CONGENITAL DEFECTS OF BEEF CATTLE BREEDS AND GENERAL PRINCIPLES OF THEIR PREVENTION

Conflict of Interest

None declared.

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Abstract

The article has considered the problem of congenital defects of beef cattle. It was analyzed the dynamics of their manifestation in XX-XXI century. There were given the classification of the common genetic diseases. It was justified the necessity of the congenital defects control. There were shown approaches of genetic defects control. Based on the analysis it was proposed the strategy of the genetic defects managing to prevent their spread.

Keywords: congenital defects, genetic diseases, beef cattle, mutation, abnormalities.

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1 Introduction

Development of beef cattle industry is one of the sub-program of the State program. Good breeding base formed by engaging the best breeding resources and matching to requirements of modern meat cattle breeding is the most important step to this aim (Agriculture development program, 2012).

Most promising for this purpose seem to be animals of Aberdeen Angus, Hereford and Limousine breeds. Among their characteristics are excellent acclimatization ability and hence well adapting to different weather conditions, rapid growth, good fertility and high genetic potential for beef productive traits (Tagirov, H.H., Iskhakov, R.S., 2015).

Unfortunately, despite of the attractiveness of the import of breeding material with high breeding value, there is an obstacle. Among all breeds were revealed congenital abnormalities of development or genetic defects, the spread of which instead the expected benefit can cause serious economic losses. Cause of the genetic defects are gene mutations damaging the synthesis of vital proteins, which in turn disrupts the body functions (Petrova, E. V., 1997). Most of these mutations have simple recessive model of inheritance i.e. mutant allele causing the abnormality of the development is recessive and hence the animal appears the signs of the disease only in case of inheritance of two recessive alleles from both parents. If the animal inherits the normal dominant allele from one parent and a mutant recessive allele from another one, it doesn’t show the signs of the disease but is a carrier of a genetic defect.

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

For this reason, careful control of the domestic and foreign breeding material by modern methods of DNA diagnostics is a necessary measure to prevent the spread of genetic defects.

2 Classification of beef cattle congenital defects

Dependent from severity of clinical picture all congenital defects divided into two classes (table 1). Defects in class I are lethal because they are causing the diseases significantly disrupting or reducing of the reproductive parameters. Defects in class II are not lethal. They do not have a significant effect on vital functions but are expressed in anomalous economically undesirable characteristics (Gholap, P.N. et al., 2014). As can be seen from the table 1 class I includes mainly defects that affect the nervous system. The majority of defects affecting musculoskeletal system and skin correspond to class II.
Table 1 – Classification of the congenital defects

<table>
<thead>
<tr>
<th>Class</th>
<th>Nervous system</th>
<th>Musculoskeletal system</th>
<th>Skin</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Multiplex arthrogriposis (AM)*</td>
<td>Osteopetrosis (OS)</td>
<td>Hypotrichosis (HY)</td>
</tr>
<tr>
<td></td>
<td>Neuropathic hydrocefaly (NH)</td>
<td>Tibial hemimelia (TH)</td>
<td>Iridis heterochromia (HI)</td>
</tr>
<tr>
<td></td>
<td>Alfa-mannosidosis (MA)</td>
<td>Contractual arachnodactyly (CA)</td>
<td>Color dilution (DL)</td>
</tr>
<tr>
<td></td>
<td>Pulmonar hepoplasia with anasarca (PHA)</td>
<td>Double musling (M1)</td>
<td>Protoporpiria (Proto)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Polimelia (DD)</td>
<td>Oculocutaneous hypopigmentation (OH)</td>
</tr>
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<td></td>
<td></td>
<td>Syndactilia (SY)</td>
<td></td>
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<td></td>
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<td>Dwarfism (D2)</td>
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</tbody>
</table>

Note. * generally accepted abbreviation of the disease.

