SCIENTIFIC HORIZONS

Journal homepage: https://sciencehorizon.com.ua
Scientific Horizons, 23(12), 29-35

UDC 636.598.082.2
DOI: 10.48077/scihor.23(12).2020.29-35

Hereditary Burden in Poultry of Different Species of the Ukrainian Gene Pool

Viktor Khvostyk*

Institute of Animal Breeding and Genetics named after M.V. Zubets of NAAS
08321, 1 Pohrebnyak Str., Chubynske, Ukraine

Article's History:
Received: 30.09.2020
Revised: 09.11.2020
Accepted: 15.12.2020

Suggested Citation:
Khvostyk, V. (2020). Hereditary burden in poultry of different species of the Ukrainian gene pool. *Scientific Horizons*, 23(12), 29-35.

Abstract. The relevance of the study is conditioned by the need to continually conduct anatomical pathology analysis of dead embryos as an integral part of genetic monitoring of harmful mutations, which may reduce the level of genetic burden in the gene pool of poultry. The study was conducted on poultry of various species of the Ukrainian gene pool: chickens of meat and egg area of productivity, turkeys of the original family lines of the Kharkiv crossing. The purpose of the study was to determine the spectrum and frequency of hereditary genetic defects in the development of embryos in land birds of various species, to establish the level of genetic burden. The spectrum and frequency of morphological and anatomical hereditary defects of chicken and Turkey embryos were established during anatomical pathology examination of incubation waste. Visual examination of the dead embryos allowed identifying morphological abnormalities in the structure of the skeleton, as well as various disproportions of its individual parts. In chickens of subpopulations with black-striped and white plumage, among the birds of all the studied groups, the widest range of morphological abnormalities of embryo development was discovered. In meat and egg hens with golden plumage, three anomalies with the same frequency of manifestation of 33.3% were found among the examined dead embryos. Only 1 anomaly "exencephaly" was found in birds with mottled plumage. Two cases of double mutation were found in birds with silver plumage among the examined dead embryos. The level of genetic burden in the studied subpopulations of meat and egg chickens was in the range of 3.45-8.72%. In birds with white and silver plumage, this figure was higher than the maximum allowed value, therefore it is necessary to carry out selection measures to eliminate lethal genes from these populations of chickens. In turkeys of the paternal line 5 and maternal line 6 of the Kharkiv crossing, 2 morphological anomalies of embryo development were found among the examined dead-in-shell embryos. The level of genetic burden in turkeys of related forms is low – 1.60-1.89%, which does not exceed the maximum allowed value (8.0%). This indicates a low share in the heredity of the used breeders of hidden carriers of "defective" genes. At this stage, the preservation of the gene pool of birds is not threatening its further breeding

Keywords: meat and egg chickens, turkeys of the Kharkiv crossing, Ukrainian poultry gene pool, anomalies, genetic burden

*Corresponding author

INTRODUCTION

Any population of animals, including birds, contains a certain number of recessive harmful genes that arise as a result of mutational processes and are called the "genetic burden". As a consequence of its existence, abnormal individuals can be found in any batch of one-day-old birds. An even greater number and variety of them are found during the autopsy of incubation waste. According to G.K. Otriganiev et al. [1], among the "dead-in-shell" and "late dead" obvious abnormal individuals make up 3-4%. Some of the abnormal individuals die at an earlier age, in the first days of incubation. The frequency of various anomalies in the early stages in some cases reaches 7-10% [2]. On average, it is estimated that 5% of embryos that die during incubation are abnormal. In addition, most deformities of embryonic aetiology are inherited as a recessive trait, and therefore their frequency increases with closely related breeding [3-5].

To prevent harmful recessive mutations from spreading in a particular group of animals, it is necessary to organise genetic control (monitoring) for the manifestation of pathology in animals [6-8]. To develop effective methods for eliminating the genetic burden from poultry populations, special importance is attached to anatomical pathology analysis, which acts as an integral part of the genetic monitoring of harmful mutations [9-10]. Thus, for example, according to the anatomical autopsy of incubation waste from eggs that were stored for a long time, the number of different degenerations is found to be higher than the standard indicators. The number of acrania and ectopia also increases [11]. It was also established that among late dead poultry from eggs with a long keeping period, the number of embryos with doubled limbs (legs and wings), double and multiple degenerations increase. Other researchers were also engaged in research aimed at studying the expression of genetic mutations in farm animals of various species [12-16].

