References

1. Kennett, J., *Marine Geology*, Prentice-Hall Publishers, Englewood Cliffs, NJ, p. 747, 1982.
2. Imbrie, J. and Imbrie, K.P., *Ice Ages: Solving the Mystery*, Enslow Publishers, Short Hills, NJ, 1979.
3. Oard, M.J., *An Ice Age Caused by the Genesis Flood*, Institute for Creation Research, El Cajon, CA, pp. 15–18, 1990.
4. Oard, M.J., *The Frozen Record: Examing the Ice Core History of the Greenland and Antarctic ice Sheets*, Institute for Creation Research, El Cajon, CA, 2005.
5. Wunsch, C., quantitative estimate of the Milankovitch-forced contribution to observed Quaternary climate change, *Quaternary Science Reviews* **23**:1001, 2004.
6. Oard, M., *Frozen in Time: The Woolly Mammoth, the Ice Age and the Bible*, Master Books, Green Forest, AR, 2004.
7. Oard, ref. 3, pp. 135–166.
8. Oard, M.J., Are pre-Pleistocene rhythmites caused by the Milankovitch mechanism? *TJ* **11**(2):126–128, 1997.
9. Liu, Z. and Herbert, T.D., High-latitude influence on the eastern equatorial pacific climate in the early Pleistocene epoch, *Nature* **427**:720–723, 2004.
10. Billups, K., Low-down on a rhythmic high, *Nature* **427**:686–687, 2004.
11. Billups, ref. 10, p. 686.
12. Liu and Herbert, ref. 9, p. 722.
13. Oard, M.J., *Ancient Ice Ages or Gigantic Submarine Landslides?* Creation Research Society Monograph No. **6**, Creation Research Society, Chino Valley, AZ, pp. 11–17, 1997.

Mutations, selection and the quest for meatier livestock

Jean K. Lightner

One place that mutations and selection can be readily studied is within the livestock industry. Mutations that naturally occur in livestock can be selected for or against depending on their ability to meet the needs of this industry. A major product of the livestock industry is meat. Several mutations exist that increase muscle mass, decrease body fat and improve feed efficiency.

Beefed-up cattle

Some of the best known and most studied of these mutations are the ones associated with the double muscling phenotype in cattle. Animals possessing this phenotype are heavily muscled, particularly in the shoulders, back and upper hind limbs. This phenotype is present in a number of breeds, although it is variable in how strongly it is expressed. It has been actively selected for within the Belgian Blue and Piedmontese to the point where it has become a characteristic of those breeds. Belgian Blues with this phenotype have an 11-nucleotide deletion in the myostatin[1] gene that causes a frameshift which results in the formation of a premature stop codon.[2] When the myostatin protein is produced, it is thus severely truncated and missing nearly all of its active region. In the Piedmontese, the mutation involves the substitution of an adenine base for a guanine (G→A) in the myostatin gene, resulting in a myostatin protein with the typical amino acid cysteine being replaced by tyrosine in the active region.[3] At least four other mutations in the myostatin gene have been described associated with double muscling in various other breeds of cattle.[4]

In each case, without the functional myostatin protein, muscle growth continues uncontrolled in the animal at the expense of other bodily functions, including reproduction and normal fat and bone deposition. This translates into cattle that often possess hypoplastic (underdeveloped) reproductive tracts, experience high rates of infertility, and are more susceptible to stress and fractures.[5]

