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GENETIC DEFECTS OF BEEF CATTLE AND NECESSITY OF THEIR MANAGING

Konovalova E.N., Leading Researcher

Institute of Animal Husbandry named after L.K. Ernst, Podolsk, Russia

E-mail: konoval-elena@yandex.ru

ABSTRACT

One of the ways to increase profitability of beef production is development of meat cattle breeding through formation good breeding base. Creation the high value herds are impossible without attraction of the best genetic resources. However, even high value animals can carry undesirable alleles of genetic conditions and using such animals in breeding programs without control can cause serious economic losses instead expected benefit. And so it's important to managing of genetic defects avoiding their manifestation in populations. In our study, we have analyzed mechanisms of appearance and inheritance of genetic conditions and principles of their classification. We have followed the dynamics of appearance of genetic diseases in XX-XXI centuries and established that the distribution of genetic abnormalities has been associated with the development of artificial insemination approach. We also have revealed that genetic defects occur in all cattle breeds independent from productivity direction. Using the data of American Aberdeen Angus Association we have counted that the frequency of the most of common genetic defects is reduced thanks of development of DNA technologies. Moreover, we have familiarized with the approaches of the breed associations of different countries in relation to genetic abnormalities. Based on obtained data we have developed the guidelines for control of genetic defects of beef cattle to prevent their spread in populations.

KEY WORDS

Genetic defects, beef cattle, breeds, congenital abnormalities, disease, genetic anomalies, genetic conditions, heredity diseases.

The agricultural development and control of agricultural market is one of the points of the State program [1]. The most important role for the fast development of the area will belong to good breeding base due attraction the best of foreign and domestic breeding resources. In relation to quantity and quality of females and bulls, the breeding base must be respond to modern requirements of breeding of the most perspective beef breeds.

The animals of Aberdeen Angus, Hereford and Limousine breeds are especially interesting for this purpose. Thanks to the excellent acclimatization ability of animals of these breeds are well adapted to various weather conditions, possess such valuable qualities as the rapid economic and physiological maturity, good reproductive ability and a high genetic potential for meat productivity indexes [2, 3].

However, almost all breeds have been identified genetic defects, the distribution of which instead of the expected profit can cause serious economic damage [4].

The reasons for such defects are gene mutations, leading to disruption of the synthesis of vital proteins and, as a result, violation the functions of body [5]. Most of them have simple recessive model of inheritance. It means that mutation causing the defect determines of recessive allele and for appearance of the disease the animal must inherit recessive alleles from both parents. In case if the animal inherits normal dominant allele from one parent and mutant recessive allele from another one it won't show clinical signs but it's a carrier of the defect.

To date, there were developed methods for accurate diagnostics based on DNA analysis for almost all of the described defects [6].

In this regard, there is a need for such measures as the careful genetic control of breeding material, which is a possible thanks to the development of modern methods of molecular genetics.

Classification of beef cattle genetic defects. The most common genetic defects of these breeds animals classified in table 1. As shown in the table, all genetic defects are divided into two categories depending from the severity of clinical signs. Defects in class I are lethal, significantly disrupt or reduce reproductive performance. Defects in class II are not lethal or do not have a significant effect on reproduction, growth or meat productivity, however, are expressed in anomalous characteristics, are economically undesirable [7]. From table 1 it's seen that the most of the meat cattle defects impress the organs of the nervous system, musculoskeletal system and skin. Moreover, if the vast majority of skin diseases refer to the class II defects, the vast majority of nervous diseases – defects of class I, i.e. lethal. Defects of the musculoskeletal system belong to two classes, but it is likely that in the case of I class defects also affects the nervous system.