This issue becomes particularly relevant when preserving the small genetic resources of poultry of various species since it allows controlling and taking timely measures to eliminate "harmful genes" from a limited poultry gene pool. This will make it possible to identify individuals who carry lethal and semi-lethal genes, the further use of which should be abandoned in order to reduce the genetic burden in the population and achieve a real, genetically determined increase in reproduction rates. Since there are few scientific publications on genetic monitoring for the detection of hereditary defects in the development of poultry embryos of the Ukrainian gene pool, such research is of particular importance.

The genetic burden can be mutational, balanced, and transient. Mutational burden occurs as a result of mutation of the dominant allele *A* into recessive *a*. The more often this process occurs, the more saturated the population is with the allele *a*. Selection counteracts population saturation with recessive alleles, eliminating them through homozygous genotypes *aa*, as the least adaptable. The general genetic burden is formed by the total effect of the genetic burden of individual loci [17].

According to the existing classification, it is proposed to divide genetic mutations according to the degree of their penetrance: lethal genes that cause 100% death of organisms; sublethal genes (semi-lethal) that cause the death of 50-90% of individuals; sub-vital genes that cause death in less than 10% of individuals [18]. Most lethal genes in poultry are recessive, but the scientific literature describes cases of pathological forms of both dominant and incompletely dominant nature. Lethal and semi-lethal anomalies are mainly associated with the transition to a homozygous state of mutant recessive genes. This means that the parents of abnormal animals are heterozygous carriers of these mutations [19]. The main danger for animal populations is represented by lethal mutations (lethals), which, being in a heterozygous state, can persist for many generations until they disappear due to the death of heterozygous carriers or in the case of infertility of homozygous animals with a lethal gene [20].

The purpose of the study was to identify the spectrum and frequency of hereditary genetic defects in embryo development in meat and egg chickens and turkeys in order to establish the level of genetic burden in various species of poultry populations of the Ukrainian gene pool.

MATERIALS AND METHODS

The study was conducted in 2018 in the laboratory of breeding, technology, and innovation management of the state experimental poultry breeding station of the National Academy of Agrarian Sciences. The object of the study was chickens of meat and egg productivity of the gene pool flock (subpopulation G1 – with black-striped plumage, subpopulation G2 – with white plumage, subpopulation G3 – with golden plumage, subpopulation G4 – with mottled plumage, subpopulation C – with silver plumage), turkeys of the initial lines (paternal line 5, maternal line 6) of the Kharkiv crossing of the middle type. All poultry is kept on the experimental farm "Preservation of the state gene pool of poultry" of the State Research Station of Poultry of the National Academy of Agrarian Sciences: meat and egg chickens in a cage battery of 6 heads per cage, turkeys on deep unchanged bedding of 20 heads per section. Feeding is carried out

with full-fledged mixed feed in accordance with the type and age of the bird.

The total number of dead-in-shell embryos of meat and egg chickens of five subpopulations was 365 pieces, turkeys of two lines – 231 pieces. During the productive period, the incubation eggs of chickens and turkeys are subjected to an incubation process to produce daily young animals. At the end of the incubation process of eggs of meat and egg chickens and turkeys of the initial lines 5 and 6 of the Kharkiv crossing, dead-in-shell embryos, the so-called "late dead", were selected among the egg incubation waste. Using a scalpel, the egg shell was broken and the dead-in-shell embryo was taken out with tweezers. Visual examination of the embryos identified morphological disorders in the skeletal structure, as well as various imbalances in individual body parts. Morphological and anatomical hereditary defects of chicken and turkey embryos were identified during anatomical pathology examination of incubation waste in accordance with a known method [21]. The description of identified anomalies of embryo development was carried out in accordance with the classification of R. Soames [5]. The level of genetic burden in each group of birds studied was determined as the proportion of detected anomalies in the development of embryos to the total number of them studied.

RESULTS AND DISCUSSION

The spectrum and frequency of hereditary genetic defects in embryo development in chickens of the studied groups are shown in Table 1.