3 Manifestation of congenital diseases in XX-XXI centuries and analysis of their frequency of occurrence in 2009-2017

The greatest amount of the animal genetic information particularly genetic defects located in the OMIA database (Online Mendelian inheritance of animals). We have analyzed the base and revealed that of 506 genetic mutations cows (Bos taurus) most (91.9%) are the cause of genetic defects, leading to structural and functional changes of the body (http://omia.angis.org.au). The rest 8.1% of mutations are the causes of only morphological changes, without disturbing of the animal vital functions (the formation of different blood groups, change in color and other) and therefore are not considered as defects. The analysis also showed that genes, mutations of which cause birth defects are known only for 27.3% of their total number. Thus, the mechanisms of appearance of the majority of congenital genetic defects are still unknown (Fig. 1).

Analysis of the dynamics of genetic defects have shown that in the first half of the XX century there has been a small number of occurrence of congenital abnormalities. This fact can be explained that most of them were separate cases of unknown genesis, related to deformities according only to clinical signs. With beginning of the using of artificial insemination approach (from 1950), the number of genetic disorders abnormalities has increased, probably, due increasing of the degree of inbreeding in populations (Johnson, J. L. et al., 1985). In 1990 the number of abnormalities has grown rapidly, could be related to more accurate diagnostics through the development of DNA approaches. The frequency of manifestation of genetic defects has decreased dramatically in 2000 (38 cases compared with 92 cases in 1990s), however, from 2010 to nowadays it was again very high (91 cases) which demonstrates that the problem needs a solution (Fig. 2).
Congenital defects of beef cattle breeds and general principles of their prevention

We have also interested the spread of congenital defects among the animals of different direction of productivity and found that the frequency of genetic defects in animals of meat breeds is lower than in milk breeds almost twice (Fig. 3). However, the highest frequency of defects (44%) was in animals of double direction of productivity that allows us to assume that abnormalities of the development in dairy and meat cattle are distributed similarly.

Most data on the prevalence of congenital defects located in database of the American Angus Association (AAA). Analysis of this database (http://www.angus.org) showed that the incidence of the genetic defects in March 2017 amounted to 0.3-20.4% (table 2). The frequency of lethal genetic defects of neuropathic encephalopathy, multiple arthrogryposis and osteopetrosis was relatively high: 18.2, 7.8 and 1.7% respectively.

Table 2 – Frequency of genetic defects in period 2009-2017

<table>
<thead>
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</tr>
</thead>
<tbody>
<tr>
<td>NH</td>
<td>648</td>
<td>25.5</td>
<td>25.7</td>
<td>23.1</td>
<td>20.7</td>
<td>17.6</td>
<td>16.5</td>
<td>17.1</td>
<td>16.4</td>
<td>18.2*</td>
</tr>
<tr>
<td>CA</td>
<td>1015</td>
<td>15.9</td>
<td>17.0</td>
<td>12.3</td>
<td>8.4</td>
<td>8.4</td>
<td>8.2</td>
<td>8.4</td>
<td>8.5*</td>
<td></td>
</tr>
<tr>
<td>AM</td>
<td>538</td>
<td>20.2</td>
<td>16.9</td>
<td>14.5</td>
<td>12.5</td>
<td>9.1</td>
<td>8.6</td>
<td>9.9</td>
<td>8.4</td>
<td>7.8*</td>
</tr>
<tr>
<td>M1</td>
<td>4645</td>
<td>10.9</td>
<td>27.3</td>
<td>13.1</td>
<td>3.2</td>
<td>6.9</td>
<td>7.1</td>
<td>4.9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DD</td>
<td>2314</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td>21.9</td>
<td>26.1</td>
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<td></td>
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<td>25.8</td>
<td>24.3</td>
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<td>20.4</td>
</tr>
</tbody>
</table>

*Note. * - at the time of the study, only females are carriers of the defect
We have also observed that at present the carriers of neuropathic hydrocephalus, contractual arachnodactyly and multiple arthrogryposis are only females. None of the bulls was carrier of these defects. (Table 2).

Analyzing table 2 we can conclude that for most of the above congenital defects, there is a tendency to reduce the frequency of their occurrence. The exceptions are neuropathic hydrocephalus with a frequency that is decreased compared to 2009, but still remains quite high (18.2%) and developmental duplication (20.4%), which was discovered only in 2013.