In cattle, the increase in muscle mass observed with myostatin gene mutations is due to hyperplasia, an increase in the number of muscle fibres.[6] This begins before birth and often results in dystocia, that is difficulty calving. A number of breeds or strains within breeds characterized by double muscling advertise that they have selected their animals for calving ease. For example, the Charolais breed, a beef breed not normally characterized by double muscling, has a strain which exhibits this trait. A trial done in the United Kingdom compared the calving performance of cows bred to a Culard (the French term for 'double-muscled') Charolais bull to those bred to a British Charolais bull. They chose a Culard bull with high calving ease scores. However, the scores seemed meaningless when four of the 16 calves died during birth and 6 of the 9 bull calves needed a calving jack to remove them from the cow. Only two of these 16 cows were able to deliver their calves without help. The economic losses didn't stop there; 35% of the cows bred to the Culard would not conceive again, likely due to internal injuries from the traumatic calvings. No premiums from the remaining calves could come close to making up for these losses. In contrast, only two of the cows bred to the British bull needed help; one needed only a little, the other needed a calving jack.[7] One must wonder what the term 'calving ease' means when applied to double-muscled animals. Less than 30% need a c-section and most of the bull calves do just fine being forcefully extracted from the dam with a calving jack. Needless to say, this does not fit most cattlemen's idea of calving ease.

Myostatin mutations are pleiotropic

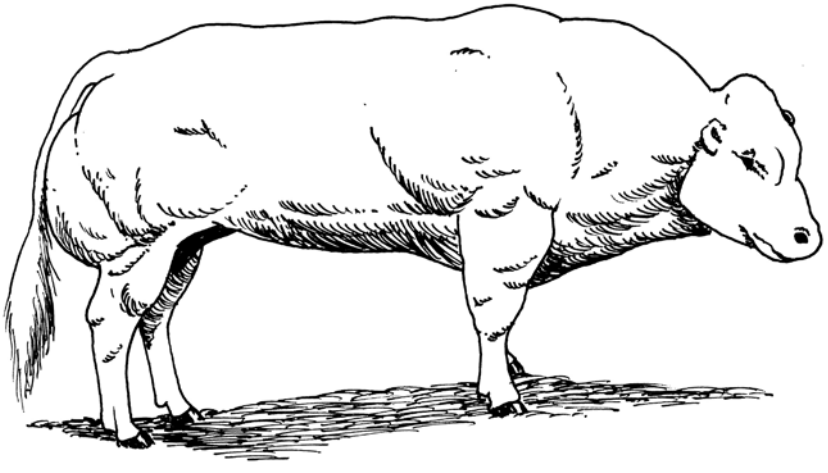
in their effects, meaning they affect a number of different body systems. Muscle mass is significantly increased; fat and bone mass are decreased along with the weight of the hide, liver and other internal organs. Studies have shown altered enzyme function[8] and plasma hormone levels[9] related to this condition. Even though there are some cattlemen who appear to be quite successful in raising these cattle, double muscling is considered a disease condition and is commonly selected against.

Beautiful buttocks in sheep

Myostatin gene mutations are not the only mutations associated with more muscular, leaner animals. There is a mutation in sheep known as callipyge, or 'beautiful buttocks'.[10] In this case, the increase in muscle mass is primarily in the back and hindquarters, the areas responsible for the highest-priced cuts of lamb. When first observed in a male Dorset in 1983, the lamb was named Solid Gold and saved for breeding.[11] Callipyge has an unusual inheritance pattern; the only lambs expressing the phenotype are those who receive the callipyge mutation from their sire. When a gene behaves differently depending on which parent it is inherited from, it is known as 'imprinting'. There are a number of genes in humans and animals that are known to behave this way. However, in callipyge any lamb receiving the mutation from the ewe will have a normal phenotype, regardless of what is inherited from the sire. This is known as 'polar overdominance' and callipyge is the first case of this to be discovered in mammals.[12]

Most body tissues are very dynamic. In muscle, protein synthesis and degradation are constantly taking place at rates that are very precisely controlled. A number of different factors are involved, many of which are just now being discovered, so that muscles are kept in optimal balance throughout different stages of growth and for different environmental conditions. It appears that the enlarged muscles of callipyge are maintained by a decrease in the normal protein degradation rate.[13]

Unlike the myostatin mutations of cattle, callipyge in sheep is the result of hypertrophy, an increase in the size of the muscle fibres. The hypertrophy begins after birth, so dystocia is not an issue. However, the meat from these lambs is characterized by a lack of tenderness. Efforts to overcome this problem have been largely unsuccessful. Although several post-slaughter methods of tenderization have been identified they are not widely used, likely due to problems with consumer acceptance and/or economic feasibility.[14] When it was initially discovered, callipyge was selected for; however, after the meat quality issue became apparent, it was usually selected against.