In chickens of subpopulations G1 and G2, among birds of all the studied groups, a wide range of morphological anomalies of embryo development were found. The triple anomaly "exencephaly + crossed beak + 4 legs" was found with the predominant frequency in black-striped chickens among the examined embryos (40.0%). At the same time, white chickens most often had the "Donald Duck" anomaly (with a frequency of 53.8% among other detected defects). With a high frequency (20.0-30.8%), this bird had such mutations as "exencephaly", "crossed beak", "shortened maxilla". Other detected anomalies in meat and egg chickens of the G2 subpopulation had the same low frequency of occurrence – 7.1%. The level of genetic burden in chickens of the G1 subpopulation was set at 6.85% (Fig. 1), not exceeding the maximum allowed value (8.0%). At the same time, in white chickens, the level of genetic burden (8.72%) was higher than this indicator and therefore requires attention in terms of taking breeding and genetic measures to identify and eliminate "defective" genes carriers from the breeding process.

***Table 1**. Frequency of occurrence (%) and spectrum of developmental anomalies of embryos of meat and egg chicken populations*

Anomalies	Group Code				
	G1	G2	G3	G4	C
"Exencephaly"	20.0	30.8	33.3	100.0	0.0
"Crossed beak"	20.0	0.0	0.0	0.0	0.0
"Shortened maxilla"	20.0	0.0	0.0	0.0	33.3
"Donald Duck"	0.0	53.8	0.0	0.0	0.0
4 legs	0.0	7.7	33.3	0.0	0.0
"Exencephaly + shortened maxilla"	0.0	0.0	0.0	0.0	66.7
"Exencephaly + crossed beak"	0.0	7.7	33.3	0.0	0.0
"Exencephaly + crossed beak + 4 legs"	40.0	0.0	0.0	0.0	0.0
Number of examined dead-in-shell embryos, pcs.	73	149	50	58	35
The number of anomalies, pcs.	5	13	3	2	3
Level of genetic burden, %	6.85	8.72	6.00	3.45	8.57

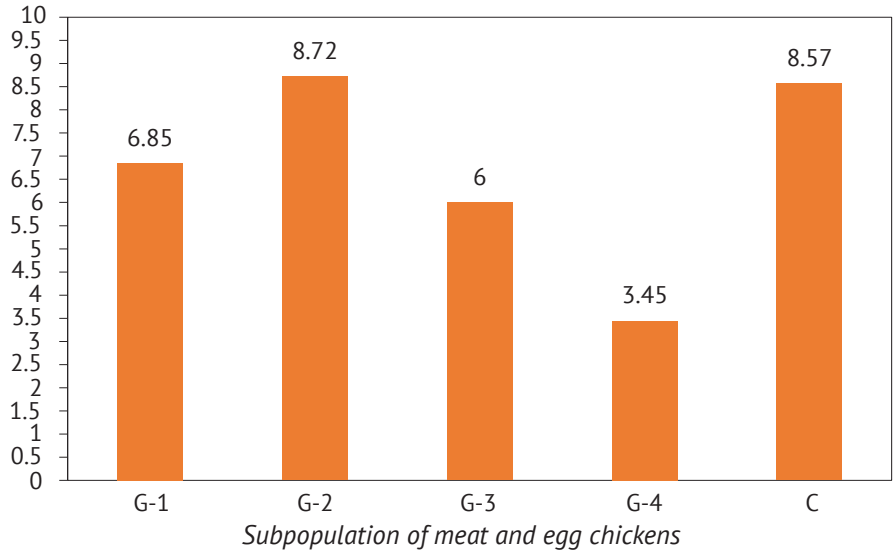

***Figure 1**. The level of genetic burden in the studied groups of chickens, %*

In chickens of the G3 subpopulation, three anomalies were found among the examined dead embryos ("exencephaly", 4 legs, "exencephaly + crossed beak") with the same frequency of manifestation of 33.3%. The level of genetic burden in this bird is low – 6.00%. Only 1 anomaly "exencephaly" was found in birds with mottled plumage. The level of genetic burden is minimal among the studied groups of meat and egg chickens – 3.45%.

