PRE- AND POST-NATAL DEATH OF LIVESTOCK RESULTING FROM ACTION OF LETHAL GENES – A REVIEW

Abdulyekeen Olanrewaju ABDULRAHEEM[1], Hajja Ashe MODU-KAGU[1], Haleema ABDULAZEEZ[1], Musa LAMIDO[2] and Hauwa ALLAMIN[1]

[1]Department of Animal Science, Faculty of Agriculture, University of Maiduguri, P.M.B. 1069, Maiduguri, Borno State, Nigeria,

[2]Department of Animal Science, Federal Universit Dutsin-Ma, Katsina State,

ABSTRACT

There is a great concern to examine the loss of livestock through pre- and post-natal death as well as anomalies that interfere with growth and production. One of the causes of these losses is the effect of lethal genes. Lethal genes and their effects on the performance of livestock species were reviewed in this work. Most of these anomalies were found to be controlled by single recessive genes while only a few recognized to be geared by single dominants. Physical identification of the heterozygote is difficult in the flock except in a few cases. What worsens the situation is some environmental factors that were found to produce phenotypes similar to genetic deformity described. However, genetic testing can confirm the actual state of the case. Elimination of lethal genes depends on the type of genes present; heterozygote can be diagnosed by blood typing or enzymes estimating, X-rays of neonates can also facilitate early diagnoses in defects where skeletal abnormality is present and a few conditions such as imperforated anus, surgical operation is the remedy. Although, several defects do not have genetic test, with advancement in molecular technology, genetic defects in farm animals can be effectively managed as DNA samples can be taken from an afflicted animal for diagnosis.

Key words: gene, lethal gene, dominant, heterozygote, autosomal.

INTRODUCTION

The segments of genetic material which determine one of the characteristics in living organisms and are passed from parents to their offspring in the reproductive process are called genes (Ibe, 1998). This in other words explained that fundamental function of a gene is the control of processes essentially for the organisms. The genes possessed by an individual of any species are numerous. However, only a few have been identified, many remained unknown. A gene that causes death or physical deformity of individual carrying it is said to be lethal (Dalton, 1981). Like other genes, the lethal genes can either be dominant or recessive (McClean, 2008). The gene may act while in a single dose such can exert its effect on coat colour, an example is albinism, or produce physical deformity as in Manx. The action may be in two doses that kill the animal either as still birth/embryo, shortly after birth or as the animal develops (Anthony *et al*., 2000). Other peculiar feature of lethal gene is that, it may be obligate (inevitable). While certain alleles are lethal in virtually all environments, some depend on the environment (Somes, 1990). More so, some genes may produce a recognizable phenotype in the heterozygote; others are fully dominant in one dose in heterozygote (Dalton, 1981). Anthony *et al*. (2000) remarked that hardly do fully recessive genes conform detectable effect in heterozygote. When heterozygotes mated, one quarter of any birth either died as embryo or died shortly after birth. Thus, the ratio expected for monohybrid self cross (1:2:1) is altered (to 1:2). Few examples of these genes are hairlessness, jaw defect, mule foot, rectal prolapsed and imperforated limbs (Dalton, 1981; Somes, 1990; Ibe, 1998; Merck, 2008). This work therefore intends to review the effects of lethal genes on the survival and performance of livestock.

Atresia animal

This condition is quite common in pigs, sheep and a time in cattle (Ibe, 1998; Merck, 2008). Affected animals may survive up to 10 days and are identified by peculiar features like depression, anorexia, colic marked abnormal abdominal distension and in most cases with no defecation. This is replaced by thick white mucus as anus failed to rupture (Merck, 2008). Surgical repair is possible in some cases. However, missing of a large segment of rectum made this impossible in other. Thus, creation of a colic fistula in the inguinal region is necessary. Atresia coli, another Atresia of alimentary tract has been recorded in horses. There is complete closure of the ascending colon at the pelvis flexure. Consequently, death of the affected animal occurs during the first few days of life. The condition appears to be geared by a single recessive gene (Radostits *et al*., 1997). Table 1 shows this condition and other genetic defects in livestock species.

Embryonic death

The gene causing this condition is lethal in homozygous genotypes as embryo died within the first two weeks of pregnancy in dairy cattle (~~Derk~~ and Steensma, 2021) as shown in Table 3. The presence of this gene in sheep has also been confirmed. Ironically, the gene has been researched to have genetic advantage in heterozygous while it increases milk production, fat and protein content in milk (Khatib, 2008; Derks and Steensma, 2021). Table 4 shows effects of lethal gene of performance of farm animal.