Table 1 – Classification of the genetic defects

n/n	Class	
	I	II
Nervous system	Multiplex arthrogriposis (AM)* Neuropathic hydrocefaly (NH) Alfa-mannosidosis (MA) Pulmonar hepoplasia with anasarca (PHA)	Idiopathic epilepsy (IE)
Musculoskeletal system	Osteopetrosis (OS) Tibial hemimelia (TH)	Contractual arachnodactyly (CA) Double mussling (M1) Polimelia (DD) Syndactilia (SY) Dwarfism (D2)
Skin	-	Hypotrichosis (HY) Iridis heterochromia (HI) Color dilution (DL) Protoporfiria (Proto) Oculocutaneous hypopigmentation (OH)

Note: * in parentheses is given the generally accepted abbreviation of the defect.

Dynamics of appearance of genetic defects in XX-XXI centuries. To date, most information on genetic defects in animals collected in the OMIA database (online Mendelian inheritance of animals), which contains information about genes of 189 animal species. Its analysis has shown that, of 506 genetic mutations of cows (*Bos taurus*) genetic defects recognized 465 mutations, because they lead to structural and functional changes of the body [8]. The remaining 41 mutation are the cause of only morphological changes, without disturbing the life of the animal (the formation of various blood groups, growth rate, temperature tolerance, fecundity, change in color, fur structure, minor scars on the skin, resistance or sensitivity to the hemolytic action of leukotoxin, color of fat) and therefore are not considered as defects. The analysis has also revealed that genes, mutations of which cause the birth defects are known only for 138 mutations, accounting for 27.3% of their total number. That is, the vast majority of genetic defects are in fact not studied.

Analysis of the dynamics of genetic defects has detected that in the first half of the twentieth century there were a small number of occurrences of congenital abnormalities. Most of them were isolated cases, related to deformities according to clinical signs. Such a low number of mutations could be due to the low degree of inbreeding and pedigree quality and diagnostic work, in particular, the absence of registration of such cases. Beginning from 1950 the number of genetic abnormalities increased twice. It happened probably due to the use of artificial insemination and, as a consequence, increases the degree of inbreeding in populations. 40 years later (1990) the number of anomalies has grown rapidly that could be related to the best of their diagnosis through the development of DNA methods. In 2000 the frequency of manifestation of genetic defects has decreased dramatically (2.4 times), but in the last decade it is at the same high level as in the 1990, that demonstrates that this problem is not solved (Figure 1).

Figure 1 – Dynamics of manifestations of genetic defects in the XX-XXI centuries

Analyzing the degree of manifestation of genetic defects depending on the direction of productivity (Figure 2), we have revealed that in dairy cattle the frequency of manifestation of genetic defects was almost two times more than in meat cattle (37% and 19%, respectively). Despite this fact, the largest share of genetic defects was in animals of universal direction of productivity (44%). This allows us to conclude that genetic defects of the development in dairy and beef cattle are manifested equally.

Figure 2 – The frequency of occurrence of genetic defects in cattle, depending on the direction of productivity

Analysis of frequency of the genetic defects. Presently, American Association of the Aberdeen Angus breed (AAA) has most data on the prevalence of genetic defects [9]. We have analyzed this database and found that currently (March 2017) the incidence of genetic defects ranging from 0.3 to 20.4% (table. 2). Moreover, the frequency of lethal genetic defects is relatively high. Among of the investigated animals 1.7% were carriers of osteopetrosis, 7.8% - of multiple arthrogryposis and 18.2% - of neuropathic hydrocephalus.

It is noteworthy that currently carriers of certain genetic defects (NH, CA and AM) are only females moreover with relatively high frequencies (11.3-26.2%). Bulls among carriers of these defects do not exist (Figure 3).

