying from a cheap one to a durable and expensive one. Some DNA has the strategy to buy a cheap one every five years; on the other hand, some buy an expensive one every

twenty years, depending on their life style, economics and surrounding environment. Each living organism lives in a different fashion. Some have a short life cycle, and others have a long life cycle, but it does not matter, from an evolutionary perspective, as long as the DNA survives.

The car-car owner analogy could be applied again (Fig. 2). In this case, each person buys a different quality of car. One might buy a Volvo (which is supposed to be a high quality car). Another one might buy a VW Rabbit (which is supposed to be a standard quality car). The person who bought a Volvo spent 3 times as much money as the one who bought a VW, but it lasted without problems 3 times as long as did the VW. The important point is that both DNAs have driven safely for ten years.

Hayflick (1980) suggested that the evolutionary success of a species depends only on the ability of its members to live long enough to procreate and raise their young. What happens after that is irrelevant to the survival of the species. Dobzhansky (1958) made the following analogy: "Some cheap watches are sold with a "guarantee" that they will function for a year, or for three months, or for some other period. However, most of these watches continue to function for some time - occasionally for a considerable period - after the

expiration of the guarantee. Surely, this is not surprising, since a watch designed to stop running immediately after the expiration of the guarantee period would have to be a very precise mechanism, difficult and expensive to make. Some of us old-timers are, then, like the cheap watches that continue ticking, beyond their guaranteed time."

Summary

Man is also mortal. Death or termination of life creates anxiety among us. However, death or aging is one of the necessary mechanisms of living organisms from an evolutionary point of view. DNA tries to be immortal and DNA controls us. For economical and efficient survival of DNA, DNA exchanges it's old clothes for brand new ones. The old clothes are discarded. That is the end of an individual organism's life, but not the end of it's DNA.

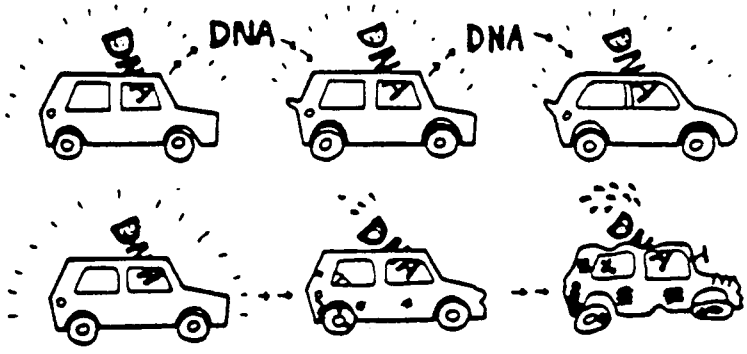


Figure 1. Importance of mortality in living organisms from the point of view of DNA.

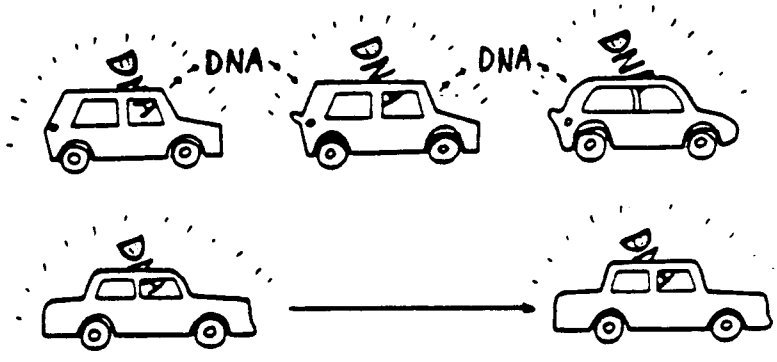


Figure 2. Difference in longevity among living organisms.

References

Absher, P. M., R. G. Absher and W. D. Barnes. 1974. Geneologies of clones of diploid fibroblasts. Exp. Cell. Res. 88:95.

Carrel, A. 1912. On the permanent life of tissues outside of the organism. J. Exp. Med. 15:516.

Comfort, A. 1964. Aging: The Biology of Senescence. Holt, Rinehart and Winston, Inc., New York.

Comfort, A. 1968. Feasibility in age research. Nature 217:320.

Cristofalo, V. J. and B. B. Sharf. 1973. Cellular senescence and DNA synthesis. Exp. Cell. Res. 76:419.

Curtis, H. J. 1963. The late effects of radiation. Proc. Am. Phil. Soc. 107:5.

Curtis, H. J. and C. Crowley. 1963. Chromosome aberrations in liver cells in relation to the somatic mutation theory of aging. Radiat. Res. 19:337.

Curtis, H. J. and K. L. Gebhard. 1958. Radiation-induced aging in mice. Proc. 2nd. Int. Conf. on Peaceful Uses of Atomic Energy. 22:53.

Curtis, H. and N. S. Barnes. 1985. Invitation to Biology, 4th edition. Worth Publishers, Inc., New York.

Dawkins, R. 1976. The Selfish Gene. Oxford Univ. Press. Oxford.

Dobzhansky, T. 1958. Genetics of homeostasis and of senility. Ann. N. Y. Acad. Sci. 71:1234.

Eklund, J. and G. E. Bradford. 1977. Longevity and lifetime body weight in mice selected for rapid growth. Nature 26:48.

Failla, G. 1958. The aging process and cancenogenesis. Ann. N. Y. Acad. Sci. 71:1124.

Harrison, B. J. and R. Holliday. 1967. Senescence and the fidelity of protein synthesis in Drosophila. Nature 213:990.

Hayflick, L. 1965. The limited in vitro lifetime of human diploid cell strains. Exp. Cell. Res. 37:614.

Hayflick, 1. 1977. The biology of aging. Nat. Hist. 7:22.

Hayflick, L. 1980. The cell biology of human aging. Sci. Amer. 242:58.

Hayflick, L. and P. S. Moorhead. 1961. The serial cultivation of human diploid cell strains. Exp. Cell. Res. 25:585.

Holliday, R. 1975. Growth and death of diploid and transformed human fibroblasts. Fedn. Proc. 34:51.

Hopfield, J. J. 1974. Kinetic proofreading: A new mechanism for reducing errors in biosynthetic processes requiring high specificity. Proc. Nat. Acad. Sci. USA. 71:4135.

Kirkwood, T. B. L. 1977. Review article: Evolution of ageing. Nature 270:301.

Laughrea, M. 1982. On the error theories of aging: A review of the experimental data. Exp. Geront. 17:305.

Lints, F. A. 1978. Genetics and Aging. S. Karger. New York.

Macieira-Coflho, A., E. Loria and L. Berumen. 1975. Relationship between cell kinetic changes and metabolic events during cell senescence in vitro. In: Cell Impairment in Ageing and Development. Plenum, New York.

Maynard Smith, J. and G. R. Price. 1973. The logic of animal conflict. Nature 246:15.

McCay, C. M., M. F. Crowell and L. A. Maynard. 1935. The effect of retarded growth upon the length of life span and upon the ultimate body size. J. Nutrition 10:63.

McCay, C. M., L. A. Maynard, G. Sperling and L. L. Barnes. 1939. Retarded growth, life span, ultimate body size

and age changes in the albino rat after feeding diets restricted in calories. J. Nutrition 18:1.

Medawar, P. B. 1952. An unsolved problem of biology. H. K. Lewis & Co Ltd, London.

Orgel, L. E. 1963. The maintenance of the accuracy of protein synthesis and its relevance to ageing. Proc. Natn. Acad. Sci. USA 49:517.

Roberts, R. C. 1961. The lifetime growth and reproduction of selected strains of mice. Heredity 16:370.

Rose, M. R. 1984a. Review: The evolution of animal senescence. Can. J. Zool. 62:1661.

Rose, M. R. 1984b. Laboratory evolution of postponed senescence in Drosophila Melanoguster. Evolution. 38:1004.

Rose, M., and B. Charlesworth. 1980. A test of evolutionary theories of senescence. Nature 287:141.

Rose, M. R., and B. Charlesworth. 1981. Genetics of life history in Drosophila melanogaster. II. Exploratory selection experiments. Genetics 97:187.

Rose, M. R., M. L. Dorey, A. M. Coyle, and P. M. Service. 1984. The morphology of postponed senescence in Drosophila melanogaster. Can. J. Zool. 52:1576.

Sacher, G. A. 1959. Relation of lifespan to brain weight and body weight in mammals, In :Ciba Foundation Colloquia on Ageing, Vol.5, The Lifespan of Animals. Churchill, London.

Sacher, G. A. 1978. Evolution for longevity and survival characteristics in mammals, In: The Genetics of Aging, edited by E. L. Schneider. Plenum press, New York & London.

Samis, H. V. 1978. Molecular genetics of aging, In : The Genetics of Aging, edited by E. L. Schneider. Plenum press, New York & London.

Sharma, A. and G. Talukder. 1979. Genetical models of senescence in man. Nucleus 22:128.

Sonneborn, T. M. 1978. The origin, evolution, nature, and causes of aging, In: The Biology of Aging, edited by

Behnke, J. A., C. E. Finch and G. B. Moment. Plenum press, New York & London.

Stevenson, K. G. and H. J. Curtis. 1961. Chromosomal aberrations in irradiated and nitrogen mustard-treated mice. Radiat. Res. 15:774.

Strehler, B. L. 1977. Time, Cells and Aging. 2nd edit. Academic press, New York.

Szilard, L. 1959. On the nature of the aging process. Proc. Natn. Acad. Sci. USA 45:30.

Longevity Studies in Dairy Cattle

Introduction

Longevity is a characteristic of potential economic importance in dairy cattle. The primary goal for commercial producers is economically efficient milk production. Increased longevity may have potential advantages for them. Those are a reduction in the annual cost of raising or purchasing replacement heifers and an increase in lifetime production per cow.

The potential disadvantages of increasing cow longevity are a reduction in annual genetic progress in milk production by increasing the generation interval of a herd, and increased health problems and involuntary culling with age.

From a genetic and economic standpoint, confusion about longevity traits exists among dairy producers and researchers. Therefore, a review of longevity studies in dairy cattle has been undertaken to evaluate the economic impact of longevity on lifetime profitability, and to evaluate evidence for the existence of genetic variation

in longevity and of phenotypic and genetic covariation of longevity with production traits.

Definitions of Longevity in Dairy Cattle

Longevity in dairy cattle is generally defined as the length of productive life in the herd. The biological ability to live a long life is not necessarily the same as commercial herd life. Longevity in a dairy herd is the same as herd life, which is influenced by general health, fertility and culling decisions based on production and other factors (Schmedt and Van Vleck, 1974).

Longevity in dairy cattle has been measured in several ways. These include age at last calving, number of lactations, length of life from first calving to disposal, age at disposal, number of successful parturitions and stayability to a specific age. Hudson and Van Vleck (1981) defined stayability as the probability of surviving to a specific age, given the opportunity to reach that age. The specific ages of 36, 48, 60, 72 and 84 mo have generally been used, because these ages are assumed to coincide with periods of management decision making with respect to culling (Van Doormaal et al. 1985). Each measurement of

longevity has inherent limitations, for example, age at last calving does not indicate whether a cow had a lactation after her last calving or how many lactations she had in her lifetime. On the other hand, number of lactations does not indicate how old a cow was when she had her last lactation. Stayability to a specific age also has an inherent problem since its "all or none" scoring of a trait has the effect of reducing information (Smith and Quaas, 1984). For example, in stayability to 48 months, two cows surviving 36 months and 47 months are considered equally dead, and two cows surviving 49 months and 60 months are treated equally alive, masking much of the variation in life span. This causes substantial information loss. More than one kind of measurement often is used in longevity studies, in order to compensate for limitations of the various measurements.

Economic Importance of Longevity

What is the relationship between longevity and lifetime profitability? Economic return from individual cows represents the net effects of several performance traits besides milk production (Gill and Allaire, 1976a).

Burnside et al. (1984) defined lifetime profitability as a function of production per lactation, length of productive life (longevity), age at first calving, calving interval and input and output prices. In the present review, the effect of longevity traits on lifetime profitability mainly will be considered.

As stated earlier, a potential reason for the desirability of longer herd life is reduction in the annual cost of raising or purchasing replacement heifers. Other factors related to length of herd life such as number of profitable lactations, genetic improvement for milk production and physiological effects of age on production and cost of production (Congleton, 1984) would also affect lifetime profitability. To find the optimum length of herd life (if such a thing exists), the ideal balance among these factors must be determined, although it is not simple. The tools to help us find this are simulation studies.

Rendel and Robertson (1950) studied four possible ways in which increased longevity might increase profits. These were : 1) by reducing the annual cost of replacements per cow in the herd; 2) by increasing the average herd yield, through an increase in the proportion of cows in the higher producing age-groups; 3) by reducing the number of replacements which have to be reared each

year, and therefore allowing an increase in size of the milking herd for a given land area and; 4) by an increased production as a response to an increase in voluntary culling. After the economic study, however, they concluded that the effect of improved longevity on milk production would be small and that longevity had little economic value compared with high yield in the commercial herd. An increase of one lactation in average productive life, according to their projections, would raise the herd mean yield by less than 1 percent. In their experiment, replacement costs accounted for only about 8 percent of total costs of milk production.

Renkema and Stelwagen (1979) also modelled the economic significance of long herd life. In their results, a herd showing average production and an average herd life of 5.3 lactations had approximately 30 percent more earned income each year than a similar herd with an average productive life of 3.3 lactations.

Pearson and Freeman (1973) simulated four cow culling systems, cross classified with three sire selection schemes. In the present review, only two cow culling systems with two sire selection schemes are compared to simplify presentation of their results. The criterion of selection in all cow systems involved only milk production. Two lengths of herd life were generated, short

and long, by two cow culling systems. Average ages at calving, average ages when cows were sold and average numbers of calvings per cow in the long life and short life herds were 60.0 mo vs 39.7 mo, 81.9 mo vs 54.2 mo and 4.8 vs 2.6, respectively. Two sire selection systems were (1) best sires : fastest genetic gain possible and (2) breed-average sires.

Their results suggested that there was no one definite length of herd life which would always earn the most profit. Optimum length of herd life was variable depending on the choice of sires and the cost of rearing replacement heifers.

It was stated earlier that long herd life compared to short herd life could result in lower annual genetic improvement in milk production because of a longer generation interval. However, this is not always true. In their study, for instance, if the choice of sires was from those close to the breed average, there was virtully no difference between short herd life and long herd life in annual genetic improvement for milk production. On the other hand, if the choice was for the best available sires, then short herd life had an advantage over long herd life in annual genetic progress for milk production because generation interval becomes more important as the rate of genetic improvement from sire selection increases.

In their study, a change from high to low rearing costs drastically affected the relationship between profitability and length of herd life. At low rearing costs, the economic advantage of long herd life resulting from raising fewer replacements was minimized. At high rearing costs, even though the profit of both long herd life and short herd life herds decreased, the reduction of profit in short herd life herds was extreme, and the economic advantage of long herd life was increased.

Congleton and King (1984) conducted a simulation study to determine the effect on profitability of extending cow herd life. The simulation was conducted on a long term basis. All runs simulated 30 yrs in a herd of approximately 80 cows which produced their own replacements selected by a pedigree index. The model incorporated estimates of the relationships between cow age and milk production, labor requirements, health costs, reproductive disease, mastitis and fertility. Culling of cows was on an estimated cost per unit of production. Cows were bred by artificial insemination with a time trend for sire genetic merit. Increasing cow herd life was achieved in the simulation by lowering the culling criteria for cow profitability.

Short herd life was expected to result in more annual genetic progress for milk production in the short term, as

compared to long herd life because of a faster turnover rate. Their study indicated that herds with a shorter herd life maintained their genetic superiority through 30 yrs but that the rate of improvement in genetic merit of those herds was slower than in herds with longer herd life. That is, the upward slope of herd genetic merit plotted against time was steeper for longer herd life than for shorter herd life simulations. Differences between the shortest and longest herd life groups at the end of simulation study (30th yr.) were significantly smaller than differences near the beginning of the simulation study (6th yr.). Herds with shorter herd life would have greater intensity of culling among cows, but herds with longer herd life would have greater intensity of pedigree selection among replacement heifers. The net effect was that intensity of cow culling had a limited impact on herd genetic merit on a long term basis.