In meat and egg chickens with silver plumage, two cases of the double mutation "exencephaly + shortened maxilla" and one "shortened maxilla" were found among the examined dead embryos. The frequency of manifestation of the first mutation was 66.7%, the second – 33.3%. The level of genetic burden in chickens of this group reached 8.57%, which is considered the maximum allowed, above which it is necessary to carry out breeding measures to eliminate lethal genes from the chicken population. It is worth noting that in chickens of different subpopulations, similarity in the spectrum of manifestation of hereditary embryonic developmental anomalies was noted. Thus, "exencephaly" was detected in chickens of all the studied populations, except for subpopulation C. The frequency of occurrence of this mutation is quite high – 20.0-100.0%. Embryos with a shortened maxilla were found in chickens with black-striped and silver plumage with a 20.0-33.3% frequency. Four legs were found in chickens of subpopulations G2 and G3 with a frequency of manifestation of 7.7-33.3%. The double mutation "exencephaly + crossed beak" was detected in birds with white and golden plumage with a frequency of 7.7-33.3%. The spectrum and frequency of hereditary genetic defects in embryo development in turkeys of the initial lines of the Kharkiv crossing are shown in Table 2.

***Table 2**. Frequency of occurrence (%) and spectrum of developmental anomalies of embryos in turkeys*

Anomalies	Line	
	Paternal 5	Maternal 6
"No beak"	50.0	–
"Giant eyes"	50.0	–
"Eye reduction"	–	50.0
"Exencephaly"	–	50.0
Number of examined dead-in-shell embryos, pcs.	106	125
The number of anomalies, pcs.	2	2
Level of genetic burden, %	1.89	1.60

In turkeys of the paternal line 5, among the examined dead-in-shell embryos, 2 morphological anomalies of embryo development were found – "no beak" and "giant eyes". These anomalies occurred with the same frequency – 50.0% each. The level of genetic burden in turkeys of the initial paternal line of the Kharkiv crossing is low – it is set at 1.89% (Fig. 2), not exceeding the maximum allowed value (8.0%). This does not pose a threat to this bird population. In turkeys of maternal line 6, among the examined dead-in-shell embryos with the same frequency of occurrence (50.0% each), 2 hereditary malformations were detected – "eye reduction" and "exencephaly". The level of genetic burden is generally insignificant at 1.60%.

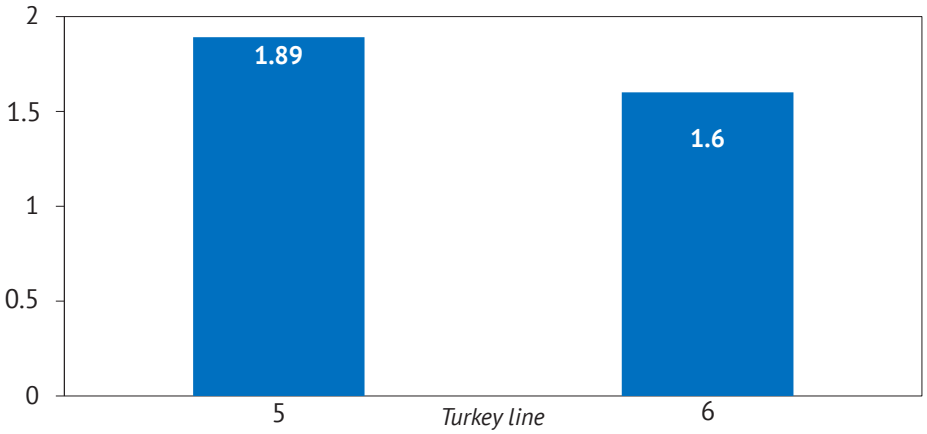

***Figure 2**. The level of genetic burden in the studied turkey lines, %*

Consequently, the established low level of genetic burden (1.60-1.89%) in turkeys of the initial related forms of the Ukrainian Kharkiv crossing indicates a low share in the heredity of the used breeders of hidden carriers of "defective" genes. At this stage, the preservation of the gene pool of birds is not threatening its further breeding. From the obtained experimental data, it can be concluded that breeding turkeys of each line "in itself" does not contribute to the accumulation and consolidation of "harmful genes" in their gene pool.

CONCLUSIONS

1. The widest range of morphological anomalies of embryo development was found in meat and egg chickens of subpopulations G1 and G2. The triple anomaly "exencephaly + crossed beak + 4 legs" was found with the predominant frequency in black-striped chickens (40.0%). White chickens most often had the "Donald Duck" anomaly (with a frequency of 53.8% among other detected defects).