Fullblood Belgian Blue bull expressing the the double muscling phenotype. Double muscling, a condition associated with myostatin gene mutations, results in heavily muscled animals and higher carcass yields. However, it is also associated with a number of problems, most notably dystocia and infertility.

Characteristics of mutations

Mutations are mistakes in the genetic code and are associated with a loss of information.[15] Even though the mutations discussed here resulted in more muscular animals, it was at the expense of normal physiologic control mechanisms.

Mutations vary in their effect. It is possible for a mutation to occur in an area where it does not affect the final protein, and thus it may be considered neutral. On the other hand, many mutations affect critically important molecules and are lethal. The mutations discussed here fall somewhere in-between these two extremes, with double muscling being associated with more severe physical problems. These mutations added variety, which could be selected for or against. In most cases, apparent mutations would be selected against. Very rarely can a mutation be considered beneficial, and even these are only beneficial under certain circumstances.[16–18]

Another important characteristic of mutations is that they tend to accumulate in animal populations over time. This is known as genetic load. In cattle, a single mutated gene for myostatin may result in an animal that shows mild signs of double muscling. In callipyge and in simple recessive traits, an animal with a defective gene may show no signs of the abnormality. This makes it difficult, if not impossible, to select against many harmful mutations. Thus, many serious and deadly disorders continue to be passed on.[19]

Mutations are incompatible with the evolutionary idea that information increases over time and animals evolve upward. Due to genetic load, there is an overall reduction in fitness over time,

making the supposed long evolutionary ages highly suspect from a biological standpoint. Studying mutations has lead to a deeper understanding of the well-designed and finely tuned metabolic pathways that allow animals to thrive under a wide range of conditions. This is not consistent with something that improves in a stepwise fashion, but rather points to a designer whose intellect far surpasses our own.

Conclusion

Current knowledge of mutations fits incredibly well with the Bible's description of world history. Information must have a source; in the beginning God created everything. After man sinned, death entered the world. Mutations and genetic load are compatible with the description of all creation being in bondage to decay.[20] The Bible's timeframe for Earth's history, around 6,000 years, is reasonable given the rate at which we see mutations building up in animal populations today. Of course the Bible not only accurately describes the problem, it also gives us the solution.[21] Real world observations involving mutations and selection support the historical account given in the Bible.