Other than the difference in genetic improvement, the results also suggested that shorter herd life would result in a lower incidence of mastitis and twin calving, shorter calving intervals and fewer days dry. Extending herd life, on the other hand, decreased replacement cost, cull cow income and milk production per cow per year, and increased excess heifer income and cow health cost.

Incorporating all these factors, they attempted to

find the most profitable length of herd life. Their results showed that increasing herd life from 2.6 to approximately 4 lactations resulted in a linear increase in income per cow. From 4 to 5.5 lactations, average income remained relatively constant, with a slight decrease in income for herds with herd life greater than 5.5 lactations. Since average herd life within the U.S. dairy industry is about 3 lactations, this result suggests that extending herd life to 4 or 5 lactations could generate additional income for dairy producers. However, this would not always be true. In their sensitivity analysis, different economic conditions, including lower feed and replacement costs, lower feed costs, or higher cull cow prices, moderated the economic advantage of increasing herd life from 2.6 to 4 lactations.

The effect of poor management on the relationship between profitability and herd life also was studied. Poor management was associated with increased culling for infertility and health cost, particularly for herds with a higher proportion of older cows, instead of culling on the expected contribution of production to profitability. This increase in culling on criteria unrelated to genetic merit for production decreased profit.

The authors recommended that, in dairy herds with an average cow herd life of approximately 3 lactations, cows

should be retained longer to increase annual net income. They cautioned that the economic advantage of extending herd life could be reduced by poor management, low feed cost or high price for cull cows.

Survival Potential and the Scope for Voluntary Culling

Economic studies have indicated that increasing average herd life beyond 3 lactations by management decision could increase annual net income. However, the comparison of income between a herd with genetically improved longevity and a herd of average genetic merit for longevity but increased herd life by management decision has not been done. For example, Congleton and King (1984) conducted sensitivity analyses of economic effects of increasing herd life under different feed costs, management systems and cull cow prices, but not involving herds with different genetic potential for longevity. This would be possible if herds with differnt age specific survivorship curves (Lx) were compared. Lx is defined as the probability, at birth or at the time of selection, of an animal surviving to age x, and is computed as (No. alive at age x)/(No. alive at age 0). Schons et al. (1985)

illustrated the use of Lx and other life history statistics for a commercial beef cattle population.

Variable Lx curves, indicating differences in inherent longevity, could be simulated and built into economic models. The assumption which must be made is that herds with genetically improved longevity would have a gently sloping Lx curve compared with herds of low or average longevity. This is illustrated by the model of throwing a stone from a cliff (Fig.3). A stone thrown with strong force in the beginning drops to the ground after following a mild trajectory. On the other hand, a stone thrown with weak force drops to the ground after following a steep trajectory. Force of the throw determines both the distance and path followed by the stone. Biological potential for longevity could be analogous to this example. A herd with superior biological potential at birth for long herd life would have greater average longevity than one with average potential for longevity (the difference in median longevity between two herds on the horizontal axis of the graph in figure 3.). Another difference (illustrated on the vertical axis of the graph) is that a herd with superior longevity would have less annual involuntary culling compared with a herd with average longevity. This illustrates that longevity of animals could be expressed not only by the length of herd

life but also by the amount of involuntary culling. When involuntary culling or natural selection is high (because of poor biological potential for life span), only a limited amount of voluntary culling or artificial selection can be practiced. What commercial producers want is good biological potential for a long life span so that they can impose the appropriate culling strategy and herd age structure that economic analysis suggests. In other words, biological potential for life span may be important, whether the producer allows an animal to live out its "biologically programmed" years or not.

Allaire and Cunningham (1980) studied economics of voluntary and involuntary culling. The greatest economic benefits from voluntary culling occurred when involuntary losses were minimal.

Van Arendork (1986) reported that the percentage of voluntary culling per year increased when involuntary replacement rates were reduced and that higher rates of voluntary replacement increased the financial advantage from lower involuntary replacement.

Genetic Variation in Longevity

If increased longevity is economically beneficial for commercial producers, then genetic improvement for the trait must be accomplished by seed stock breeders. The primary concerns would be the existence and exploitation of genetic variation for the trait. If there is no usable genetic variation for longevity, there is no way to improve the characteristic genetically.

Heritability estimates for longevity in dairy cattle might be criticized by some, since the traits generally assessed do not reflect the true biological potential of animals for life span. Cows are selected based on milk production and low producers with high biological potential for long life could be culled at an early stage. However, as stated earlier, longevity in dairy cattle is generally defined as the length of productive life in the herd, which is influenced by general health, fertility and production. The dairy producers' primary concern is sustainable profit. Therefore, heritability estimates of herd life defined in this fashion are still valid and useful to the industry.

Heritabilities have been estimated for age at last calving, number of lactations, length of life from first calving to disposal, age at disposal and stayability to a specific age. Estimated heritabilities are summarized in table 1. All studies involved Holstein cattle, except as

indicated in the table.

There are a few high heritability estimates of longevity traits. According to Gaalaas and Plowman (1963), when heritability of longevity is estimated from dam-daughter regression, there is likely to be some upward bias in the estimate. Since there is a positive correlation between milk yield of dam and daughter, and since the primary selection criterion in the dairy industry is milk production, the retention of higher producing cows would establish some automatic correlation for longevity between daughters and dams.

In Hudson and Van Vleck's study (1981), the stayability records were coded 1 if a cow survived to a certain age and 0 otherwise, and then subjected to paternal half-sib correlation analysis to estimate heritabilities of 36-, 48-, 60-, 72-, and 84-mo stayability. The authors suggested that increasing heritabilities with increasing age might have been caused by reductions in proportions of cows surviving to successive ages, because variance of a binomial trait increases with more equal proportions in each category.

Robertson and Barker (1966) also considered survival as an all-or-none trait. The heterogeneity X^2 between sires for survival of their offspring (Robertson and Lerner, 1949) was used to estimate the heritabilities of

survival to 2nd, 3rd, 4th, 5th and 6th lactations in Ayrshire cows and 2nd, 3rd, 4th and 5th lactation in Holstein-Friesians.

Estimates of heritability for longevity measured in the various ways have generally been low, ranging from 0.00 to 0.39 with a mean of 0.12. Because of the low apparent heritability of longevity, the effectiveness of direct selection for this characteristic is questionable unless a high selection differential and a short generation interval could be achieved.

If selection for longevity is based on the direct measurement of longevity in the animals which will be selected, a major potential problem is a decrease in annual genetic improvement of all traits under selection, due to the increase in generation interval. One way to overcome this problem is to select on early life traits which are genetically correlated to length of herd life.

Phenotypic and Genetic Covariation of Longevity with Early Production Traits

Today, the best newly proven AI sires frequently are selected on the basis of their daughters' first lactation

milk yields. However, a persistent idea among farmers is that high milk production in the first lactation may shorten the productive life of dairy cattle and result in poor lifetime performance.

In order to clarify this relationship, phenotypic and genetic correlations between first lactation and longevity traits have been estimated in many studies. From the phenotypic relationship between first lactation milk yield and longevity, the length of individual herd life could be estimated at an early stage of life, but with accuracy only if the correlation were fairly high. The genetic relationship, on the other hand, is important since it allows prediction of the change in length of herd life in response to selection for high first-lactation milk production.

Phenotypic correlations between first lactation milk yield and several longevity traits are summarized in table 1. Based on all these estimates, phenotypic correlations between first lactation milk yield and longevity traits ranged from 0.05 to 0.38 with a mean of 0.23. These positive phenotypic correlations suggest that first lactation milk yield is a moderate indicator of herd life and that cows with high first-lactation milk yield would tend to stay in a herd longer, contrary to the expectations of some dairy producers.

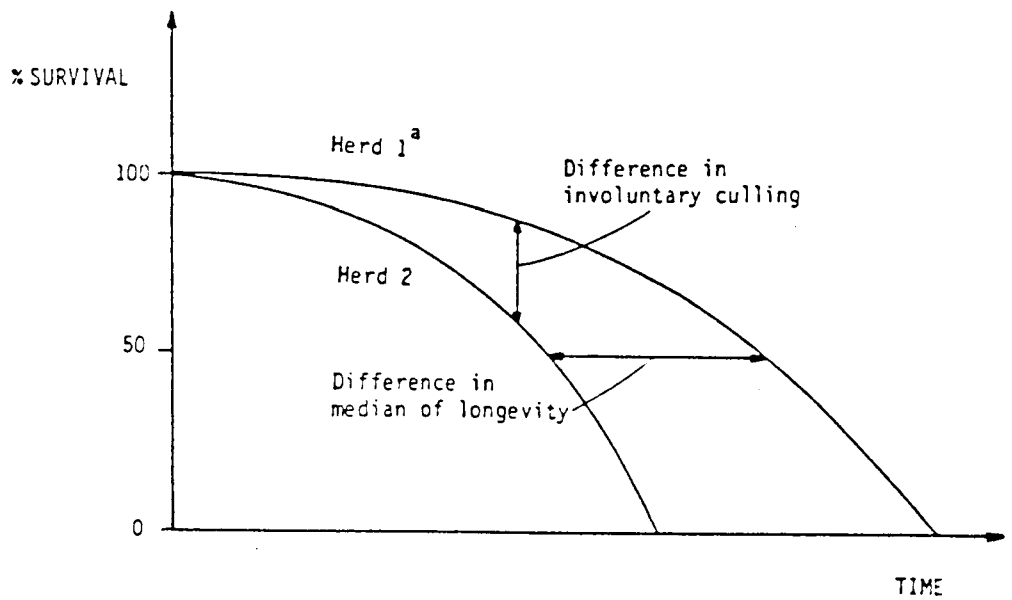
Genetic correlations also are summarized in table 1. Genetic correlations between first-lactation milk yield and longevity traits ranged from 0.27 to 0.82 with a mean of 0.59. These high positive genetic correlations suggest that selection on high first lactation milk production could increase herd life, but the magnitude of the correlated response will be limited because of the apparent low heritability of longevity itself. Both Miller et al.(1967) and Hoque and Hodges (1980) postulated that these relatively high genetic correlations arose from selection for milk production. If a cow has poor milk production, she is more likely to be culled. Therefore, her "longevity" is forced by the dairy producer to be low, even if she had the biological potential for long life.

Summary

Longevity is a characteristic of potential economic importance in dairy cattle. The possible advantages of increased herd life are reduced annual costs of raising or purchasing replacement heifers and an increase in lifetime production of cows. Most simulation studies have suggested that extending herd life would merit

consideration. Extending herd life from the U.S. average of 3 lactations to approximately 4-5 lactations was estimated to generate more income per year. The optimum length of herd life, however, is dependent upon management of the herd and economic conditions at the time. Longevity can influence not only the length of herd life but also the amount of involuntary culling. A herd with superior biological potential for long herd life would have less involuntary culling each year and greater economic benefit from voluntary culling than a herd with average potential for longevity. Estimates of heritability for longevity measured in various ways have generally been low, ranging from 0.00 to 0.39 with a mean of 0.12. Phenotypic correlations between first lactation milk yield and longevity traits ranged from 0.05 to 0.38, with a mean of 0.23. Genetic correlations between first lactation milk yield and longevity traits ranged from 0.27 to 0.82, with a mean of 0.59.

Figure 3. Hypothetical model for longevity differences between 2 herds.



[a] Herd 1 - herd with genetically improved longevity
Herd 2 - average herd

TABLE 1. ESTIMATES OF HERITABILITY(h^2) FOR LONGEVITY TRAITS, AND PHENOTYPIC(r_p) AND GENETIC(r_g)CORRELATIONS BETWEEN LONGEVITY TRAITS AND FIRST-LACTATION MILK YIELD IN DAIRY CATTLE

Longevity trait	h^2 [a]		r_g	r_p	Source
Age at last calving	0.00	R&C		0.19 0.18c	Parker et al. (1960)
	0.014 to 0.09	U			Plowman & Gaalaas (1960)
	0.14	C		0.25	White & Nichols (1965)
Number of lactations	0.13	C			White & Nichols (1965)
	0.05 to 0.14	C	0.54 to 0.77	0.19 to 0.25	Miller et al. (1967)
	0.14	R	0.62	0.38	Hargrove et al. (1969)
	0.09	C	0.41	0.26	Hoque & Hodges (1980)
Length of life	0.39 0.00	C R		0.22	Evans et al. (1964)
	0.15	C	0.76		Hargrove et al. (1969)
	0.25	C	0.81	0.13	Gill & Allaire (1976b)
	0.10	C	0.44	0.28	Hoque & Hodges (1980)
Age at disposal	0.26	C			Gill & Allaire (1976b)
	0.10	C	0.44	0.28	Hoque & Hodges (1980)

TABLE 1. CONTINUED

Longevity trait	h^2		r_g	r_p	Source
Successful parturitions	0.37	R			Wilcox et al. (1957)
	0.23	C	0.82	0.05	Gill & Allaire (1976b)
Stayability	0.07(2nd)b	X^2	0.85		Robertson & Barker (1966)
	0.13(3rd)		0.89		
	0.13(4th)		0.92		
	0.15(5th)		0.90		
	0.20(6th)		0.86		
	0.04(2nd)	X^2	0.67		
	0.05(3rd)		0.75		
	0.06(4th)		0.76		
	0.03(5th)		0.71		
			0.27(36mo)	0.18	Everett et al.(1976)
			0.41(48mo)	0.25	
			0.55(60mo)	0.36	
			0.51(72mo)	0.32	
			0.51(84mo)	0.31	
	0.02(36mo)	C	0.56	0.22	Hudson & Van Vleck (1981)
	0.04(48mo)		0.64	0.27	
	0.05(60mo)		0.65	0.25	
	0.05(72mo)		0.58	0.22	
	0.05(84mo)		0.47	0.17	

a R - h^2 estimated by daughter-dam regression
C - h^2 estimated by half-sib correlation
X^2 - h^2 estimated by X^2
U - Unknown

b Ayrshire

c Jersey

References

Allaire, F. R. and E. P. Cunningham. 1980. Culling on low milk yield and its economic consequences for the dairy herd. Livestock. Prod. Sci. 7:349.

Batra, T. R., E. B. Burnside and M. G. Freeman. 1971. II. Effect of herd size and production level on dairy cow disposal patterns. Can. J. Anim. Sci. 51:85.

Burnside, E. B., A. E. McClintock and K. Hammond. 1984. Type, production and longevity in dairy cattle: A review. Anim. Breed. Abstr. 52:711.

Burnside, E. B. and J. W. Wilton. 1970. Anatomical traits as they relate to productive utility. J. Dairy Sci. 53:837.

Congleton, W. R., Jr. 1984. Dynamic model for combined simulation of dairy management strategies. J. Dairy Sci. 67:644.

Congleton, W. R., Jr. and L. W. King. 1984. Profitability of dairy cow herd life. J. Dairy Sci. 67:661.

Evans, D. L., C. Brantron and B. R. Farthing. 1964. Heritability estimates and interrelationships among production per day of productive life, longevity, breeding efficiency, and type in a herd of Holstein cows. J. Dairy Sci. (Suppl. 1):699 (Abstr.).

Everett, R. W., J. F. Keown and E. E. Clapp. 1976. Relationships among type, production, and stayability in Holstein cattle. J. Dairy Sci. 59:1505.

Gaalaas, R. F. and R. D. Plowman. 1963. Relationship between longevity and production in Holstein-Friesian cattle. J. Dairy Sci. 46:27.

Gill, G. S. and F. R. Allaire. 1976a. Relationship of age at first calving, days open, days dry, and herdlife to a profit function for dairy cattle. J. Dairy Sci. 59:1131.

Gill, G. S. and F. R. Allaire. 1976b. Genetic and

selection method for optimizing profit in dairy cattle. J. Dairy Sci. 59:1325.