Familial polycythemia

In Jersey cattle, the defect has been observed to be responsible for congestion of mucosa, dyspnea, poor growth and early calf death. It appears that a single autosomal recessive gene controls this anomaly (Radostits *et al*., 1997).

Table 1: Lethal Defects in Livestock Species

Lethal gene/genetic defect	Animal affected	Reference
Embryonic death	Cattle (dairy), sheep	Derk and Steensma (2021)
Atresia ani	Pigs, sheep and cattle	Ibe (1998); Merck (2008)
Atresia coli	Horses	Radostits *et al*. (1997)
Familia polycythemia	Cattle	Radostits *et al*. (1997)
Parakeratosis of calve	Cattle (calve)	Radostits *et al*. (1997)
Manx gene/tail deformity	Cattle, pigs, cats	Anthony *et al*. (2000)
Ovine prosencephally	Sheep	Radostits *et al*. (1997)
Psedoalbinism and lethal white	Sheep, cattle and horses	Radostits *et al*. (1997); Merk (2008)
Retrovaginal constriction	Cattle	Radostits *et al*. (1997)

Table 2: Lethality and Stage of Death in Livestock Species

Gene	**Animal affected**	**Stage of death**
Atresia ani	Pig, sheep, cattle	Up to 10 days
Lethal white	Cattle, horse	2-4 days after birth
Embryonic death	Cattle	in the uterus
Ovine prosencephally	Sheep	Short mandible, blindness
Atresia coili	Cattle, horse	First few days of life
Manx	Cattle, cat	In the uterus
Baldly calves	Cattle	About 6 months of age

Sources: Bernier *et al*. (1987); Somes (1990); Radostits et al. (1997); Anthony *et al*. (2000);

Lethal trait/parakeratosis of calve

This defect is controlled by a single autosomal recessive gene which affect calves to live a normal life at birth but the signs appear at 4-8 weeks of age; there is loss of hair especially on the legs, very poor growth rate and eventually dies (Balbin, 2022). The condition is also characterized by parakeratosis around the mouth and eyes under the jaw and on the neck and legs. Thus, the name, parakeratosis of calves (Radostits *et al*., 1997).

Manx gene/tail deformity or tailless

Animals that suffer this defect include Holstein cattle, Land race, large white pigs and cat (Radostits *et al*., 1997; Anthony *et al*., 2000). It is characterized by complete absence or presence of tail but short or deformity of the appendages. In some cases, it is seen in combination with other deformities of the hindquarters such as Atresia ani and urinogenital tract abnormalities (Merck, 2008).

Ovine prosencephally

This condition has been recorded in Border Leicester sheep, more common in Australian breed. Affected lambs are mostly still born; life ones have dyspnea due to gross shortening of the nasomaxillary region creating a severely over short mandible. Constant symptoms are ~~suckling~~ interference, blindness and recumbence (Radostits *et al*., 1997).

Table 3: Lethal Gene and Physical Deformity in Farm Animal Species

Gene	Animal affected	Manifestation
Manx	Cattle	Tailless or short tail
Creeper	Domestic fowl	Short legs and wings
Shankless	Poultry	Reduction in meta carpal length
Missing maxillae	Domestic fowl	Absence or reduced maxillae
Lethal white overo	Horse	Pink/salmon skin

Source: McGibbon (1973); Crawford (1990); Somes (1990); Anthony *et al*. (2000) and Okimoto (2007)

Pseudo-albinism and lethal white

This defect is peculiar to sheep, cattle and horses (Merck, 2008) with similar features. Generally, the animals are photophobic (Radostits *et al*., 1997). Sheep suffer complete albinism; pin eyes and impaired vision in bright light. In cattle, it is more common in Angus, Brown Swiss, Holstein and Hereford breeds. It is characterized by brown coat and two-tone irises with an outer pale brown ring and inner blue one. In horses, lethal white *overo* resulted in producing an all-white foal; the offspring carrying the gene are born with pink/salmon skin, white crème hair and blue eyes (Merck, 2008). The homozygous foal may die in-utero (Table 2). However, the heterozygous survives though passes the condition to its offspring. This condition may be confused with phenotypic expression of crème dilution gene meanwhile, animal with double dilute lives a normal healthy life.