4 Diagnostics of hereditary defects

It’s known that abnormalities may result from genetic or environmental causes. In this regard, for the manifestation of any abnormality in the herd of cows it is necessary to determine whether the disease is of a genetic nature.

The basic signs allowing differentiating genetic disorders from disorders causing by environmental factors summarized in table 3 (Blakely, D., 1993).

Table 3 – Signs of genetic defects and diseases caused by environmental factors

<table>
<thead>
<tr>
<th>Environmental causes</th>
<th>Genetic abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. The abnormality coincided with an environmental factor and was absent upon removal of the factor.</td>
<td>1. The abnormality is more common in a group of related animals.</td>
</tr>
<tr>
<td>2. The abnormality occurred in groups of non-related individuals.</td>
<td>2. The symptoms are similar to those of an abnormality identified through test matings. Study of an animal’s chromosomes using blood samples can identify several genetic defects.</td>
</tr>
<tr>
<td>3. The symptoms are similar to those of an abnormality known to result from environmental factors.</td>
<td></td>
</tr>
</tbody>
</table>

5 The main principle of congenital defects control

According to experts (Beever, J., 2009; Spangler, M.L. et al., 2011; Whitlock, B.K., 2010), the herd animal with obvious signs of deformities from the farmer may be four choices:

1. To ignore the problem and not to worry about the risk of its occurrence in the future.
2. Completely remove the genetic source, i.e. all the animals of this line.
3. To avoid further spread of the abnormalities in the herd with a defect without the culling of the cows-carriers, use in breeding bulls-not carriers unrelated herd. Every possible way to avoid inbreeding.
4. To accurately identify a genetic defect and its carriers through DNA testing.

The first option is certainly doomed, because this attitude contributes to the spread of a genetic defect in the herd that over time has the economic losses.

The complete removal of all sick animals and relatives will probably solve the problem, but this approach is contrary to the strategy of General improvement of the breed, as the animals are carriers can have good genetics in relation to economic-useful signs.

It is important to understand the principle of the inheritance of genetic defects. As in most cases, the model of inheritance of 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.

Attracting of additional unrelated genetics is the most practical option for commercial herds, animals which are terminal crosses, i.e. not used for breeding or genetic improvement.

The fourth option is the most win-win, because accurately diagnosing a genetic defect, the farmer gets the opportunity of its control, crossing animals-carriers with the animals free of mutations and, thus, preventing the emergence and spread of genetic anomalies in the herd.

When the environment is the cause, adjustments can reduce further economic losses. However, genetic (inherited) causes are much more complex and difficult to correct (Blakely, D., 1993) and therefore it’s necessary to develop methods of genetic defects prevention.

6 Role of breed associations in genetic defect management

For successful breeding of certain breeds currently in a number of countries were organized breed Associations.

Besides the fact that they assist in the sale or purchase of animals, these organizations attract members of the scientific organizations to develop optimal methods of animal health maintaining and, in particular, prevention and fight to genetic defects.

The pedigrees and registration certificates of animals must indicate the status of the genetic defects that are established for each concrete association. In these documents have been made a note of the carrier of the disease, for example, AMF or AMC (AMF - arthrogryposis multiple free - means that the animal is not carrier of the mutation, and AMC - multiple arthrogryposis carrier - means that the animal is carrier of the mutant allele).

As gaining knowledge of the defect and the development of methods of diagnosis, the Association offers testing of animals to members of the Association.

The using of biological material from animals-carriers are also strictly regulated by Rules or Policy of the association.


7 Conclusion

It’s known the appearance of genetic defects are easier to prevent than to fight them experiencing serious economic damage.

To date, there are no species of animals free from genetic abnormalities. Since mutations occur in each generation, it’s impossible to eradicate them completely.

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.

Such measures as organization of breed associations, registration of all data relating to genetic conditions, creation
of shared database of known animal carriers of genetic defects, developing DNA test system for accuracy diagnostics of genetic abnormalities will help to prevent appearance and manifestation of genetic conditions.

Experience of the organized breed associations is a good example of that only in the cooperation of science and production it’s possible to find solutions to many problems in meat cattle breeding.

References