Hargrove, G. L., J. J. Salazar, and J. E. Legates. 1969. Relationships among first-lactation and lifetime measurements in a dairy population. J. Dairy Sci. 52:651.

Hoque, M. and J. Hodges. 1980. Genetic and phenotypic parameters of lifetime production traits in Holstein cows. J. Dairy Sci. 63: 1900.

Hudson, G. F. S. and L. D. Van Vleck. 1981. Relationship between production and stayability in Holstein cattle. J. Dairy. Sci. 64:2246.

Miller, P., L. D. Van Vleck and C. R. Henderson. 1967. Relationship among herd life, milk production and calving interval. J. Dairy Sci. 50:1283.

Parker, J. B., N. D. Bayley, M. H. Fohrman and R. D. Plowman. 1960. Factors influencing dairy cattle longevity. J. Dairy. Sci. 43:401.

Pearson, R. E. and A. E. Freeman. 1973. Effect of female culling and age distribution of the dairy herd on profitability. J. Dairy. Sci. 56:1459.

Plowman, R. D. and R. F. Gaalaas. 1960. Heritability estimates of longevity in Holstein-Friesian cattle. J. Dairy. Sci. 43:877(Abstract P-64).

Rendel, J. M. and A. Robertson. 1950. Some aspects of longevity in dairy cows. J. Exp. Agric. 18:49.

Renkema, J. A. and J. Stelwagen. 1979. Economic evaluation of replacement rates in dairy herds. 1. Reduction of replacement rates through improved health. Livestock Prod. Sci. 6:15.

Robertson, A. and J. S. F. Barker. 1966. The correlation between first lactation milk production and longevity in dairy cattle. Anim. Prod. 8:241.

Robertson, A. and I. M. Lerner. 1949. The heritability of all-or-none traits: viability of poultry. Genetics 34:395.

Schmedt, G. H. and L. D. Van Vleck. 1974. Principles of Dairy Science. W. H. Freeman, San Francisco.

Schons, D., W. D. Hohenboken and J. D. Hall. 1985. Population analysis of a commercial beef cattle herd. J. Anim. Sci. 61:44.

Smith, S. P. and R. L. Quaas. 1984. Productive lifespan of bull progeny groups: Failure time analysis. J. Dairy Sci. 67:2999.

Van Arendork, J. A. M. 1986. Economic importance and possibilities for improvement of dairy cow herd life. Third World Congress on Genetics Applied to Livestock Production 9:95.

Van Doormaal, B. J., L. R. Schaeffer and B. W. Kennedy. 1985. Estimation of genetic parameters for stayability in Canadian Holsteins. J. Dairy Sci. 68:1763.

White, J. M. and J. R. Nichols. 1965. Relationships between first lactation, later performance, and length of herd life in Holstein-Friesian cattle. J. Dairy Sci. 48:468.

Wilcox, C. J., K. O. Peau and J. W. Bartlett. 1957. An investigation of the inheritance of female reproductive performance and longevity, and their interrelationships within a Holstein-Friesian herd. J. Dairy Sci. 40:942.

Longevity Studies in Beef Cattle and Sheep

Introduction

Longevity in beef cattle and sheep is likely to be as important as in dairy cattle, since the potential advantages of increased longevity are common in these livestock industries. Those advantages are the reduction of annual replacement costs and an increase in the duration of productive life. Compared with the dairy industry, both beef and sheep populations more often lack complete production records and pedigree information to make longevity studies possible. The main objective of the present review is to summarize available information on longevity in both species. Most longevity studies in beef cattle focus on the optimum age for cow culling and on replacement decisions from an economic point of view. Although genetic parameters for certain longevity traits have been reported in sheep, no heritability estimations for longevity traits were available in beef cattle.

Definition of Longevity

The term longevity as used in the production of beef cattle often refers to reproductive life span, or the length of time a cow or bull remains productive (O'Mary and Dyer, 1978). The same definition of longevity could be applied to sheep. Longevity in these species means ability of animals to have a long and stable productive herd life as well as the biological capacity for extended survival. Longevity is a function both of death losses and culling decisions based upon actual or anticipated substandard production (Bradford and Meyer, 1986).

Optimum Age of Culling in Beef Cattle

Rogers (1972) studied the economics of replacement rates in commercial beef herds. Four variables influenced by age of the cow were included in the study. These were percent calf crop weaned, weaning weight of calves, sale value of cows and death losses of cows. He concluded that the optimal economic life of beef cows was around 9 or 10 years and that retention of cows beyond 10 years of age

was likely to result in reduced long term profits.

Congleton and Goodwill (1980a) simulated breeding plans using Hereford, Angus and Charolais sires mated to Hereford, Angus and Hereford-Angus dams. The culling policy in the simulation study (light culling) was to cull a cow only if she failed to produce a calf for two consecutive years. They realized that productivity of a herd was influenced by the age structure of the herd, since cow fertility, cow and calf mortality and preweaning growth of the calf were influenced by the age of the cow, as well as by the genotypes of the cow and calf. In a subsequent simulation study (Congleton and Goodwill, 1980b), they examined the effect of heavy culling on herd productivity. The culling policy in that simulation was to cull a cow as soon as it became apparent that she had failed to produce a calf. The breeds of sire used in the simulation were Angus, Hereford, Charolais, Jersey, South Devon, Limousin and Simmental. They were mated to Angus dams by artificial insemination. Results suggested that the culling option (heavy culling vs. light culling) had diverse effects on the structure and productivity of the herd. The heavy culling option increased both the replacement rate and the proportion of the herd three years of age or less. With heavy culling, all crosses had more young cows in the herd, particularly those involving

large breeds of sire. Since calf mortality from young cows was higher than from older cows, and since young cows weaned lighter calves and had decreased fertility owing to calving difficulty, heavy culling decreased productivity per cow bred. Light culling for productivity in the cow herd resulted in higher gross income than did heavy culling, but these results could be dependent upon whether replacement females were raised or purchased.

Clarke et al. (1982) simulated a typical western range cow-calf operation. The cow herd, including heifers, was held at 200 head during the breeding season each year. Cows were culled from the simulated herd twice during the year, at the end of the calving season and when calves were weaned. The priority order of culling in the fall was: (1) cows over 15 yr, (2) unsound cows, (3) cows that failed to wean a calf (4) nonpregnant cows. Annual death loss of cows was assumed to average 3 percent and to increase with cow age. They indicated that several of the effects of variation in the culling criteria on net income were mediated through the annual replacement rate. Culling criteria that increased the replacement rate, also increased the proportion of young cows in the herd. This resulted in lighter average weaning weights because of lower production from immature cows compared with mature cows. At the same time, the rapid replacement rate

required that more heifer calves were kept for replacement and fewer heifer calves were therefore available for sale. That raised the average calf sale weight since calves that were sold were predominantly male. Total calf weight sold changed very little because the two effects cancelled one another.

Culling criteria also affected the number of calves sold through the replacement rate. As the replacement rate increased, fewer calves were sold due to the necessity of keeping a greater proportion of the calves as replacements. One other reason that the number of calves sold decreased as replacement rate increased was that the increase in the proportion of immature cows resulted in fewer calves produced.

Another effect of increasing the replacement rate was the decreased annual death loss of cows, because death loss was weighted according to age class. Since the death loss was lower for younger ages, the weighted average death loss decreased as a greater proportion of the cow herd consisted of immature cows.

Because only a single year was modeled in their study, it was not possible to incorporate the genetic gains resulting from selection and to evaluate the effect of replacement rate on the genetic gain.

Clarke et al. (1984a) subsequently created a model of

a 500-head, spring calving cow-calf enterprise in which the number of cows in the calving herd was held constant. One of their objectives was to determine the optimum cow replacement ages for different mating plans.

Their study showed that the optimum replacement age was dependent upon the mating plan in use. The cow replacement ages that maximized gross margin per cow were 5,9 and 12 yr for a three-breed rotation, for the two breeds in the rotational part of a criss-out-cross program and a three-breed terminal cross, respectively.

The cow replacement age that maximized gross income per cow also varied with reproductive culling strategy. A cow replacement age of 9 yr was optimal when culling was based only on unsound cows and those that had reached the maximum allowable age, or any cow that failed to wean a calf. The optimum cow replacement age was 5 yr when culling was practiced on nonpregnant cows in the fall, culling all cows without a live calf at the end of the calving season, or culling nonpregnant cows in the fall and then culling any cow that subsequently lost her calf in the spring. Since genetic trends were ignored in that study, optimum replacement ages could have been different had the trends been included.

Kay and Rister (1977) studied cow replacement strategy, incorporating the effect of income tax on the

optimal replacement age. It was shown that a 9 or 10 calving-year replacement policy was the best under marginal tax rates below 48 percent. At the 70 percent tax rate, however, the indicated replacement age was uniformly at three calving years or before the cow had reached her full physical production capability.

Melton (1980) evaluated the economics of beef cow culling and replacement decisions when genetic progress was assumed to exist. He suggested that annual decisions of which animals to cull and replace affected not only current revenues, but, by altering the genetic composition of the herd, also affected future profitability. He gave the optimal culling age of 11 yr when no genetic progress was occurring. When genetic progress was occurring, with the selection of 2 percent of male calves annually, there was a reduction to 8 yr in the optimal culling age of the average cow.

Clarke et al (1984b) simulated a closed beef herd of 500 cows and heifers over 40 yr. The major differences between this study and their previous studies (Clarke et al.,1982; Clarke et al., 1984a) were that this simulation covered an extended time period, selection was practiced to increase weaning weight while holding birth weight constant and the population was composed entirely of straightbred cattle. They indicated that both genetic

progress and economic efficiency were affected by cow replacement age, fraction of the herd culled for poor production and culling for reproductive failure. Decreasing cow replacement age decreased the generation interval, thereby increasing genetic gain per year. Increasing the level of culling for poor production also decreased the generation interval (although to a lesser degree) and increased the genetic value of the females producing the replacement males and females.

However both strategies, by increasing the replacement rate, increased the number of replacement females needed, and thereby decreased selection intensity placed on replacement females. Apparently this resulted in the leveling off or decline in genetic gain both at the highest percentage of culling and at the youngest age of replacement.

Genetic and Phenotypic Parameters of Longevity

Although many heritability estimates of longevity and of genetic and phenotypic correlations between longevity traits and other traits have been reported in dairy cattle, few estimates of genetic and phenotypic parameters

of such performance traits have been reported in beef cattle or sheep.

Lifetime number of lambs born is indicative of longevity within contemporary breed groups of sheep. Several studies have suggested the existence of genetic variation. Tanida and Hohenboken (unpublished results,1986) indicated that regression coefficients of descendent on ancestor performance for lifetime number of lambs born were not significantly different from zero but predominantly were positive, which suggested that ancestor prolificacy and (or) productive life span were predictive of those traits in their descendants. This result is supported by studies in which heritability estimates for lifetime number of lambs born per crossbred ewe lambing were 0.14 (Thapan et al. 1970) and 0.12 (Clarke and Hohenboken, 1983), and per straightbred ewe lambing were 0.18 to 0.26 (Shelton and Menzies, 1968) and -0.12 to 0.19 (Basuthakur et al., 1973).

Direct selection for longevity would lead to increased generation intervals. Some early life traits could be indicative of longevity for individual animals. One way to select for longevity without increasing generation intervals would be to select animals at a young age, based upon their phenotypic merit for the indicator traits.

Phenotypic correlations between early life traits of a ewe and her lifetime production also have been estimated. Lifetime number of lambs born was significantly correlated with yearling and weaning weights of the ewe (0.09 and 0.10, respectively, by Basuthakur et al., 1973; 0.13 and 0.10, respectively, by Shelton and Menzies, 1968). Genetic correlations also were estimated between these traits by Shelton and Menzies (1968). The genetic correlations between weaning weight of the ewe and lifetime number of lambs born and raised were both negative and of low magnitude (-0.03 and -0.11, respectively). By contrast, there were positive genetic relationships between yearling weight and both lifetime production traits (0.18 and 0.11, respectively). The authors suggested that rapid early development was not necessarily desirable in a range ewe, but adequate size as yearlings was important.

Saoud and Hohenboken (1984a) reported that some components of lifetime production efficiency (computed as estimated gross income minus feed and ewe purchase cost) could be predicted by early life traits such as ewe's type of birth, actual and adjusted weaning weight, postweaning weight and first year gross income.

In a subsequent study of Saoud and Hohenboken (1984b), ewes which survived for the entire duration of

their experiment did not differ significantly from their entire group for early life traits. Identifying potential indicator traits for longevity therefore was not successful.

When longevity was measured as a ewe's age in months when she died or was culled from the experiment or, for surviving ewes, her age at termination of the experiment, Hohenboken and Clarke (1981) reported that Cheviot crossbreds had shorter lifespans than Finnsheep, Dorset or Romney crossbred ewes on irrigated pastures. On hill pastures, the ranking of ewe sire-breed groups for longevity was Dorset, Cheviot, Romney and Finnsheep. The existence of genotype x environment interactions of this sort should be granted more attention as exotic breeds and new genotypes are introduced widely throughout the U.S. The authors further suggested that at least part of breed differences in cumulative lamb production were dependent upon breed differences in longevity, a trait that has received little emphasis in breed evaluation experiments. Vesely and Peters (1974) reported that longevity of ewes was highest for the Romnelet and Columbia breeds and lowest for the North Country Cheviot.

Boylan (1985) studied breed effects and heterosis for longevity in a population involving Finnsheep, Suffolk and Targee breeds and their crosses. A practical

sheep growers' concern pointed out by him is that highly prolific breeds such as the Finnsheep may have a shorter productive life than less prolific breed groups. In fact, Finnsheep had the shortest survival age in the flock (56 months). Survival ages for Suffolks and Targhees were similar at 63 months. The survival ages exhibited substantial heterosis, which was greater in Suffolk crosses than in Targhee crosses (18.5 vs 8.2 %).

Even though the number of reports on inheritance of longevity is limited, existence of genetic variation in longevity traits and the possibility of genetic improvement in this characteristic is suggested, at least in sheep. In beef cattle, a few reports suggest the existence of heterosis and breed effects on longevity, but heritability estimates for the trait have not been reported.

Nunez et al. (1984) studied heterosis for longevity in beef cattle. Heterosis for longevity averaged 1.36 $\pm$ .58 yr or 16.2 $\pm$ 6.9 % and was greater for reciprocal crosses of Hereford - Angus (21.1 %) than for Angus - Shorthorn (14.8 %) or Hereford - Shorthorn (12.8 %).

Stewart and Martin (1981) studied long-term productivity in purebred and crossbred cows of Angus and Milking Shorthorn parentage. Contrasts among the breed types indicated a breed effect for maternal characters.

Angus cows were in the herd longer and produced more calves and, therefore, more total calf weight at weaning than Shorthorn cows, whose calves had higher average birth and weaning weights. Heterotic effects were also observed. Crossbred cows were in the herd longer, produced more calves of higher average weight and more calf weight per year in the herd than their straightbred counterparts. Consequently, crossbred cows produced 230 kg (22%) more total weight weaned than the mean of the straightbreds. They indicated that this 22% increase in production was the cumulative result of greater longevity resulting from higher fertility of the crossbred cows and higher average weaning weights.

Improvement of Longevity through General Health

There are traits which are directly related to survival potential or longevity of animals. Examples are specific resistance and tolerance to certain diseases (e.g. cancer eye, facial eczema and internal parasites), immunological mechanisms for survival, and physical and physiological characteristics which enhance survival(e.g. teeth wear rate).

Although it is beyond the scope of this review to cover traits related to general health and survivability in beef cattle and sheep, we should be aware of the importance of such traits to the improvement of longevity.

Summary

The subject of longevity in beef cattle and sheep is as important as in the dairy industry. However, both beef and sheep populations often lack complete production records and pedigree information, and not many longevity studies have been reported in these species. Although genetic parameters for certain longevity traits have been reported in sheep, no estimations of genetic parameters related to longevity were available in beef cattle. Most existing longevity studies in beef cattle focus on the optimum age of cow culling and on replacement decisions from an economic point of view.