Table 2 – Frequency of genetic defects in 2017

Defect	n	Frequency, %
Polimelia (DD)	4645	20,4
Neuropathic hydrocephalus (NH)	648	18,2
Contractural arachnodactilia (CA)	1015	8,5
Multiple arthrogriposis (AM)	538	7,8
Double muscling (nt821)	444	4,9
Osteopetrosis (OS)	2314	1,7
Oculocutaneous hypopigmentation (OH)	5347	0,3

Figure 3 – The dynamics of the frequencies of multiple arthrogriposis, neuropathic hydrocephalus and contractural arachnodactyly depending on the sex of animals

Figure 4 – Dynamics of the frequencies of genetic defects

In addition, the analysis of the occurrence frequency of various defects from 2009 to the present has shown that due the application of DNA technology, there is a downward trend in the number of genetic defects carriers (Figure 4). If at the beginning of the

diagnostics, the frequency of many defects was more than 20%, to date it does not exceed 10%. Exceptions are developmental duplication and neuropathic hydrocephalus defects, the frequency of which remains high (with 20.4% and 18.2%, respectively).

Management strategy of genetic defects. When in the herd there are animals with signs of hereditary diseases there are various options for further action, ranging from a complete disregard of the problem and ending full culling of sick animals and their relatives. The most correct, according to most experts [4, 10], is the accurate identification of the genetic defect and identify its carriers through DNA diagnostics.

It is important to understand the principle of the genetic defects inheritance. As in most cases, the model of inheritance is simple recessive defect, in order to be born sick calf must cross two carriers of the mutation. Even such crossing the resulting offspring will have a genetic defect in only 25% of cases.

Thus, if the complete ignoring of genetic defect cases contributes to its manifestation, on the other hand, full culling of animals-carriers can delete from the herd the animals with the best production values that will affect negatively for economic growth [10].

Currently, most genetic defects, occurring in animals of the Aberdeen Angus and Hereford breeds have been investigated, and are controlled by breed associations [11-19]. These organizations were organized in some countries for successful breeding of certain breeds. In addition to providing support in the purchase or sale of animals, these organizations attract of scientific experts in various areas of animal husbandry for the development of methods of optimal content to maintain animal health and particularly of control over genetic defects. All data relating to genetic anomalies are recorded in the pedigrees and registration certificates of animals. As the study of the defect and developing of methods for its diagnosis, the Association members are offered genetic testing for their animals. The registration rules of animals-carriers of genetic diseases or having similar ancestors, clearly written, as well as rules for the using of biological material from these animals. The largest Associations of the Aberdeen Angus, Hereford and Limousine breeds of cattle are in USA, Canada and Australia. Depending on the specific geographical conditions and breed, Association prescribes in its rules the list of genetic defects which animal should be tested. Without the activity of these structures, progress in the fight against genetic defects would be impossible.

Recommendations. For the prevention of congenital genetic defects, in our opinion it is necessary to take the following measures: to create breed associations on example of the existing; within breed associations to conduct ongoing analysis of all available data on the incidents of any defects in animals to have information about how economic damage can be expected from their symptoms; to make a list of possible defects specific to the breed that can constantly monitor and, if necessary, to replenish; to create a shared database of known animal-carriers of genetic defects to be able the best use of breeding material; to develop test systems based on DNA-analysis for studies of breeding material for carriers of genetic defects; to carry out a detailed study of breed origin (pedigree) the newly imported animals, and in the presence of crossbred animals to examine for the defects common to two or more breeds; constantly to educate breeders of breeding farms, explaining the benefits of the information obtained from DNA-testing of animals for genetic anomalies, and that in the event of defect, there is no need to immediate culling of animals-carriers.

Conclusion. It is no secret that there is not any animal species free from mutations. Due to the fact, mutations occur in each generation, genetic defects can never be completely eradicated [5]. However, modern genetics techniques significantly accelerate the process of identification of animals-carriers of mutations that allows us to develop effective methods for the removal of defective genes from the breeds [20-24]. If during the appearance of the first case of dwarfism that took several years to cope with the disease, now to the solution of such problems takes only a few weeks [25]. It's safe to say that it is easier to prevent the appearance of genetic disease than later to fight it, experiencing considerable economic losses. Today, the meat industry uses modern science to solve problems, which is a good example of cooperation of science and production, and, thus, are able to find answers to important questions that appear in the meat industry.