Retro-vaginal constriction

This anomaly is one of the conditions that have serious effects on farm animals' performance. It is characterized by small and hard udder and low productivity. Affected animals are also difficult to be inseminated and have difficulty in calving. Heterozygote animal may be identified by detection of collage type II in muscle biopsies (Radostits *et al*., 1997). It is controlled by a single autosomal recessive gene.

Table 4 Lethal Genes and the Effects on Animals' Performance

Gene	Affected animal	Effect on performance
Retrovaginal constriction	Cattle	Low productivity, difficulty in calving
Ovine prosencephally	Sheep	Suckling problem as a result of short mandible
Lethal trait	Cattle	Poor growth rate
Recessive white lethal	Domestic fowl	Poor hatchability

Somes (1990); Radostits *et al*. (1997) and Khatib (2008)

The way out

The heterozygote that poses great challenge can be diagnosed by blood typing or enzyme estimation and provided adequate data are available, the techniques may be of good use. X-ray of neonates may facilitate early diagnosis in defects involving skeletal abnormality. Retro-vaginal constriction heterozygote can be identified by detection of collage type II in muscle biopsies. A few conditions like Atresia ani, surgical operation may be a good remedy. With advancement in molecular technology genetic defects can be effectively managed in farm animal populations as DNA sample can be taken from an affected animal for diagnosis (Spangler and Anderson, 2011).

CONCLUSIONS

Lethality in farm animals is mostly geared by single autosomal recessive genes; a few are controlled by dominant. The former are lethal in homozygous recessive and the latter are lethal under homozygous dominant condition. Meanwhile, both cause defects under heterozygous conditions. Thus, heterozygote posed challenge to the breeders. With a few exceptions, phenotypic identification of heterozygote in a population is difficult. In few cases, environmental factors produce phenotypes similar to the genetic defects but with advancement in molecular technology, genetic defects can be effectively managed in farm animal populations thereby improving farm animal productivity.

REFERENCES

Anthony, T. F., Jeffry, H.M., David, T. S., Richard, C. L. and Williams, M. G. (2000). *An Introduction to Genetic Analysis.* 7th edn. W.H. Freeman and Company, New York, pp860.

Balbin, M.M. (2022). Common Genetic Defects in Domestic Animals. On line power point, Pp53 assessed on 08/12/2022.

Bernier, P. E., Erway, L. C., and Ballarino, J. (1987). Ataxia in Delaware chickens: a low penetrant dominant causing defective atacoma *Poultry Science* 66(1):66.

Crawford, R. D. (1990). *Poultry Breeding and Genetics* Elsevier Amsterdam.

Dalton, D. C. (1981). *An Introduction to Practical Animal Breeding***.** The English language Book Society and Canada; London Dunen.

Derk, M.F.L. and Steensma, M. (2021). Review: Balancing selection for deleterious alleles in livestock. *Frontier in Genetics*, 12: 1-13. Doi:10.3389/fgene.2021.761728

Ibe, S. N. (1998). *An Introduction to Genetics and Animal Breeding* Longman, Nigeria plc; Nigeria. pp 150.

Khatib, H. (2008). News and Feature. www.cals.wise.edu/downloads/khatib.

McClean, P.(2008). Pleiotropic effects and lethal genes. www.ndsu.edu/instruct/mcclean/plsc431/ mendel/mendelishtm

McGibbon, W. H. (1973). Crazy – a nervous disorder in Ancina chicks *Journal of Heredity* 65:125-126.

Merck (2008). Small and large intestine. www.merckvetmanual.com/mum/htm/be/20209.htm Retrieved on 24/04/2008.

Okimoto (2007). Poultry mutation submenu2.http./homeazweb.com.au/%7Ekasbalka/genetics/ mutations2-print.html. retrieved on 14/04/2008.

Radostits, O. M., Blood, D. C., and Gray, C. C. (1997). *Veterinary Medicine: A Text Book of the Diseases of Cattle, Sheep, Pigs, Goats and Horses.* 8th edn. W. B. Sannders Co. Ltd., London.

Somes, R. G., Jr. (1990). Lethal mutants in chickens. In: *Poultry Genetics and Breeding* (Ceawford, R. D. ed.), Elsevier, Amsterdam.

Spangler, M. L. and D. L. Anderson (2011). Genetic Defects in Beef Cattle. NebGuide, *Institute of Agricultural Resources.* www.pfizeranimalgenetics.com/default.aspx