References

Basuthakar, A. K., P. J. Burfening, J. L. Van Horn and R. L. Blackwell. 1973. A study of some aspects of lifetime production in Targhee and Columbia sheep. J. Anim. Sci. 36:813.

Boylan, W. J. 1985. Crossbreeding for fecundity, in : Genetics of Reproduction in Sheep, edited by Land R. B. and D. W. Robinson. Butterworths, London.

Bradford. G. E. and H. H. Meyer. 1986. Economic evaluation of breeding objectives for sheep and goats : Practical considerations and examples. Third World Congress on Genetics Applied to Livestock Production. 9:479.

Clarke, S. E., C. T. Gaskins and J. K. Hillers. 1982. Systems analysis of beef production - effects of culling criteria on net income. J. Anim. Sci. 55:1982.

Clarke, S. E., C. T. Gaskins, J. K. Hillers and W. D. Hohenboken. 1984a. Mathematical modeling of afternative culling and crossbreeding strategies in beef production. J. Anim. Sci. 58:6.

Clarke, S. E., C. T. Gaskins, J. K. Hillers and W. D. Hohenboken. 1984b. Mathematical modeling of alternative selection strategies for beef production. J. Anim. Sci. 59:308.

Clarke, S. E. and W. D. Hohenboken. 1983. Estimation of repeatability, heritability and breed differences for lamb production. J. Anim. Sci. 56:309.

Congleton, W. R. Jr. and R. E. Goodwill. 1980a. Simulated comparisons of breeding plans for beef production - part 2 - Hereford, Angus and Charolais sires bred to Hereford, Angus and Hereford-Angus dams to produce feeder calves. Ag. Systems. 5:221.

Congleton, W. R. Jr. and R. E. Goodwill. 1980b. Simulated comparisons of breeding plans for beef production : part 3 - systems for producing feeder calves involving intensive culling and additional breeds of sire. Ag. Systems. 5:309.

Hohenboken, W. D. and S. E. Clarke. 1981. Genetic, environmental and interaction effects on lamb survival, cumulative lamb production and longevity of crossbred ewes. J. Anim. Sci. 53:966.

Kay, R. D. and E. Rister. 1977. Income tax effects on beef cow replacement strategy. South. J. Agr. Econ. 9:169.

Melton, B. E. 1980. Economics of beef cow culling and replacement decisions under genetic progress. West J. Agr. Econ. 5:137.

Nunez, R. D., L. V. Cundiff, G. E. Dickerson and K. E. Gregory. 1984. Heterosis of longevity in beef cattle. J. Anim. Sci. 59:Suppl. 1:180.

O'Mary, C. C. and I. A. Dyer. 1978. Commercial Beef Cattle Production. 2nd edition. Lea & Febiger, Philadelphia.

Rogers, L. F. 1972. Economics of replacement rates in commercial beef herds. J. Anim. Sci. 34:921.

Saoud, N. B. and W. D. Hohenboken. 1984a. Phenotypic relationships among early life traits and lifetime ewe production efficiency. J. Anim. Sci. 59:607.

Saoud, N. B. and W. D. Hohenboken. 1984b. The effect of selection in retrospect on lifetime production efficiency in sheep. J. Anim. Sci. 59:620.

Shelton, M. and J. W. Menzies. 1968. Genetic parameters of some performance characteristics of range fine-wool ewes. J. Anim. Sci. 27:1219.

Stewart, T. S. and T. G. Martin. 1981. Mature weight, maturation rate, maternal performance and their interrelationships in purebred and crossbred cows of Angus and Milking Shorthorn parentage. J. Anim. Sci. 52:51.

Thapan, P. C., D. S. Bell and C. F. Parker. 1970. Reproductive performance in Targhee sheep. Indian J. Anim. Sci. 40:223. (Anim. Breed Abstr. 39:528).

Vesely, J. A. and H. F. Peters. 1974. Lamb production from ewes of four breeds and their two breed and three breed crosses. Can. J. Anim. Sci. 54:543.

CHAPTER 3

THE INHERITANCE OF LONGEVITY TRAITS IN BEEF CATTLE

Introduction

Longevity of beef cows can be defined as reproductive lifespan or length of time that a cow remains productive (O'Mary and Dyer, 1978). It can be expressed in several ways. Examples are age at last calving, lifetime number of calves weaned, length of life from first calving to disposal and age at disposal. Since each measurement involves some loss of infomation, the use of more than one measurement of longevity generally is ideal.

Compared with the dairy industry, beef herds often lack complete production records and pedigree information to make longevity studies possible, and estimates of genetic parameters for longevity traits in beef cattle have not been reported. According to dairy studies, genetic variation for longevity exists but heritability generally is low, in the proximity of 0.1 to 0.2. In beef

cattle, the effect of heterosis on longevity has been reported (Stewart and Martin, 1981; Nunez et al., 1984), but most longevity studies have focused on the optimum age for culling of cows and on replacement strategies from an economic point of view.

Longterm data sets were available from purebred Angus and Hereford herds to study the inheritance of longevity. The objectives of this experiment were 1) to characterize the populations for certain traits indicative of longevity or herd life, 2) to estimate heritabilities of the traits, 3) to estimate genetic and phenotypic correlations among the longevity traits and between longevity and traits measured early in a cow's life and 4) to compare survival patterns of paternal half-sib groups of cows by graphical examination of life history statistics (Schons et al., 1985).

Justification

A well known principle from quantitative genetic theory is that the response to selection per unit time is directly related to selection intensity and accuracy of selection but inversely related to the generation

interval. This suggests that a rapid turnover of cows should increase selection response per unit of time and that there is little justification to select cattle for greater longevity. Cost of cow ownership, on the other hand, (replacement female cost minus salvage value divided by years in the herd) generally would be expected to decrease with increasing average longevity. Cow age, and consequently herd age structure, is well known to influence weaning weight. From an economic standpoint, there will be some optimum age for cow culling that balances these and other effects. From results of simulation studies, a maximum herdlife of 9 to 10 years (Rogers, 1972; Kay and Rister, 1977) was reported to be optimum. Melton (1980) reported optimal culling ages of 8 and 11 years, respectively, in herds in which there was or was not genetic progress taking place.

Variation among herds in longevity could be illustrated by the analogy of throwing a stone from a cliff. A stone thrown with strong force drops to the ground after following a mild trajectory for an extended distance. A stone thrown with weak force drops to the ground closer to the base of the cliff, after following a steep trajectory. Force of the throw determines both the distance and path followed by the stone.

Cows in a herd with superior potential for herd life

would have greater average longevity than those in a herd with poorer longevity potential. Fewer cows would be lost each year for involuntary reasons (eg. death and debilitating illness), so fewer replacements would be needed and(or) there would be greater scope for voluntary culling on production. Commercial cattle producers would benefit from cows with good biological potential for a long life span so that they could impose the appropriate culling strategy and herd age structure that economic analysis suggests. In other words, biological potential for life span may be important, whether the producer allows an animal to live out its "biologically programmed" years or not.

Allaire and Cunningham (1980) studied economics of voluntary and involuntary culling in dairy cattle. The greatest economic benefits from voluntary culling occurred when involuntary losses were minimal. Van Arendork (1986), also work in dairy cattle, reported that the percentage of voluntary culling per year could be increased when involuntary replacement rates were reduced and that the higher rates of voluntary replacement possible because of lower involuntary replacement were a financial advantage.

Materials and Methods

Populations. Data for the genetic study of longevity must include cows' complete individual life histories, production records and pedigree information, ideally spanning many generations. Records from the San Carlos Apache Indian tribe Hereford herd in Arizona and the One-Bar-Eleven Angus Ranch in Wyoming met these requirements.

The University of Arizona and the U. S. Department of Agriculture cooperate with the San Carlos Apache tribe in management of the Hereford herd and use it as a research resource. Young bulls to supply the tribal commercial Hereford herds in east-central Arizona are produced within the herd. The Angus ranch represents a typical progressive Intermountain Western cow/calf operation, has been subjected to consistent management and has been used for population analysis by Schons et al. (1985). Thirty years (1957 through 1986) and 23 years (1959 through 1981) of production and identification records were available from the Hereford and Angus herds, respectively.

The Hereford herd averaged 325 $\pm$ 20 cows and 98 $\pm$ 6 replacement heifers, while the Angus herd averaged 672 $\pm$ 21 cows and 110 $\pm$ 9 replacement heifers per year. In both herds, all replacement heifers were selected from within

the herd. The Angus herd utilized artificial insemination and used fewer sires per 100 cows per year (producing more daughters per sire) than in the Hereford herd.

In the Hereford herd, replacement bulls and heifers were selected for appearance using standard characteristics of the breed. There was no culling until animals were over 2 years of age. First calving was usually at 3 years of age, and cows generally were culled at 10 years of age. The major culling criteria were physical unsoundness (such as problems relating to locomotion and inability to consume range forage), cancer eye, and udder unsoundness affecting more than one quarter. For more information on this herd, see DeNise and Ray (1987).

In the Angus herd, selection criteria for heifers were weaning and yearling weights, dam's record and, in some cases, information on their sire. First calving was usually at 2 years of age. Culling criteria for cows were nonpregnancy, poor production records, calving difficulty and poor mothering ability. The herd experienced reduction sales in 1967 and 1974. For more information on this herd, see Hellbusch (1982) and Schons et al. (1985).

Statistical Analysis. Original data sets had to be processed and reorganized to make longevity studies possible. Three steps were involved: 1) Longevity and number of calves weaned by each cow were summarized and

that information was appended to each of her annual records. 2) Each cow's early-life data (e.g., weaning weight) were appended to each of her annual records. 3) Longevity and number of calves weaned by each replacement daughter of a cow were appended to the dam's record for the year of that daughter's birth.

In this study, only "complete cohort groups" were utilized. A cohort is all replacement heifers born in a particular year (e.g., the 1959 cohort). A complete cohort is a birth year group from which all cows had left the herd, by 1986 and 1981 for the Hereford (1957 - 1971) and the Angus herds (1959 - 1966), respectively. Including records from incomplete cohorts in the analyses would have biased the results, as will be discussed.

Descriptive statistics (mean, SE, median, range and coefficient of variation) were calculated for three longevity traits of cows. These were length of life from first calving to disposal (FST), age at disposal (AGE), and lifetime number of calves weaned per cow (NUM). One Lx and one Px curve for each herd, pooled across sires and birth years, were computed and plotted, in order to compare attrition patterns between herds. Lx (age-specific survivorship) is defined as the probability, at birth, of an animal surviving to age x and is computed as (No. alive at age x) / (No. alive at age 0). Px (age-specific

survival rate) is defined as the probability, assuming survival to age x, of survival to age x + 1 and is computed as (Lx+1/Lx).

In a preliminary least-squares analysis of data from each herd, the effects of birth year, age of a cow's dam and the cow's own weaning age as a calf were tested as significant fixed sources of variation in the longevity traits. Based upon results of the preliminary analyses, longevity traits were adjusted for birth year in the Hereford herd, but for both birth year and age of a cow's dam in the Angus herd.

The heritabilities of longevity traits were estimated from daughter-dam regression and paternal half-sib analysis. The number of daughters per dam was variable, and consideration of this fact was taken when daughter-dam regressions were computed. Two practices have been widely used (Kempthorne and Tandon, 1953). One is to repeat the parent's record with each offspring's record (method 1). The other is to average all the offspring of a parent and regress each such average on the appropriate parent's record (method 2). Kempthorne and Tandon (1953) proposed a third method (method 3 - the weighted regression technique) in which weights are assigned to progeny means, the weights being functions of the number of progeny per parent and an estimated value of the correlation

coefficient between deviations from regression associated with two progeny of the same parent. Kempthorne and Tandon (1953) suggested that method 1 would be most valid if the correlation among the offspring of a parent were zero, while method 2 would be most valid if the correlation among members of each progeny group were unity. Bohren et al. (1961) reported that there was little difference in the efficiencies of methods 1 and 3 but that method 2 produced regressions with the largest standard errors. The correlation among sibs for longevity traits in the current study was expected to be low rather than high. This expectation, as well as the simplicity of method 1 resulted in our choosing it over methods 2 or 3.

Once daughter-dam regressions were calculated, they were multiplied by 2 (the reciprocal of the additive genetic relationship between dam and daughter) to obtain heritability estimates.

The paternal half-sib relationship was also used to estimate heritabilities of longevity traits. In the Angus herd, birth year and sire effects largely were confounded since a single artificial insemination sire was used in certain years. The herd used clean-up bulls in multiple sire mating pastures. Some cows, therefore, did not have sire identification and were not used in the paternal half-sib analyses. In the Hereford herd, the model

included birth year and sires nested within birth years. Observed variance was partitioned into the between-sire and within sire components. Computed intra-class correlation coefficients were multiplied by 4 (reciprocal of the additive genetic relationship between half-sibs) to obtain heritability estimates.

Genetic correlations were computed between early life traits and longevity of cows, and also among longevity traits themselves, from daughter-dam regression and paternal half-sib analysis. From daughter-dam regression, genetic correlations (Hazel,1943) and their standard errors (Reeve,1955) were estimated from covariances between different traits measured in daughters and dams. In paternal half-sib analyses, the same model and procedures were used as for heritability estimations, but the analysis of covariance was conducted, that is, a comparison of observed covariance of relatives for the two traits. Based upon results of preliminary analyses of early life traits in the Hereford herd, birth weight and weaning condition score were adjusted for birth year and age of dam, and weaning weight was adjusted for birth year, age of dam and weaning age. Since a substantial number of animals lacked records for early life traits, the number of records was reduced for the analyses.

The relationship between a cow's own weaning weight

and her subsequent longevity was further examined by redefining (as a statistical convenience) longevity as an independent variable, along with weaning age and age of dam, in a model for which the cow's weaning weight was the dependent variable. The least-squares means for weaning weight for each age class (excluding age classes with a paucity of data) were plotted to examine whether the relationship were linear, curvilinear or nonexistent. Since sire was not included in the model, these relationships are strictly phenotypic.

To characterize differences among sires in patterns of attrition of their progeny, Lx and Px statistics as described by Schons et al. (1985) were calculated for several sires (and for progeny of clean-up bulls in some years). There were insufficient replacement daughters per sire in the Hereford herd for meaningful comparisons among the Lx and Px statistics, so these computations were done only for Angus sires with at least 20 replacement daughters.

Once Lx and Px for each age were calculated, Lx and Px curves were plotted for each sire. Heritability estimates of Lx and Px at each age were possible by the Chi square method proposed by Robertson and Lerner (1949) and described in Turner and Young (1969). In this method, Lx and Px proportions for the different sires are assessed

to determine whether they vary about overall or pooled herd Lx and Px values more than could be attributed to chance; and total variation is partitioned among and within paternal half-sib groups. (Survival of a sire's daughters to age x is summarized in Lx and Px statistics.)

Results and Discussion

Descriptive Statistics. Means, standard errors, medians, maximum and minimum values, and coefficients of variation of longevity traits in the Hereford and Angus herds are presented in table 2. These statistics were computed only from complete cohorts. The approximately one year difference in AGE is due primarily to the fact that the Angus herd initiated first calving at 2 years of age whereas 3 years of age at first calving was common in the Hereford herd. The Hereford herd had lower coefficients of variation than the Angus herd, primarily because cows generally were culled at 10 years of age in the former herd.

Lx and Px curves for the Hereford and Angus herds, based upon data pooled across all complete cohorts, are shown in figure 4. The Lx curve for the Angus herd assumed

a boomerang shape (steep decline followed by mild decline), whereas Lx for the Hereford herd was linear between 3 and 11 years of age. The difference in Px curves between herds reflects the herd difference in Lx. For the Herefords, Px dropped quickly to zero after 10 years of age, whereas for Angus, it remained high through 15 years. Differences between herds do not suggest a genetic difference, but they demonstrate that these life history statistics can easily illustrate attrition patterns of the herds.