2. In chickens of the G3 subpopulation, three anomalies were found among the examined dead embryos ("exencephaly", 4 legs, "exencephaly + crossed beak") with the same frequency of manifestation of 33.3%. Only 1 anomaly "exencephaly" was found in birds with mottled plumage. In meat and egg chickens with silver plumage, two cases of the double mutation "exencephaly + shortened maxilla" and one "shortened maxilla" were found among the examined dead embryos.

3. Among the studied subpopulations of meat and egg chickens of the Ukrainian gene pool, the lowest level of genetic burden (3.45%) was determined in the group of chickens with mottled plumage, while the highest (8.72%) was identified in white chickens.

4. In turkeys of the paternal line 5, among the examined dead-in-shell embryos, 2 morphological anomalies of embryo development were found – "no beak" and "giant eyes". Turkeys of maternal line 6 also had 2 hereditary malformations – "eye reduction" and "exencephaly".

5. In Turkeys of the initial Kharkiv crossing lines, the level of hereditary burden is low (1.60-1.89%), which is not of threatening importance for breeding poultry in closed populations.

REFERENCES

[1] Otryganiev, G.K., Bessarabov, B.F., & Isaev, Yu.V. (1981). *Diseases of bird embryos*. Moscow: Rosselkhozizdat.
[2] Otryganiev, G.K. (1976). Embryonic deformities and lethal genes. *Poultry*, 12, 24-25.
[3] Auerbach, S. (1979). *Problems of mutagenesis*. Moscow: Mir.
[4] Orlov, M.V. (1987). *Biological control in incubation*. Moscow: Rosselkhozizdat.
[5] Somes, R.G.Jr. (1990). Lethal mutant traits in chickens. *Poultry Breeding and Genetics*, 11, 293-316.
[6] Khvostyk, V.P., & Bondarenko, Y. V. (2016). Hereditary burden in populations of chickens of the domestic gene pool. *Bulletin of SNAU. Series "Livestock"*, 7(30), 112-114.

[7] Bondarenko, Yu.V., Tkachik, T.E., & Kutnyuk, P.I. (2005). Genetic load in land poultry populations. *Poultry*, 57, 94-98.
[8] Bulchenko, I.O. (2012). Subvital mutations of poultry. *Bulletin of SNAU. Series "Livestock"*, 12(21), 93-96.
[9] Prokudina, N.A. (2008). Analysis of the causes of embryonic mortality in meat and meat-and-egg production. In *Actual problems of modern poultry farming: Materials IV Ukrainian poultry conference* (pp. 161-168). Alushta: YEFPIT.
[10] Bondarenko, Yu.V., Breslavets, V.A., Kuchmistov, V.A., Romanov, M.N., Ivanova, T.V., & Kutnyuk, P.I. (1996). Homological variation of embryonic abnormalities in poultry. In *Proceedings of the XX World's Poultry Congress: Abstracts and students' papers contributed in international youth programme* (Vol. 4, p. 94). New Delhi: World's Poultry Science Association.
[11] Prokudina, N. (2019). Long-term storage of eggs. *Our Poultry Breeding*, 2, 24-27.
[12] Derks, M.F.L., Megens, H.J., Bosse, M., Lopes, M.S., Harlizius, B., & Groenen, M.A.M. (2017). A systematic survey to identify lethal recessive variation in highly managed pig populations. *BMC Genomics*, 18, article number 858. doi: 10.1186/s12864-017-4278-1.
[13] Charlier, C., Li, W., Harland, C., Littlejohn, M., Coppieters, W., Creagh, F., Davis, S., Druet, T., Faux, P., Guillaume, F., Karim, L., Keehan, M., Kadri, N.K., Tamma, N., Spelman, R., & Georges, M. (2016). NGS-based reverse genetic screen for common embryonic lethal mutations compromising fertility in livestock. *Genome Research*, 26(10), 1333-1341. doi: 10.1101/gr.207076.116.
[14] Smeds, L., Qvarnström, A., & Ellegren, H. (2016). Direct estimate of the rate of germline mutation in a bird. *Genome Research*, 26(9), 211-218. doi: 10.1101/gr.204669.116.
[15] Adams, H.A., Sonstegard, T.S., Van Raden, P.M., Null, D.J., Van Tassell, C.P., Larkin, D.M., & Lewin, H.A. (2016). Identification of a nonsense mutation in APAF1 that is likely causal for a decrease in reproductive efficiency in Holstein dairy cattle. *Journal of Dairy Science*, 99(8), 6693-6701. doi: 10.3168jds.2015-10517.
[16] Derks, M.F.L., Megens, H.-J., Bosse, M., Visscher, J., Peeters, K., Bink, M., Vereijken, A., Gross, C., De Ridder, D., Reinders, M., & Groenen, M. (2018). A survey of functional genomic variation in domesticated chickens. *Genetics Selection Evolution*, 50, article number 17. doi: 10.1186/s12711-018-0390-1.
[17] Merkurieva, E.K., Abramova, Z.V., Bakay, A.V., & Kochish, I.I. (1991). *Genetics.* Moscow: Agropromizdat.
[18] Glazko, V.I., & Glazko, G.V. (2003). *Introduction to genetics, bioinformatics, DNA technology, gene therapy, DNA ecology, proteomics, metabolics*. Kyiv: KVITS.
[19] Kogan, Z.M. (1979). *Exterior and interior characteristics of chickens (genetics and economic importance)*. Novosibirsk: Nauka.
[20] Dubinin, N.P. (1986). *General genetics*. Moscow: Nauka.
[21] Tishenkov, A.N. (1982). *Methodical recommendations for zootechnical laboratories of poultry enterprises*. Zagorsk: All-Russian Research and Technological Institute of Poultry Breeding.