REFERENCES

1. Agriculture development program 2013-2020. Approved by the government of the Russian Federation of July 14, 2012 № 717, p. 66.
2. Cattle Cow at first calving (firstborn) Aberdeen Angus. OAO "Rosagroleasing". Available from <https://www.rosagroleasing.ru/catalog/554866/>.
3. Limousin. Meat encyclopedia Meatinfo.ru. Available from <http://meatinfo.ru/info/show?id=217>.
4. Whitlock B.K. (2010). Heritable Birth Defects in Cattle. Applied Reproductive Strategies Conference Proceedings. Nashville, TN, pp. 146-151.
5. Petrova E. V. (1997). Fundamentals of classical genetics. Saratov, Dobrogea, 80 p.
6. Jolly R.D., Windsor P.A. Genetic diseases of cattle. Diseases of cattle in Australia. Chapter: 21, Publisher: VetLearn Foundation, Wellington, New Zealand. 2010. P.760-761
7. Amerchanov Kh.A., Zinovieva N.A (2009). Analysis of national accounting systems and genetic evaluation of Angus cattle in the USA. Guidance, pp. 16-20.
8. OMIA – on-line Mendelian inheritance in animals. Available from www.omia.org.au.
9. Genetic conditions and Factors. American Angus Association. Available from <http://www.angus.org>.
10. Beever J. (2009). Managing Genetic Abnormalities/ Range Beef Cow Symposium 2009 Casper, WY December 1.
11. Genetic defects: policy and related rules abnormalities and genetic defects American Angus association. Available from <http://www.angus.org>.
12. Genetic conditions. Angus Australia. Available from www.angusaustralia.com.au.
13. Genetic defects monitored by RAAA. Red Angus Association of America. Available from <http://redangus.org>.
14. Genetic conditions. Canadian Angus Association. Available from www.cdnangus.ca.
15. DNA testing. Hereford Australia. Available from www.herefordsaustralia.com.au.
16. AHA Rules and Regulations. American Hereford Association. Available from www.hereford.org.
17. DNA instructions. Canadian Hereford Association. Available from www.hereford.ca.
18. Managing Genetic Abnormalities. Canadian Hereford Association. Available from www.hereford.ca.
19. NALF's Policy concerning specific genetic conditions. North American Limousin Foundation. Available from www.nalf.org.
20. Beever J., Marron B.M. (2011). Screening for arthrogryposis multiplex in bovines. USA Patent № US 2011/0151440 A1.
21. Beever J. (2006). Tibial hemimelia (TH) and pulmonary hypoplasia with anasarca (PHA) what are they, where are they and how are they relevant.
22. Markey AD, JF Taylor, RD Schnabel, SD McKay, MC McClure and JE Beever. (2010). A Deletion Mutation In KRT71 Is Associated With Congenital Hypotrichosis In Hereford Cattle. Poster P552 at Plant & Animal Genomes XVIII Conference, San Diego, CA.
23. Nietfield J. (2007). Osteopetrosis in calves. Diagnostic Insights. p. 3.
24. Grobet, L.; Royo Martin, L.J.; Poncelet, D.; Pirottin, D.; Brouwers, B.; Riquet, J.; Schoeberlein, A.; Dunner, S.; Menissier, F.; Massabanda, J.; Fries, R.; Hanset, R.; Georges, M. (1997). A deletion in the myostatin gene causes double muscled phenotype in cattle. Nat. Genet.; 17, pp.71–74.
25. Cavanagh J.A., Tammen I., Windsor P.A., Bateman J.F., Savarirayan R., Nicholls F.W., Raadsma H.W. (2007). Bulldog dwarfism in Dexter cattle is caused by mutations in ACAN. Mamm Genome.; 18(11), pp. 808-14.