Effects of Birth Year, Age of Dam and Weaning Age on Longevity. The effects of birth year, age of a cow's dam and weaning age on longevity traits are presented in table 3. Birth year was a significant source of variation for longevity traits in both herds, but there was no systematic increase or decrease in longevity associated with advancing birth years. Age of dam affected longevity traits only in the Angus herd. When the dam of a cow was 4 years of age that cow tended to remain in the herd longer (an average of 7.6 years for AGE) than was true of progeny of younger or older dams. Ages of dam from 5 to 7 years were associated with shortest herd life, with averages for AGE of 6.0 to 6.2 years. Weaning age of a calf was not a significant source of variation in either herd.

Heritabilities and Genetic Correlations. Heritabilities

of longevity traits and genetic correlations among them estimated from daughter-dam regression and paternal half-sib correlation for the Hereford and Angus herd are presented in tables 4 and 5, respectively. Heritability estimates from paternal half-sib analysis in the Angus herd were biased upwards by the partial confounding of birth year with sire and were much higher than the other estimates.

Heritability estimates of longevity in beef cattle are not available from previous studies. However, many heritability estimates on similar traits have been reported in dairy cattle. Such heritability estimates have ranged from 0.00 to 0.39 with a mean of 0.12 (Wilcox et al., 1957; Parker et al., 1960; Plowman and Gaalaas, 1960; Evans et al., 1964; White and Nichols, 1965; Miller et al., 1967; Hargrove et al., 1969; Gill and Allaire, 1976; Hoque and Hodges, 1980). The estimates from the Hereford herd in the present study are within the range of those previous results, but higher than their average.

It was not explained in previous dairy studies whether only complete cohorts were utilized for the analyses. If incomplete cohorts were included, then heritability estimates likely were biased. In regression analysis, when a daughter and a dam are paired, a dam can be paired only with those of her daughters which have left

the herd. (Otherwise longevity traits of daughters could not have been computed.) Some daughters of some dams will still be in the herd when incomplete cohorts are included. The dam is then allowed to pair with daughters which left the herd at relatively young ages but cannot pair with daughters that are still surviving. This would tend to bias the daughter-dam regression downward from what it would have been in a population of complete cohorts.

In paternal half-sib analyses, daughters are nested within a sire, and if incomplete cohorts are included, then only those daughters which left the herd at relatively early ages would be included. This would reduce the apparent variance within the paternal half-sib group, deflate the relative variance among sires, and bias the apparent heritability of the trait.

White and Nichols (1965) suggested, from reviewing studies in dairy cattle, in their dairy study that heritability estimates of herd life using paternal half-sib analyses had been higher, on average, than estimates from daughter-dam regression. For instance, Evans et al. (1964) reported that heritability estimates of length of herd life from daughter-dam regression and paternal half-sib analysis were 0.00 and 0.39, respectively. A possible cause for the difference is the likely inclusion of incomplete cohorts in their analyses. In the present

study, in spite of having utilized only complete cohort groups, estimates in Herefords from paternal half-sib analysis were slightly but not significantly higher than the regression estimates. A possible explanation would be that daughters of a sire, on average, would have an additive genetic relationship slightly higher than .25, because some would also be related through their dams. We were not able to quantify and therefore account for this possibly increased additive genetic relationship. This would also be true in Angus.

Although heritability estimates for longevity in beef cattle are not available, a few reports suggest the existence of heterosis and breed effects on longevity. From Nunez et al. (1984), heterosis for longevity averaged 1.36 $\pm$ 0.58 yr or 16.2 $\pm$ 6.9 % and was greater for reciprocal crosses of Hereford - Angus (21.1 %) than for Angus - Shorthorn (14.8 %) or Hereford - Shorthorn (12.8 %). Stewart and Martin (1981) studied long-term productivity in purebred and crossbred cows of Angus and Milking Shorthorn parentage. Angus cows were in the herd longer and produced more calves and more total calf weight at weaning than Shorthorn cows, whose calves had higher average weaning weights. Crossbred cows were in the herd longer, produced more calves of higher average weight and produced more calf weight per year in the herd and per

lifetime than their straightbred counterparts.

Genetic and Phenotypic Correlations. In the Hereford herd, genetic correlations between early life traits and longevity traits from daughter-dam regression were negative and generally of low magnitude for either birth weight or weaning weight, but positive and moderate for weaning condition score (table 4). Estimates from paternal half-sib analysis were positive and moderate for either birth weight or weaning condition score and longevity traits, and high for the genetic correlations of weaning weight and longevity.

In the Angus herd, weaning weight was missing for daughter and(or) dam in a significant number of daughter-dam pairs. There was, therefore, too little data to justify estimating genetic correlations from daughter-dam regression, and genetic correlations were estimated only from paternal half-sib analysis (table 5). Also birth weight and weaning condition score were not available in that herd. Genetic correlations between weaning weight and longevity ranged from 0.53 to 0.57, which are consistent with results from paternal half-sib analysis in the Hereford herd.

Phenotypic correlations between early life traits and longevity all were low, ranging from -0.04 to 0.16 in both herds (table 4 and table 5). Thus, early life traits

examined in this study were not good indicators of subsequent lifespan. Saoud and Hohenboken (1984) in sheep were likewise not able to identify early life traits that were predictive of a ewe's subsequent longevity.

The phenotypic relationship between longevity and weaning weight was further examined by plotting least-squares means for adjusted weaning weight against classes of longevity, for which length of life from birth to disposal (AGE) is presented as an example (figure 5). See appendix figure 1 and 2 for FST and NUM, respectively. In both the Hereford and the Angus herd, there was no definite linear nor quadratic trend in the relationship of weaning weight with longevity traits.

Lx and Px Statistics. To illustrate Lx curves for the Angus herd, curves from several sires of the 1959, 1960 and 1961 cohorts are plotted in figure 6. See appendix figure 3 and 4 for Lx curves from sires of 1962 to 1964 and 1965 to 1966, respectively. "Home sire" means daughters of composite unknown home bulls (clean-up bulls) in contrast to bulls used by artificial insemination.

First of all, notice that several of the curves resemble a composite line which consists of two straight lines, the first of which has a steeper decline than the second. Such Lx curves resemble a boomerang.

The Lx curves varied markedly among sires, and the

differences among some sires became apparent in the beginning few years. Some sires had a very good persistency, and no replacement daughters were lost until daughters reached 4 years of age. Other sires began losing daughters at 2 years of age. Throughout the entire range in age, the rates of daughter loss were quite different. If the median or half life of Lx (Lx = 0.5) is compared among sires, 50% of daughters were lost by approximately 5 years of age in three sires but at approximately 7 and 9 years of age in the other two that are illustrated in figure 6. If sires are compared at a fixed daughter's age of 10 years, these bulls had lost 97%, 94%, 95%, 84% and 54% of replacement daughters by that time. Sire A : 61' had a nearly straight Lx curve, in consequence of a slow and consistant rate of daughter loss. In contrast, other sires had the typical boomerang shape of Lx.

Within birth year, Lx curves for sire A and home sires were similar for 1960 but remarkably different for 1961. Between birth years 1960 vs. 1961, Lx curves were quite different. This is in agreement with preliminary analyses in which birth year was a significant source of variation in the longevity traits.

Home sires and sire A in 1961, which differed dramatically in their Lx curves, were chosen to illustrate graphically the Px statistic (Figure 7). As expected, the

amplitude of variation for Px of sire A was less than for that of the home sires, which had the steeper and shorter Lx curve. By advanced ages, the Px statistic has small numbers of individuals in both the numerator (Lx+1) and denominator (Lx), so it is not surprising that it fluctuates drastically after x equals 10 to 12 years of age.

The variations of Lx and Px curves among sires shown in figures 6 and 7 led us to estimate heritabilities of those statistics. Since only those sires which had more than 20 replacement daughters were chosen for Lx and Px computation, 14 sires from 8 birth years were available for the analysis, and analysis within birth year was not feasible.

Heritability estimates of Lx and Px at most ages were quite high (table 6), due probably to the partial confounding of birth year and sire. These estimates were similar to the heritability estimates of longevity traits from paternal half-sib analyses in the Angus herd, which also were biased by the confounding effect. Even though these heritability estimates of Lx and Px likely are biased upwards, the results are interesting since they show a trend of variation through different ages of daughters. When daughters were 3 years to 6 years of age, heritability estimates of Lx and Px were the highest. This

suggests that most of the difference (partly genetic and partly environmental) in daughters' lifespan among sires was expressed during the period from second calving to fifth.

If they were based upon sufficient data and were not confounded with birth year or other effects, both Lx and Px statistics could clearly characterize the superiority or inferiority of sires in thier daughters'longevity. The oldest age that a daughter can achieve probably is of limited importance. Rather the difference in proportion of daughters remaining in the herd at some fixed age is economincally more important. The reference age depends upon many factors, such as economics, environment and management; and simulation studies might help to decide the optimum age for a particular herd. Variable Lx curves, indicating differences in inherent longevity, could be simulated and built into economic models.

Conclusions

The distinction between complete and incomplete cohorts is important in studies of longevity in animals. Including only complete cohorts is suggested for future

longevity studies, in order to avoid biased results. This study suggested the existence of moderate genetic variation for longevity traits in beef cattle and therefore the possibility of genetic improvement for this trait. However, if selection for longevity is based on the direct measurement of longevity in the animals which will be selected, a major problem is a decrease in annual genetic improvement of all traits under selection, due to the increase in generation interval. For instance, it is not practical to progeny test most bulls for longevity. One way to overcome this would be to select on early life traits which were genetically correlated to length of herd life. The efficacy of using birth weight, weaning weight and weaning condition score as early life selection criteria for longevity is dubious, because genetic correlation estimates from daughter-dam regressions and paternal half-sib analyses were not in agreement. Another approach would be breeding value estimation, using information from relatives belonging to complete cohorts. Such data, however, will be limited for many individuals, and estimated breeding values consequently will have low accuracy. Phenotypic correlations between early life traits and longevity traits in this study all were too low to allow accurate prediction of subsequent herd life from the early life traits. This study suggested that life

history statistics Lx and Px effectively could illustrate attrition patterns of herds and of daughters of a sire. The Lx curves varied markedly among sires, and the differences among some sires became apparent in the beginning few years. The use of those statistics for future longevity studies is recommended.

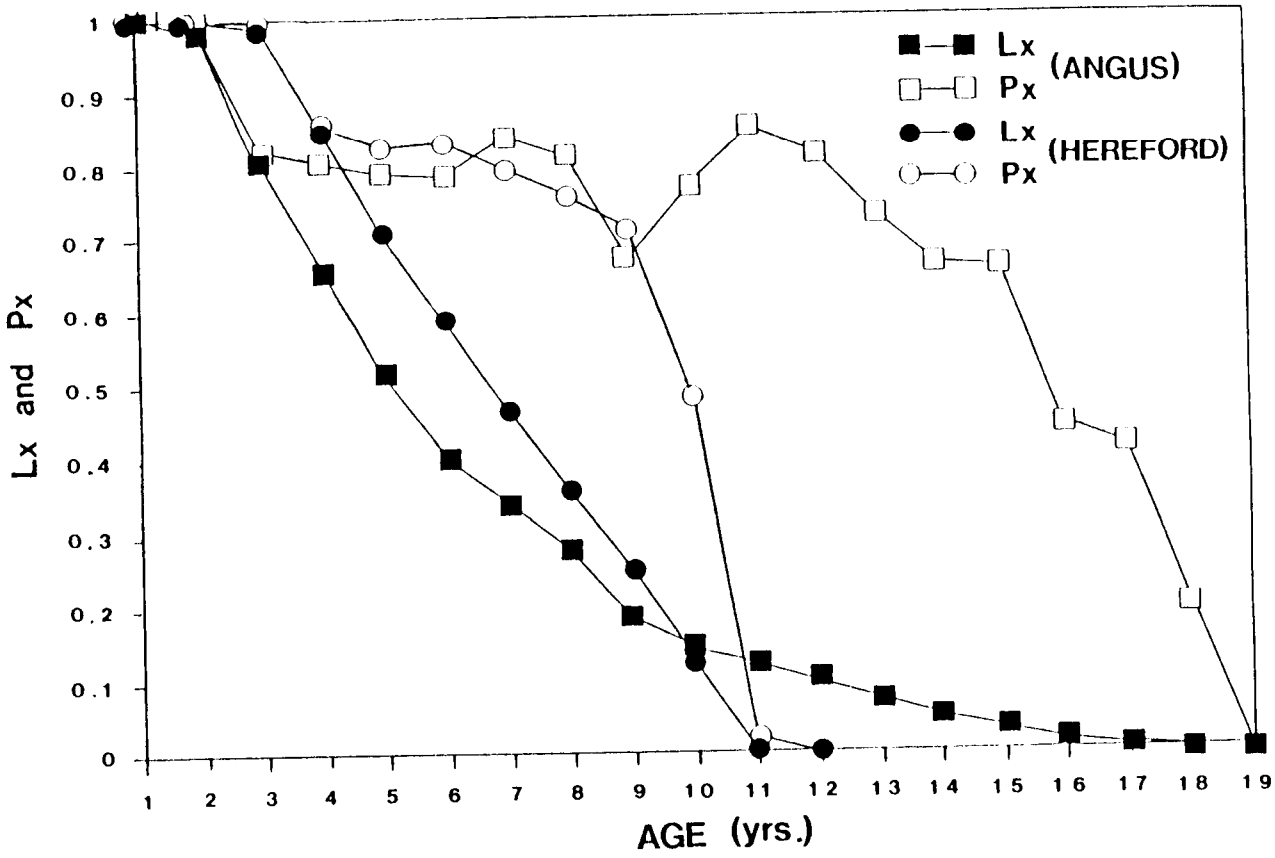


Figure 4. Relationship between age and survivorship (Lx) and survival rate (Px) for the Hereford and Angus herds.

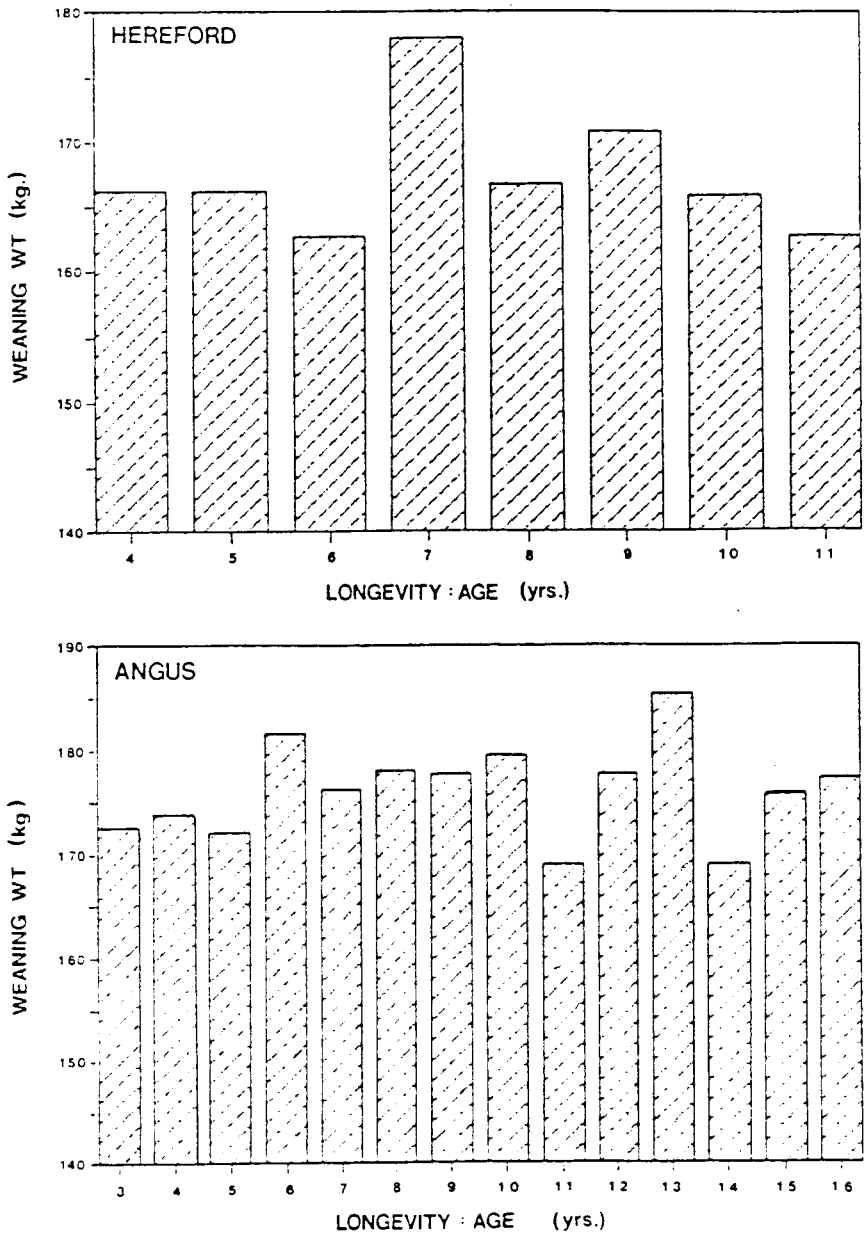


Figure 5. Relationship between adjusted weaning weight and AGE for the Hereford (upper diagram) and Angus (lower diagram) herds.