СПИСОК ВИКОРИСТАНИХ ДЖЕРЕЛ

[1] Отрыганьев Г.К., Бессарабов Б.Ф., Исаев Ю.В. Болезни эмбрионов птиц. Москва: Россельхозиздат, 1981. 136 с.
[2] Отрыганьев Г.К. Уродства эмбрионов и летальные гены. *Птицеводство*. 1976. Вып. 12. С. 24–25.
[3] Ауэрбах Ш. Проблемы мутагенеза. Москва: Мир, 1979. 304 с.
[4] Орлов М.В. Биологический контроль в инкубации. Москва: Россельхозиздат, 1987. 221 с.
[5] Somes R.G.Jr. Lethal mutant traits in chickens. *Poultry Breeding and Genetics*. 1990. Vol. 11. P. 293–316.
[6] Хвостик В.П., Бондаренко Ю.В. Спадковий тягар у популяціях курей вітчизняного генофонду. *Вісник СНАУ. Серія «Тваринництво»*. 2016. № 7(30). С. 112–114.
[7] Бондаренко Ю.В., Ткачик Т.Э., Кутнюк П.И. Генетический груз в популяциях сухопутной сельскохозяйственной птицы. *Птицеводство*. 2005. Вып. 57. С. 94–98.
[8] Бульченко І.О. Субвітальні мутації сільськогосподарської птиці. *Вісник СНАУ. Серія «Тваринництво»*. 2012. № 12(21). С. 93–96.
[9] Прокудина Н.А. Анализ причин эмбриональной смертности кур мясного и мясо-яичного направления продуктивности. *Актуальные проблемы современного птицеводства*: материалы IV Укр. конф. по птиц-ву с междунар. уч. (г. Алушта, 15–18 сен. 2008 г.). Алушта, 2008. С. 161–168.
[10] Homological variation of embryonic abnormalities in poultry / Yu.V. Bondarenko et al. *Proceedings of the XX World's Poultry Congress: Abstracts and students' papers contributed in international youth programme* (New Delhi, 2–5 Sept. 1996). New Delhi, 1996. P. 94.
[11] Прокудіна Н. Тривале зберігання яєць. *Наше птахівництво*. 2019. Вип. 2. С. 24–27.