References

1. Myostatin was formerly known as growth and differentiation factor 8 (GDF-8). The gene for this protein is found on bovine chromosome 2. Myostatin has been demonstrated to alter the expression of well over a dozen genes in myoblasts (cells from which mature muscle cells may develop). Yang, W., Zhang, Y., Ma, G., Zhao, X., Chen, Y. and Zhu, D., Identification of gene expression modifications in myostatin-stimulated myoblasts, *Biochemical & Biophysical Research Communications* **326**(3):660–666, 2005.
2. Three consecutive nucleotides form a codon which codes for a specific amino acid and sometimes for a stop codon. Since an 11-nucleotide deletion is not a multiple of three, it causes a shift in the normal reading frame of the myostatin gene.
3. Kambadur, R., Sharma, M., Smith, T.P.L. and Bass, J.J., Mutations in myostatin (GDF8) in double-muscled Belgian Blue and Piedmontese Cattle, *Genome Research* **7**(9):910–915, 1997, <www.genome.org/cgi/content/full/7/9/910>, 16 February 2005.
4. Karim, L., Coppieters, W., Grobet, L., Valentini, A. and Georges, M., Convenient genotyping of six myostatin mutations causing double-muscling in cattle using a multiplex oligonucleotide ligation assay, *Animal Genetics* **31**(6):396–399, 2000. There are a few examples of the double muscling phenotype that are not associated with mutations in the myostatin gene. Also, within the South Devon breed, some animals were identified as homozygous for the same 11-nucleotide deletion as found in Belgian Blues, yet they were not of the double-muscled phenotype. Thus, in some instances, the involvement of other genes seems likely. Smith, J.A., Lewis, A.M., Wiener, P. and Williams, J.L., Genetic variation in the bovine myostatin gene in UK beef cattle: allele frequencies and haplotype analysis in the South Devon, *Animal Genetics* **31**(5):306–309, 2000.
5. Double-muscled cattle, <www.clues.abdn.ac.uk:8080/mirrors/growth/ch7.1.html>, 16 February 2005.
6. In mice both hyperplasia and hypertrophy are seen. Ref. 3.
7. Wright, S., Double muscling is a disaster, *Farmers Weekly,* 25 October, p. 48, 2002.
8. Double-muscling, *The Merck Veterinary Manual;* <www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/90306.htm>, 16 February 2005.
9. Schwartzkopf-Genswein, K., Marbling in double muscled steers, 2003; <www.piedmontese-napa.com/Marbling%20in%20double%20muscled%20steers.htm>.
10. From the Greek *calli-* meaning beautiful and *-pyge* meaning buttocks.
11. Winstead, E.R., Genetic mutation explains 'beautiful buttocks' in sheep, *Genome News Network*, 2002; <www.genomenewsnetwork.org/articles/10_02/beautiful_buttocks.shtml>, 16 February 2005.
12. The result of an A to G transition in an intergenic region of ovine chromosome 18, the callipyge mutation is in an area some had presumptuously termed 'junk DNA'. It is now postulated that this portion of DNA is a long-range control element (LRCE), controlling the rate of transcription of surrounding imprinted genes. Davis, E., Jensen, C.H., Schroder, H.D., Farnir, F., Shay-Hadfield, T., Kliem, A., Crockett, N., Georges, M. and Charlier, C., Ectopic Expression of DKL1 protein in skeletal muscle of padumnal heterozygotes causes the callipyge phenotype, *Current Biology* **14**(20):1858–1862, 2004.
13. Lorenzen, C.L., Koohmaraie, M., Shackelford, S.D., Jahoor, F., Freetly, H.C., Wheeler, T.L., Savell, J.W. and Fiorotto, M.L., Protein kinetics in callipyge lambs, *J. Animal Science* **78**(1):78–87, 2000; <meat.tamu.edu/pdf/Jan78_2.pdf>, 17 Februaury 2005.
14. Solomon, M.B., The Callipyge phenomenon: tenderness intervention methods, *J. Animal Science* **77**, suppl. 2:238–242, 1999; <www.asas.org/JAS/papers/1999/am/am026.pdf>, 17 February 2005.
15. This is not meant to imply that all changes in the genome are necessarily mistakes. See: Ashcraft, C.W., Genetic variability by design, *TJ* **18**(2):98–104, 2004.
16. An example of a beneficial mutation in livestock would be the one causing the polled condition in Dorsets. When sheep are raised in small areas, the loss of horns reduces stress on both the sheep and those who raise them. Lightner, J., The Riddle, *Creation* (in press).
17. Bergman, J., Ancon sheep: just another loss mutation, *TJ* **17**(1):18–19, 2003.
18. Muscular cattle: a beneficial mutation? <www.answersingenesis.org/creation/v20/i4/news.asp#cattle>.
19. Examples in cattle include photosensitivity (protoporphyria), weaver calf (progressive bovine myeloencephalopathy), and marble bone (osteopetrosis). Each of these disorders is inherited as a simple recessive trait; two defective genes are present when the trait is seen. The second and third examples are lethal. Many more examples exist. Schalles, R.R., Leipold, H.W. and McCraw, R.L., *Beef Cattle Handbook: Congenital Defects in Cattle;* <www.iowabeefcenter.org/pdfs/bch/01900.pdf>, 16 February 2005.
20. Romans 8:20, 21.
21. For example, the rest of Romans.