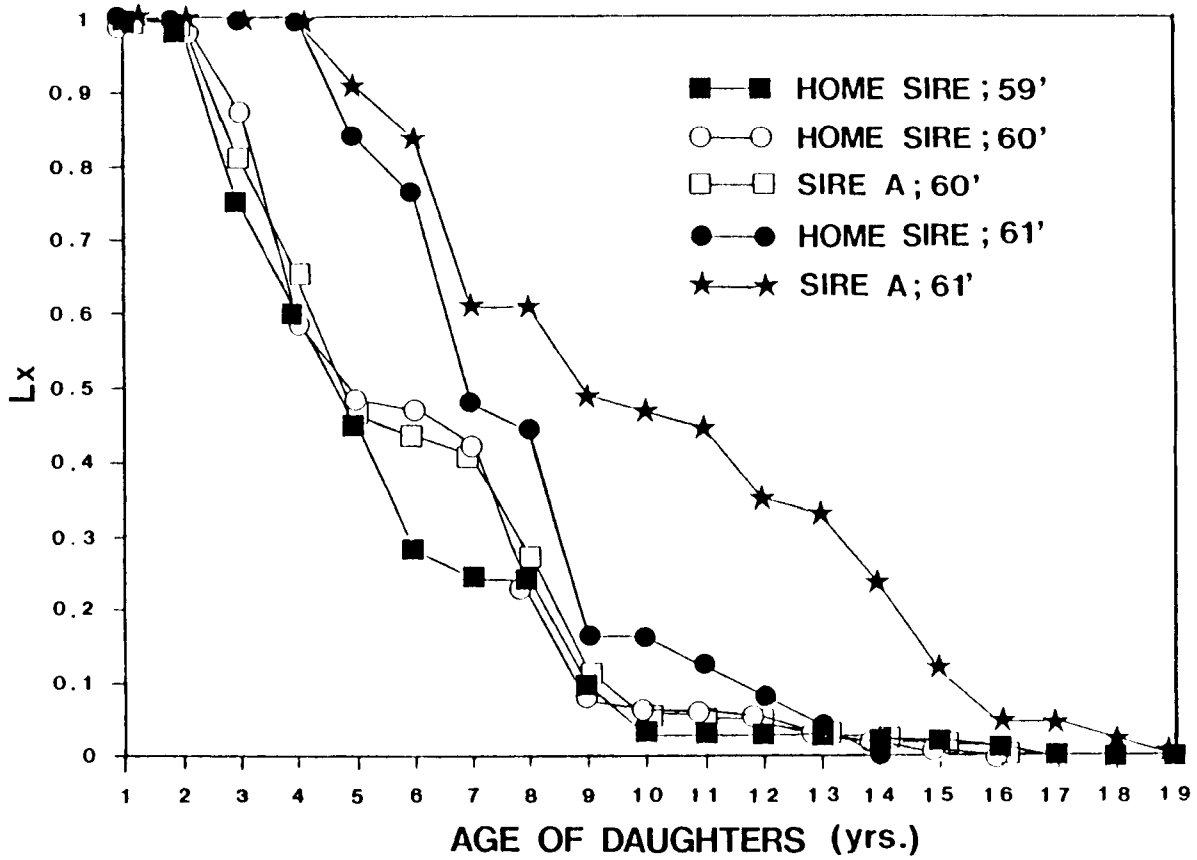


Figure 6. Relationship between age and survivorship (Lx) for several sires in the Angus herd (Cohorts 1959 - 1961).

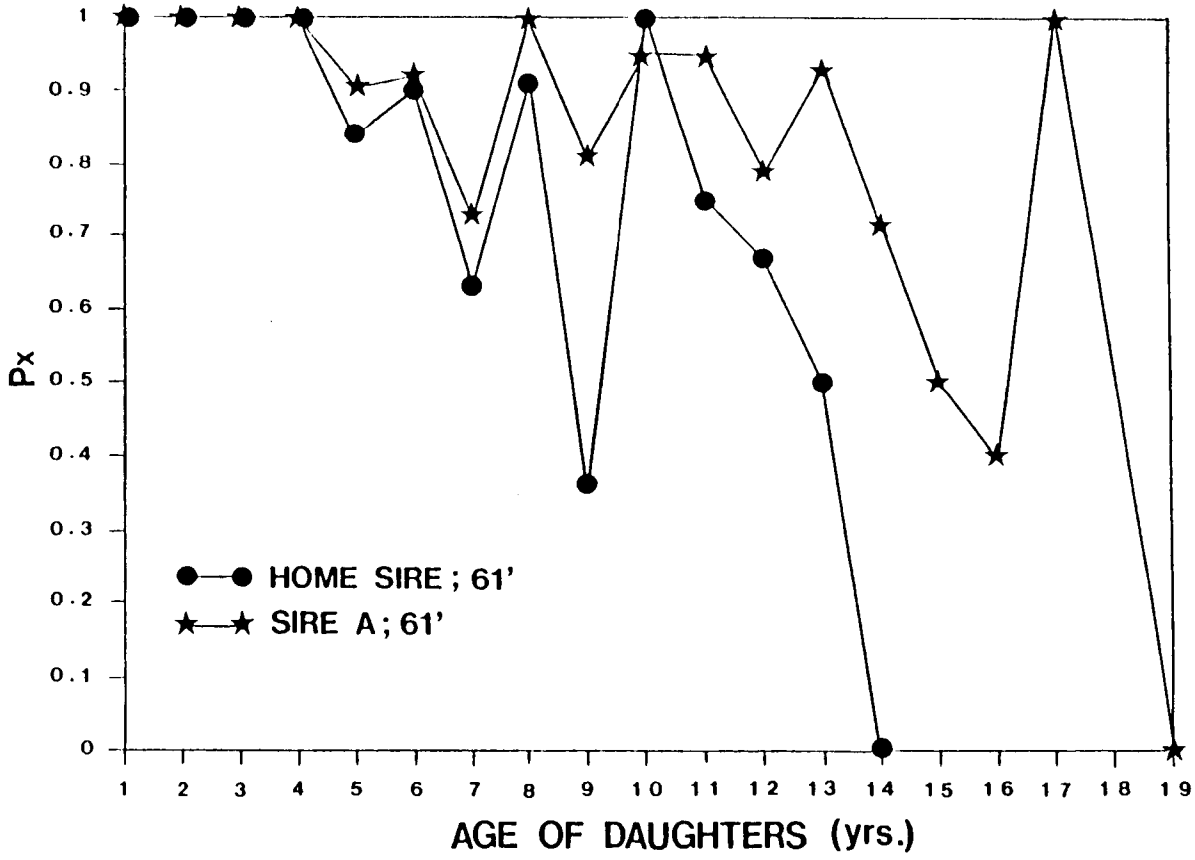


Figure 7. Relationship between age and survival rate (Px) for daughters of composite cleanup bulls vs. one artificial insemination sire for the 1961 cohort in the Angus herd.

TABLE 2. MEANS, STANDARD ERRORS, MEDIANS, MAXIMUM (MAX) AND MINIMUM (MIN) VALUES AND COEFFICIENTS OF VARIATION (CV) OF LONGEVITY TRAITS IN THE HEREFORD AND ANGUS HERDS

Trait[a]	Mean	SE	Median	Max	Min	CV(%)
FST	4.21[b]	0.06	4	10	1	57
	4.49[c]	0.13	3	17	0	81
AGE	7.40	0.06	7	14	3	32
	6.68	0.12	6	19	2	54
NUM	3.46	0.06	3	9	0	62
	3.66	0.11	3	16	0	89

[a]Length of life from first calving to disposal (FST), age at disposal (AGE) and lifetime number of calves weaned per cow (NUM).
[b]The Hereford herd (1452 cows:1957 - 1971 cohorts).
[c]The Angus herd (836 cows:1959 - 1966 cohorts).

TABLE 3. LEAST-SQUARES ANALYSES OF VARIANCE FOR LONGEVITY TRAITS (FST, AGE, AND NUM) WHEN BIRTH YEAR, AGE OF A COW'S DAM AND WEANING AGE WERE INCLUDED IN THE MODEL

	Mean squares (Hereford)				Mean squares (Angus)			
Source	df	FST	AGE	NUM	df	FST	AGE	NUM
Birth year	14	12.4 **	11.9 **	10.5 **	7	116.9 **	116.8 **	92.2 **
Age of dam	8	9.1	9.9	5.30	10	27.2 **	27.4 **	22.3 **
Weaning age regression	1	5.43	8.03	2.84	1	.21	.01	3.45
Residual	1428	5.8	5.7	4.6	817	12.0	11.7	9.6
R2		.03	.03	.03		.11	.11	.12

**P<.01.

TABLE 4. ESTIMATES OF HERITABILITIES, GENETIC CORRELATIONS AND PHENOTYPIC CORRELATIONS[a] FOR LONGEVITY AND EARLY LIFE TRAITS FROM DAUGHTER-DAM REGRESSIONS AND PATERNAL HALF-SIB ANALYSES IN THE HEREFORD HERD

Trait[b]	FST	AGE	NUM	BW	WW	WC
FST	.16±.08[c]	1.00±.02	.96±.03	-.13±.25	-.14±.36	.51±.49
	.26±.08	.97±.02	1.00±.02	.10±.28	.58±.22	.19±.24
AGE	.95	.18±.08	.92±.05	-.03±.21	-.11±.30	.44±.42
	.96	.24±.08	.99±.03	.16±.29	.65±.24	.31±.23
NUM	.94	.90	.16±.08	-.10±.27	-.42±.41	.44±.49
	.93	.90	.22±.08	.33±.29	.72±.23	.23±.23
BW	.01	.01	.00	.23±.07	.14±.33	-.87±.62
	.00	.01	-.01	.23±.09	.51±.20	.09±.24
WW	-.03	-.04	-.02	.29	.11±.08	-.48±.90
	-.03	-.03	-.02	.28	.61±.23	.52±.14
WC	-.01	-.01	.01	.04	.44	.08±.09
	.01	.01	.02	.06	.50	.41±.10

[a]Heritabilities on the diagonal; genetic correlations above the diagonal; phenotypic correlations below the diagonal.
[b]Length of life from first calving to disposal (FST), age at disposal (AGE), lifetime number of calves weaned per cow (NUM), birth weight (BW), weaning weight (WW) and weaning condition score (WC).
[c]Daughter-dam (919 pairs) : first row.
Half-sib (an average of 7 daughters/12 sires/15 years) : second row.

TABLE 5. ESTIMATES OF HERITABILITIES, GENETIC CORRELATIONS AND PHENOTYPIC CORRELATIONS[a] FOR LONGEVITY AND WEANING WEIGHT FROM DAUGHTER-DAM REGRESSIONS AND PATERNAL HALF-SIB ANALYSES IN THE ANGUS HERD

Trait[b]	FST	AGE	NUM	WW
FST	.05±.15[c] .66±.24[d]	.97±.07 1.00±.00	1.05±.25 .99±.01	.54±.24[e]
AGE	.99 1.00	.05±.15 .69±.25	1.00±.30 .99±.01	.53±.24
NUM	.96 .96	.95 .96	.03±.14 .62±.23	.57±.23
WW	.16	.15	.16	.41±.10

[a]Heritabilities on the diagonal; genetic correlations above the diagonal; phenotypic correlations below the diagonal.
[b]Length of life from first calving to disposal (FST), age at disposal (AGE), lifetime number of calves weaned per cow (NUM) and weaning weight (WW).
[c]Genetic parameter estimates from daughter-dam regression (255 pairs) are shown in the first row for FST, AGE and NUM.
[d]Genetic parameter estimates from paternal half-sib analysis (average of 24 daughters per 17 sires) are shown in the second row for FST, AGE and NUM.
[e]Heritability, genetic correlations and phenotypic correlations involving WW are from paternal half-sib analysis, because of insufficient data for daughter-dam regression.

TABLE 6. HERITABILITY ESTIMATES OF Lx and Px STATISTICS BY THE CHI-SQUARE METHOD FOR THE ANGUS HERD (14 SIRES) INCLUDING BIRTH YEAR FROM 1959 TO 1966.

Age of Daughters (yr)	Life Table Statistic	
	Lx	Px
2	0.47 ± 0.19	0.47 ± 0.19
3	0.73 ± 0.26	0.85 ± 0.29
4	0.75 ± 0.26	0.77 ± 0.27
5	0.54 ± 0.21	0.92 ± 0.31
6	0.43 ± 0.18	0.76 ± 0.29
7	0.14 ± 0.08	0.06 ± 0.10
8	0.19 ± 0.10	0.46 ± 0.24
9	0.27 ± 0.13	0.64 ± 0.31
10	0.39 ± 0.17	0.62 ± 0.36
11	0.36 ± 0.16	-0.15 ± 0.22
12	0.26 ± 0.13	-0.36 ± 0.20
13	0.28 ± 0.13	-0.06 ± 0.40
14	0.20 ± 0.11	0.43 ± 0.64

References

Allaire, F. R. and E. P. Cunningham. 1980. Culling on low milk yield and its economic consequences for the dairy herd. Livestock Prod. Sci. 7:349.

Bohren, B. B., H. E. Mckean and Y. Yamada. 1961. Relative efficiencies of heritability estimates based on regression of offspring on parent. Biometrics 17:481.

DeNise, R. S. K. and D. E. Ray. 1987. Postweaning weights and gains of cattle raised under range and gain test environments. J. Anim. Sci. 64:969.

Evans, D. L., C. Brantron and B. R. Farthing. 1964. Heritability estimates and interrelationships among production per day of productive life, longevity, breeding efficiency, and type in a herd of Holstein cows. J. Dairy Sci. (Suppl. 1):699 (Abstr.).

Gill, G. S. and F. R. Allaire. 1976. Genetic and phenotypic parameters for a profit function and selection method for optimizing profit in dairy cattle. J. Dairy Sci. 59:1325.

Hargrove, G. L., J. J. Salazar, and J. E. Legates. 1969. Relationships among first-lactation and lifetime measurements in a dairy population. J. Dairy Sci. 52:651.

Hazel, L. N. 1943. The genetic basis for constructing selection indexes. Genetics 28:476.

Hellbusch, C. 1982. One Bar Eleven Ranch: Its owner, its Angus. Angus J. Nov.:170.

Hoque, M. and J. Hodges. 1980. Genetic and phenotypic parameters of lifetime production traits in Holstein cows. J. Dairy Sci. 63: 1900.

Kay, R. D. and E. Rister. 1977. Income tax effects on beef cow replacement strategy. South. J. Agr. Econ. 9:169.

Kempthorne, O. and O. B. Tandon. 1953. The estimation of heritability by regression of offspring on parent.

Biometrics 9:90.