[12] A systematic survey to identify lethal recessive variation in highly managed pig populations / M.F.L. Derks et al. *BMC Genomics*. 2017. Vol. 18. Article number 858. doi: 10.1186/s12864-017-4278-1.
[13] NGS-based reverse genetic screen for common embryonic lethal mutations compromising fertility in livestock / C. Charlier et al. *Genome Research*. 2016. Vol. 26, No. 10. P. 1333–1341. doi: 10.1101/gr.207076.116.
[14] Smeds L., Qvarnström A., Ellegren H. Direct estimate of the rate of germline mutation in a bird. *Genome Research*. 2016. Vol. 26, No. 9. P. 211–218. doi: 10.1101/gr.204669.116.
[15] Identification of a nonsense mutation in APAF1 that is likely causal for a decrease in reproductive efficiency in Holstein dairy cattle / H.A. Adams et al. *Journal of Dairy Science*. 2016. Vol. 99, No. 8. P. 6693–6701. doi: 10.3168jds.2015-10517.
[16] A survey of functional genomic variation in domesticated chickens / M.F.L. Derks et al. *Genetics Selection Evolution*. 2018. Vol. 50. Article number 17. doi: 10.1186/s12711-018-0390-1.
[17] Меркурьева Е.К., Абрамова З.В., Бакай А.В., Кочиш И.И. Генетика. Москва: Агропромиздат, 1991. 446 с.
[18] Глазко В.И., Глазко Г.В. Введение в генетику, биоинформатика, ДНК-технология, генная терапия, ДНК-экология, протеомика, метаболика. Киев: КВІЦ, 2003. 640 с.
[19] Коган З.М. Признаки экстерьера и интерьера у кур (генетика и хозяйственное значение). Новосибирск: Наука, 1979. 295 с.
[20] Дубинин Н.П. Общая генетика. Москва: Наука, 1986. 561 с.
[21] Тишенков А.Н. Методические рекомендации для зоотехнических лабораторий птицеводческих предприятий. Загорск: Всероссийский научно-исследовательский и технологический институт птицеводства, 1982. 155 с.

Спадковий тягар у сільськогосподарської птиці різних видів українського генофонду

Віктор Павлович Хвостик

Інститут розведення і генетики тварин імені М.В. Зубця НААН
08321, вул. Погребняка, 1, с. Чубинське, Україна

Анотація. Актуальність дослідження пов'язана з необхідністю постійно проводити патолого-анатомічний аналіз загиблих ембріонів як невід'ємної частини генетичного моніторингу шкідливих мутацій, завдяки чому можливе зниження рівня генетичного тягаря у генопулі сільськогосподарської птиці. Дослідження проведено на сільськогосподарській птиці різних видів українського генофонду: курях м'ясо-яєчного напряму продуктивності, індиках вихідних родинних ліній кросу Харківський. Метою роботи було визначити спектр і частоту прояву спадкових генетичних дефектів розвитку ембріонів у сухопутної птиці різних видів, встановити рівень генетичного тягаря. Спектр і частоту прояву морфологічних та анатомічних спадкових вад ембріонів курей та індиків встановлювали при патолого-анатомічному обстеженні відходів інкубації. При візуальному огляді загиблих ембріонів визначали морфологічні порушення у будові скелету, а також різні диспропорції окремих його частин. У курей субпопуляцій з чорно-смугастим і білим забарвленням оперення, серед птиці всіх досліджених груп, виявлено найширший спектр морфологічних аномалій розвитку ембріонів. У м'ясо-яєчних курей із золотистим забарвленням оперення серед обстежених загиблих ембріонів виявлено три аномалії з однаковою частотою прояву 33,3 %. У птиці з рябим забарвленням оперення виявлено лише 1 аномалію «екзенцефалія». У птиці зі сріблястим забарвленням оперення серед обстежених загиблих ембріонів виявлено два випадки подвійної мутації. Рівень генетичного тягаря у досліджених субпопуляціях м'ясо-яєчних курей знаходився у межах 3,45–8,72 %. У птиці із білим і сріблястим опереннями цей показник був вищим за максимально допустиме значення, тому необхідно здійснювати селекційні заходи щодо елімінації летальних генів з цих популяції курей. У індиків батьківської лінії 5 та материнської лінії 6 кросу Харківський серед обстежених завмерлих ембріонів виявлено по 2 морфологічні аномалії розвитку ембріонів. Рівень генетичного тягаря у індиків родинних форм невисокий – 1,60–1,89 %, що не перевищує максимально допустиме значення (8,0 %). Це свідчить про невисоку долю у спадковості використаних плідників прихованих носіїв «дефектних» генів. На цьому етапі збереження генофонду птиці не представляє загрозливого значення для її подальшого розведення

Ключові слова: м'ясо-яєчні кури, індики кросу Харківський, український генофонд птиці, аномалії, генетичний тягар