Melton, B. E. 1980. Economics of beef cow culling and replacement decisions under genetic progress. West J. Agr. Econ. 5:137.

Miller, P., L. D. Van Vleck and C. R. Henderson. 1967. Relationship among herd life, milk production and calving interval. J. Dairy Sci. 50:1283.

Nunez, R. D., L. V. Cundiff, G. E. Dickerson and K. E. Gregory. 1984. Heterosis of longevity in beef cattle. J. Anim. Sci. 59:Suppl. 1:180.

O'Mary, C. C. and I. A. Dyer. 1978. Commercial Beef Cattle Production. 2nd edition. Lea & Febiger, Philadelphia.

Parker, J. B., N. D. Bayley, M. H. Fohrman and R. D. Plowman. 1960. Factors influencing dairy cattle longevity. J. Dairy. Sci. 43:401.

Plowman, R. D. and R. F. Gaalaas. 1960. Heritability estimates of longevity in Holstein-Friesian cattle. J. Dairy. Sci. 43:877.

Recve, E. C. R. 1955. The variance of the genetic correlation coefficent. Biometrics 11:357.

Robertson, A. and Lerner, I. M. 1949. The heritability of all-or-none traits : Viability of poultry. Genetics 34:395.

Rogers, L. F. 1972. Economics of replacement rates in commercial beef herds. J. Anim. Sci. 34:921.

Saoud, N. B. and W. D. Hohenboken. 1984. The effect of selection in retrospect on lifetime production efficiency in sheep. J. Anim. Sci. 59:620.

Schons, D., W. D. Hohenboken and J. D. Hall. 1985. Population analysis of a commercial beef cattle herd. J. Anim. Sci. 61:44.

Stewart, T. S. and T. G. Martin. 1981. Mature weight, maternal performance and their interrelationships in purebred and crossbred cows of Angus and Milking Shorthorn parentage. J. Anim. Sci. 52:51.

Turner, H. N. and S. S. Y. Young. 1969. Quantitative Genetics in Sheep Breeding. Cornell University Press,

Ithaca, New York.

Van Arendork, J. A. M. 1986. Economic importance and possibilities for improvement of dairy cow herd life. Third World Congress on Genetics Applied to Livestock Production 9:95.

White, J. M. and J. R. Nichols. 1965. Relationships between first lactation, later performance, and length of herd life in Holstein-Friesian cattle. J. Dairy Sci. 48:468.

Wilcox, C. J., K. O. Peau and J. W. Bartlett. 1957. An investigation of the inheritance of female reproductive performance and longevity, and their interrelationships within a Holstein-Friesian herd. J. Dairy Sci. 40:942.

BIBLIOGRAPHY

Absher, P. M., R. G. Absher and W. D. Barnes. 1974. Geneologies of clones of diploid fibroblasts. Exp. Cell. Res. 88:95.

Allaire, F. R. and E. P. Cunningham. 1980. Culling on low milk yield and its economic consequences for the dairy herd. Livestock Prod. Sci. 7:349.

Batra, T. R., E. B. Burnside and M. G. Freeman. 1971. II. Effect of herd size and production level on dairy cow disposal patterns. Can. J. Anim. Sci. 51:85.

Basuthakar, A. K., P. J. Burfening, J. L. Van Horn and R. L. Blackwell. 1973. A study of some aspects of lifetime production in Targhee and Columbia sheep. J. Anim. Sci. 36:813.

Bohren, B. B., H. E. McKean and Y. Yamada. 1961. Relative efficiencies of heritability estimates based on regression of offspring on parent. Biometrics 17:481.

Boylan, W. J. 1985. Crossbreeding for fecundity, In : Genetics of Reproduction in Sheep, edited by Land, R. B. and D. W. Robinson. Butterworths, London.

Bradford. G. E. and H. H. Meyer. 1986. Economic evaluation of breeding objectives for sheep and goats : Practical considerations and examples. Third World Congress on Genetics Applied to Livestock Production. 9:479.

Burnside, E. B., A. E. McClintock and K. Hammond. 1984. Type, production and longevity in dairy cattle: A review. Anim. Breed. Abstr. 52:711.

Burnside, E. B. and J. W. Wilton. 1970. Anatomical traits as they relate to productive utility. J. Dairy Sci. 53:837.

Carrel, A. 1912. On the permanent life of tissues outside of the organism. J. Exp. Med. 15:516.

Clarke, S. E., C. T. Gaskins and J. K. Hillers. 1982.

Systems analysis of beef production - effects of culling criteria on net income. J. Anim. Sci. 55:1982.

Clarke, S. E., C. T. Gaskins, J. K. Hillers and W. D. Hohenboken. 1984a. Mathematical modeling of afternative culling and crossbreeding strategies in beef production. J. Anim. Sci. 58:6.

Clarke, S. E., C. T. Gaskins, J. K. Hillers and W. D. Hohenboken. 1984b. Mathematical modeling of alternative selection strategies for beef production. J. Anim. Sci. 59:308.

Clarke, S. E. and W. D. Hohenboken. 1983. Estimation of repeatability, heritability and breed differences for lamb production. J. Anim. Sci. 56:309.

Comfort, A. 1964. Aging: The Biology of Senescence. Holt, Rinehart and Winston, Inc., New York.

Comfort, A. 1968. Feasibility in age research. Nature. 217:320.

Congleton, W. R., Jr. 1984. Dynamic model for combined simulation of dairy management strategies. J. Dairy Sci. 67:644.

Congleton, W. R. Jr. and R. E. Goodwill. 1980a. Simulated comparisons of breeding plans for beef production - part 2 - Hereford, Angus and Charolais sires bred to Hereford, Angus and Hereford-Angus dams to produce feeder calves. Ag. Systems 5:221.

Congleton, W. R. Jr. and R. E. Goodwill. 1980b. Simulated comparisons of breeding plans for beef production : part 3 - systems for producing feeder calves involving intensive culling and additional breeds of sire. Ag. Systems 5:309.

Congleton, W. R., Jr. and L. W. King. 1984. Profitability of dairy cow herd life. J. Dairy Sci. 67:661.

Cristofalo, V. J. and B. B. Sharf. 1973. Cellular senescence and DNA synthesis. Exp. Cell. Res. 76:419.

Curtis, H. J. and K. L. Gebhard. 1958. Radiation-induced aging in mice. Proc. 2nd. Int. Conf. on Peaceful Uses of Atomic Energy. 22:53.

Curtis, H. J. 1963. The late effects of radiation. Proc. Am. Phil. Soc. 107:5.

Curtis, H. J. and C. Crowley. 1963. Chromosome aberrations in liver cells in relation to the somatic mutation theory of aging. Radiat. Res. 19:337.

Curtis, H. and N. S. Barnes. 1985. Invitation to Biology, 4th edition. Worth Publishers, Inc., New York.

Dawkins, R. 1976. The Selfish Gene. Oxford Univ. Press. Oxford.

DeNise, R. S. K. and D. E. Ray. 1987. Postweaning weights and gains of cattle raised under range and gain test environments. J. Anim. Sci. 64:969.

Dobzhansky, T. 1958. Genetics of homeostasis and of senility. Ann. N. Y. Acad. Sci. 71:1234.

Eklund, J. and G. E. Bradford. 1977. Longevity and lifetime body weight in mice selected for rapid growth. Nature 26:48.

Evans, D. L., C. Brantron and B. R. Farthing. 1964. Heritability estimates and interrelationships among production per day of productive life, longevity, breeding efficiency, and type in a herd of Holstein cows. J. Dairy Sci. (Suppl. 1):699.

Everett, R. W., J. F. Keown and E. E. Clapp. 1976. Relationships among type, production, and stayability in Holstein cattle. J. Dairy Sci. 59:1505.

Failla, G. 1958. The aging process and cancenogenesis. Ann. N. Y. Acad. Sci. 71:1124.

Gaalaas, R. F. and R. D. Plowman. 1963. Relationship between longevity and production in Holstein-Friesian cattle. J. Dairy Sci. 46:27.

Gill, G. S. and F. R. Allaire. 1976a. Relationship of age at first calving, days open, days dry, and herdlife to a profit function for dairy cattle. J. Dairy Sci. 59:1131.

Gill, G. S. and F. R. Allaire. 1976b. Genetic and phenotypic parameters for a profit function and selection method for optimizing profit in dairy cattle. J. Dairy Sci. 59:1325.

Hargrove, G. L., J. J. Salazar, and J. E. Legates. 1969. Relationships among first-lactation and lifetime measurements in a dairy population. J. Dairy Sci. 52:651.

Harrison, B. J. and R. Holliday. 1967. Senescence and the fidelity of protein synthesis in Drosophila. Nature 213:990.

Hayflick, L. 1965. The limited in vitro lifetime of human diploid cell strains. Exp. Cell. Res. 37:614.

Hayflick, 1. 1977. The biology of aging. Nat, Hist. 7:22.

Hayflick, L. 1980. The cell biology of human aging. Sci. Amer. 242:58.

Hayflick, L. and P. S. Moorhead. 1961. The serial cultivation of human diploid cell strains. Exp. Cell. Res. 25:585.

Hazel, L. N. 1943. The genetic basis for constructing selection indexes. Genetics 28:476.

Hellbusch, C. 1982. One Bar Eleven Ranch: Its owner, its Angus. Angus J. Nov.:170.

Hohenboken, W. D. and S. E. Clarke. 1981. Genetic, environmental and interaction effects on lamb survival, cumulative lamb production and longevity of crossbred ewes. J. Anim. Sci. 53:966.

Holliday, R. 1975. Growth and death of diploid and transformed human fibroblasts. Fedn. Proc. 34:51.

Hopfield, J. J. 1974. Kinetic proofreading: A new mechanism for reducing errors in biosynthetic processes requiring high specificity. Proc. Nat. Acad. Sci. USA 71:4135.

Hogue, M. and J. Hodges. 1980. Genetic and phenotypic parameters of lifetime production traits in Holstein cows. J. Dairy Sci. 63: 1900.

Hudson, G. F. S. and L. D. Van Vleck. 1981. Relationship between production and stayability in Holstein cattle. J. Dairy. Sci. 64:2246.

Kay, R. D. and E. Rister. 1977. Income tax effects on beef

cow replacement strategy. South. J. Agr. Econ. 9:169.

Kempthorne, O. and O. B. Tandon. 1953. The estimation of heritability by regression of offspring on parent. Biometrics 9:90.

Kirkwood, T. B. L. 1977. Review article: Evolution of ageing. Nature 270:301.

Laughrea, M. 1982. On the error theories of aging: A review of the experimental data. Exp. Geront. 17:305.

Lints, F. A. 1978. Genetics and Aging. S. Karger. New York.

McCay, C. M., M. F. Crowell and L. A. Maynard. 1935. The effect of retarded growth upon the length of life span and upon the ultimate body size. J. Nutrition 10:63.

McCay, C. M., L. A. Maynard, G. Sperling and L. L. Barnes. 1939. Retarded growth, life span, ultimate body size and age changes in the albino rat after feeding diets restricted in calories. J. Nutrition. 18:1.

Macieira-Coflho, A., E. Loria and L. Berumen. 1975. Relationship between cell kinetic changes and metabolic events during cell senescence in vitro. In Cell Impairment in Ageing and Development. Plenum, New York.

Maynard Smith, J. and G. R. Price. 1973. The logic of animal conflict. Nature 246:15.

Medawar, P. B. 1952. An Unsolved Problem of Biology. H. K. Lewis & Co Ltd, London.

Melton, B. E. 1980. Economics of beef cow culling and replacement decisions under genetic progress. West J. Agr. Econ. 5:137.

Miller, P., L. D. Van Vleck and C. R. Henderson. 1967. Relationship among herd life, milk production and calving interval. J. Dairy Sci. 50:1283.

Nunez, R. D., L. V. Cundiff, G. E. Dickerson and K. E. Gregory. 1984. Heterosis of longevity in beef cattle. J. Anim. Sci. 59:Suppl. 1:180.

O'Mary, C. C. and I. A. Dyer. 1978. Commercial Beef Cattle

Production. 2nd edition. Lea & Febiger, Philadelphia.

Orgel, L. E. 1963. The maintenance of the accuracy of protein synthesis and its relevance to ageing. Proc. Natn. Acad. Sci. USA 49:517.

Parker, J. B., N. D. Bayley, M. H. Fohrman and R. D. Plowman. 1960. Factors influencing dairy cattle longevity. J. Dairy. Sci. 43:401.

Pearson, R. E. and A. E. Freeman. 1973. Effect of female culling and age distribution of the dairy herd on profitability. J. Dairy. Sci. 56:1459.

Plowman, R. D. and R. F. Gaalaas. 1960. Heritability estimates of longevity in Holstein-Friesian cattle. J. Dairy. Sci. 43:877.

Reeve, E. C. R. 1955. The variance of the genetic correlation coefficent. Biometrics 11:357.

Rendel, J. M. and A. Robertson. 1950. Some aspects of longevity in dairy cows. J. Exp. Agric. 18:49.

Renkema, J. A. and J. Stelwagen. 1979. Economic evaluation of replacement rates in dairy herds. 1. Reduction of replacement rates through improved health. Livestock Prod. Sci. 6:15.

Roberts, R. C. 1961. The lifetime growth and reproduction of selected strains of mice. Heredity 16:370.

Robertson, A. and J. S. F. Barker. 1966. The correlation between first lactation milk production and longevity in dairy cattle. Anim. Prod. 8:241.

Robertson, A. and I. M. Lerner. 1949. The heritability of all-or-none traits: viability of poultry. Genetics 34:395.

Rogers, L. F. 1972. Economics of replacement rates in commercial beef herds. J. Anim. Sci. 34:921.

Rose, M. R. 1984a. Review: The evolution of animal senescence. Can. J. Zool. 62:1661.

Rose, M. R. 1984b. Laboratory evolution of postponed senescence in Drosophila melanoguster. Evolution 38:1004.

Rose, M., and B. Charlesworth. 1980. A test of evolutionary theories of senescence. Nature 287:141.

Rose, M. R., and B. Charlesworth. 1981. Genetics of life history in Drosophila melanogaster. II. Exploratory selection experiments. Genetics 97:187.

Rose, M. R., M. L. Dorey, A. M. Coyle, and P. M. Service. 1984. The morphology of postponed senescence in Drosophila melanogaster. Can. J. Zool. 52:1576.

Sacher, G. A. 1959. Relation of lifespan to brain weight and body weight in mammals, In :Ciba Foundation Colloquia on Ageing, Vol.5, The Lifespan of Animals. Churchill, London.

Sacher, G. A. 1978. Evolution of longevity and survival characteristics in mammals, In: The Genetics of Aging, edited by E. L. Schneider. Plenum press, New York & London.

Samis, H. V. 1978. Molecular genetics of aging, In: The Genetics of Aging, edited by E. L. Schneider. Plenum press, New York & London.

Saoud, N. B. and W. D. Hohenboken. 1984a. Phenotypic relationships among early life traits and lifetime ewe production efficiency. J. Anim. Sci. 59:607.

Saoud, N. B. and W. D. Hohenboken. 1984b. The effect of selection in retrospect on lifetime production efficiency in sheep. J. Anim. Sci. 59:620.

Schmedt, G. H. and L. D. Van Vleck. 1974. Principles of Dairy Science. W. H. Freeman, San Francisco.

Schons, D., W. D. Hohenboken and J. D. Hall. 1985. Population analysis of a commercial beef cattle herd. J. Anim. Sci. 61:44.

Sharma, A. and G. Talukder. 1979. Genetical models of senescence in man. Nucleus 22:128.

Shelton, M. and J. W. Menzies. 1968. Genetic parameters of some performance characteristics of range fine-wool ewes. J. Anim. Sci. 27:1219.

Smith, S. P. and R. L. Quaas. 1984. Productive lifespan of bull progeny groups: Failure time analysis. J. Dairy Sci. 67:2999.

Sonneborn, T. M. 1978. The origin, evolution, nature, and causes of aging, In: The Biology of Aging, edited by Behnke, J. A., C. E. Finch and G. B. Moment. Plenum press, New York & London.

Stevenson, K. G. and H. J. Curtis. 1961. Chromosomal aberrations in irradiated and nitrogen mustard-treated mice. Radiat. Res. 15:774.

Stewart, T. S. and T. G. Martin. 1981. Mature weight, maturation rate, maternal performance and their interrelationships in purebred and crossbred cows of Angus and Milking Shorthorn parentage. J. Anim. Sci. 52:51.

Strehler, B. L. 1977. Time, Cells and Aging. 2nd edit. Academic press, New York.

Szilard, L. 1959. On the nature of the aging process. Proc. Natn. Acad. Sci. USA. 45:30.

Thapan, P. C., D. S. Bell and C. F. Parker. 1970. Reproductive performance in Targhee sheep. Indian J. Anim. Sci. 40:223.

Turner, H. N. and S. S. Y. Young. 1969. Quantitative Genetics in Sheep Breeding. Cornell University Press, Ithaca, New York.

Van Arendork, J. A. M. 1986. Economic importance and possibilities for improvement of dairy cow herd life. Third World Congress on Genetics Applied to Livestock Production. 9:95.

Van Doormaal, B. J., L. R. Schaeffer and B. W. Kennedy. 1985. Estimation of genetic parameters for stayability in Canadian Holsteins. J. Dairy Sci. 68:1763.

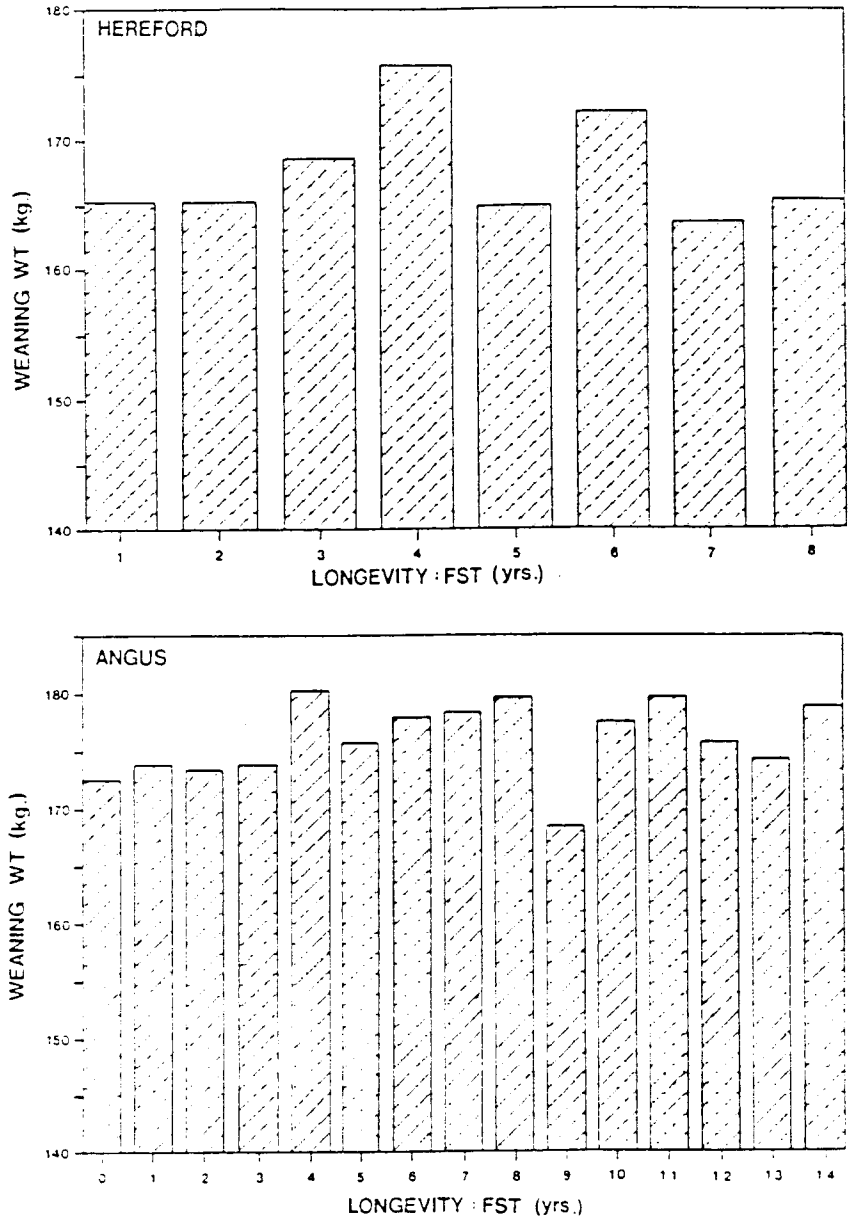
Vesely, J. A. and H. F. Peters. 1974. Lamb production from ewes of four breeds and their two breed and three breed crosses. Can. J. Anim. Sci. 54:543.

White, J. M. and J. R. Nichols. 1965. Relationships between first lactation, later performance, and length of herd life in Holstein-Friesian cattle. J. Dairy Sci. 48:468.

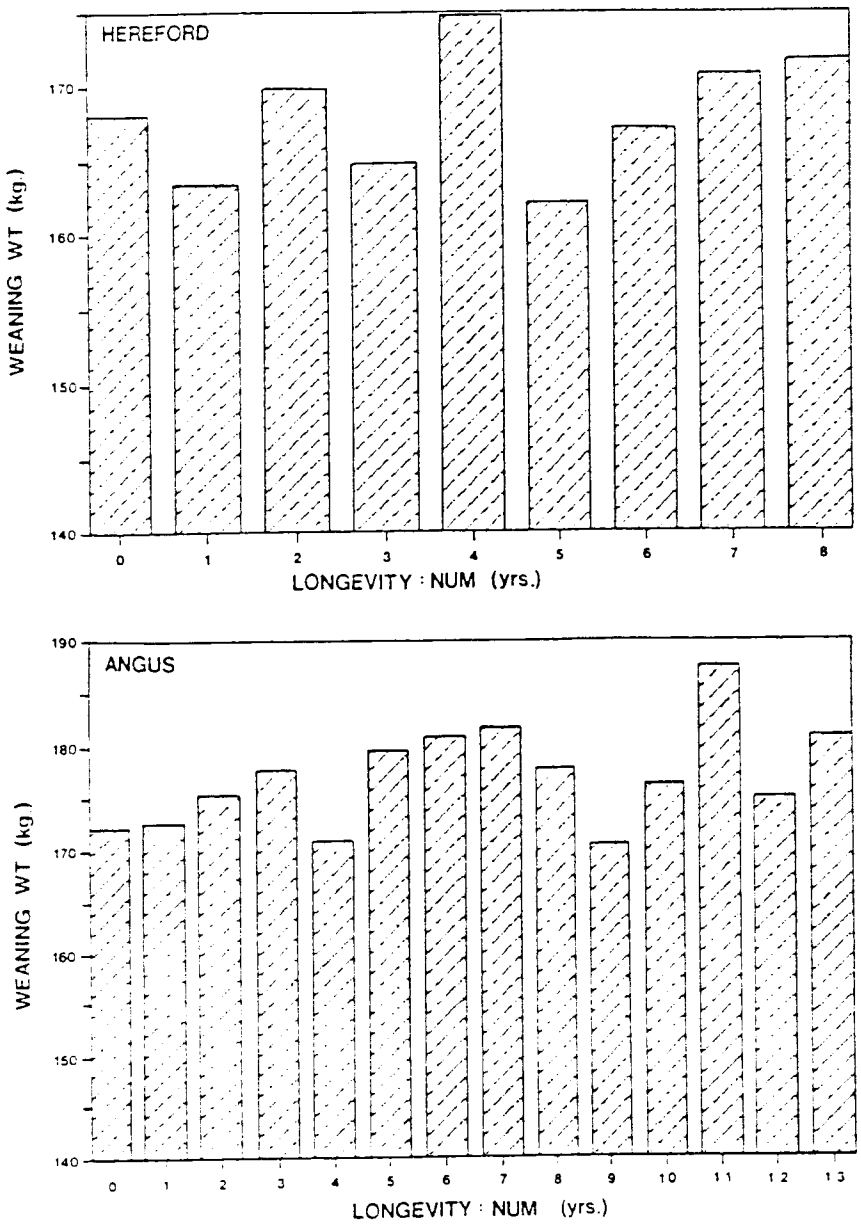
Wilcox, C. J., K. O. Peau and J. W. Bartlett. 1957. An

investigation of the inheritance of female reproductive performance and longevity, and their interrelationships within a Holstein-Friesian herd. J. Dairy Sci. 40:942.

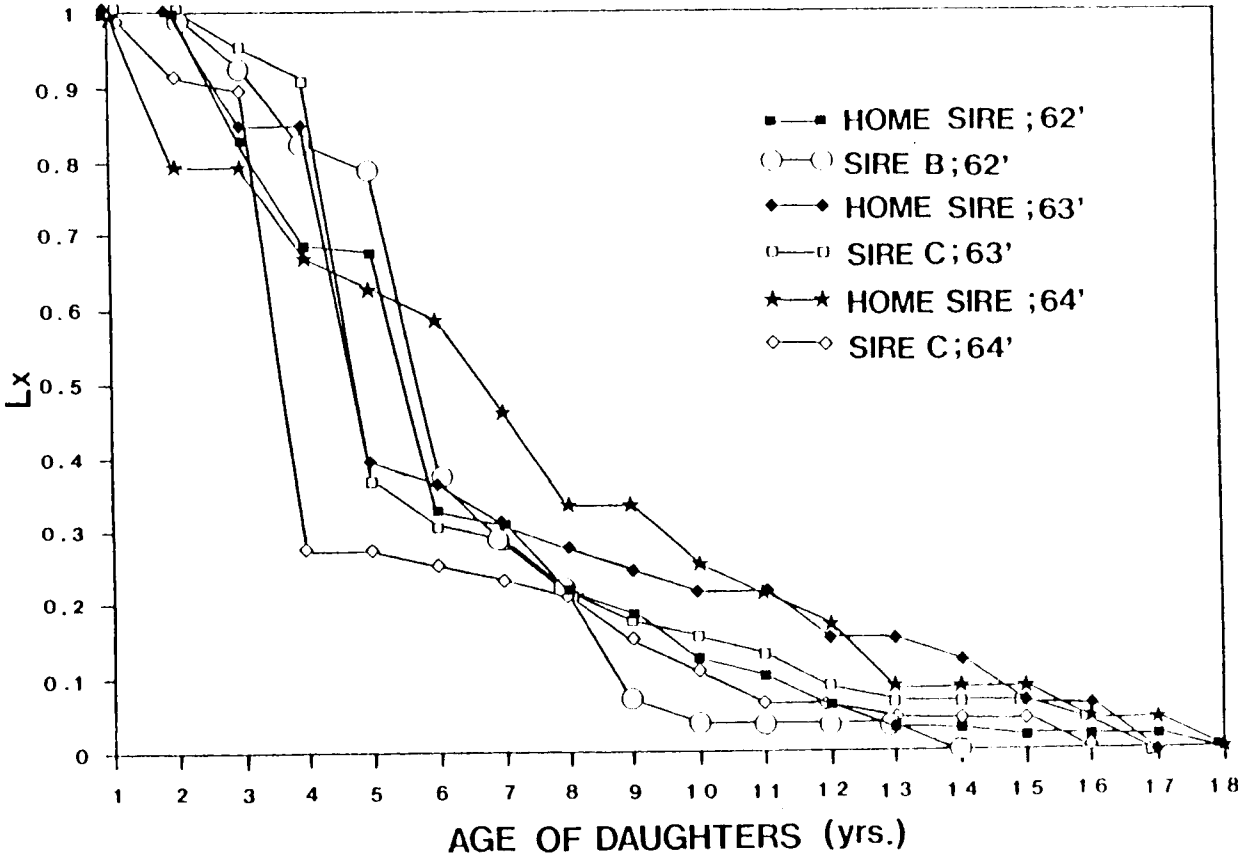
APPENDICES



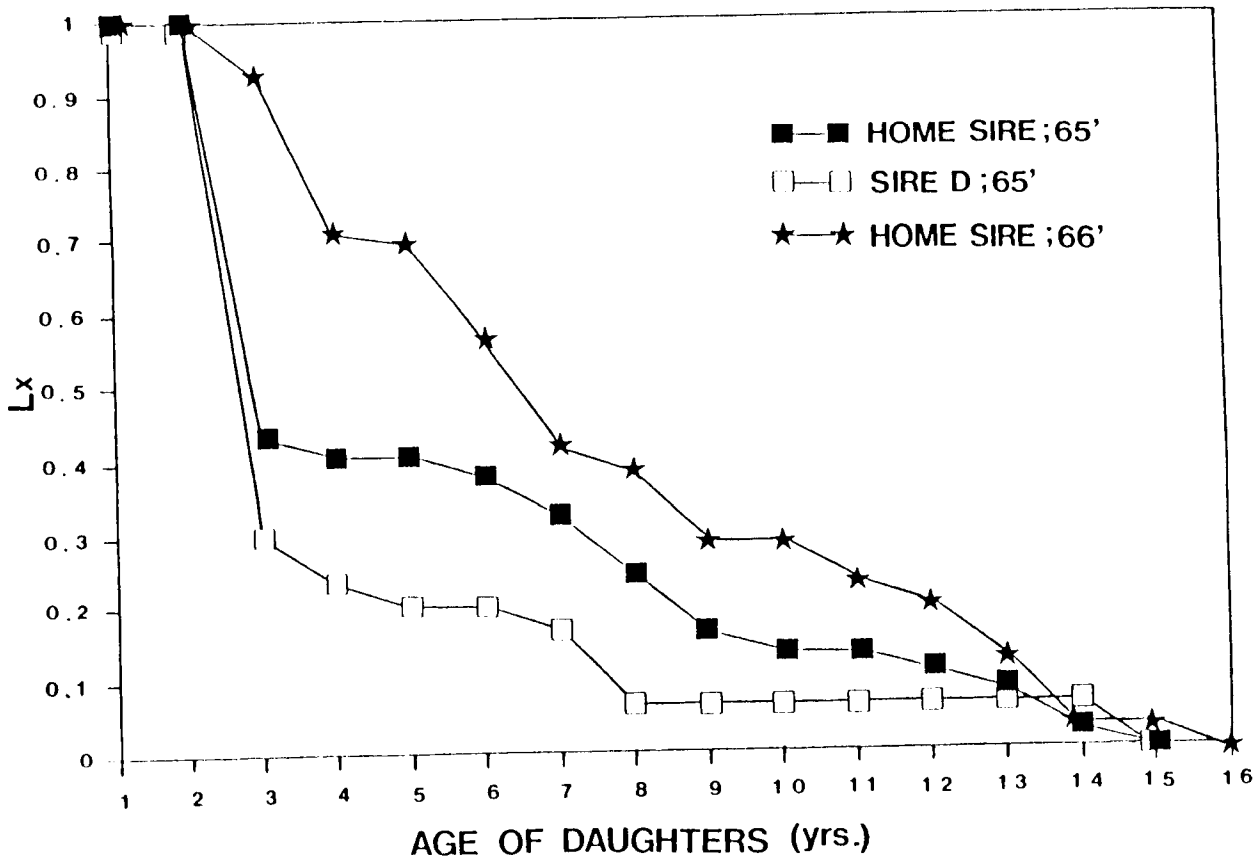
Appendix Figire 1. Relationship between adjusted weaning weight and FST for the Hereford (upper diagram) and Angus (lower diagram) herds.



Appendix Figure 2. Relationship between adjusted weaning weight and NUM for the Hereford (upper diagram) and Angus (lower diagram) herds.



Appendix Figure 3. Relationship between age and survivorship (Lx) for several sires in the Angus herd (Cohorts 1962 - 1964).



Appendix Figure 4. Relationship between age and survivorship (Lx) for several sires in the Angus herd (Cohorts 1965 - 1966).