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*Chromosome Aberrations in Chicken Embryos from Families
with Developmental Abnormalities of Spine*

Aberracje chromosomowe u zarodków kurzych w rodzinach z rozwojowymi
wadami kręgosłupa

Numerous chromosome abnormalities such as haploids and haploid-euploid chimeras, diploid-tetraploid mosaics, polyploids and aneuploids are often observed in chicken embryos, particularly in the first days of their development (1, 3). Those abnormalities are the result of errors during meiosis, conception and early stages of cleavage. General level of chromosome abnormalities in chicken embryos displays high variability depending on the strain, line or selection direction and it amounts to from several to a dozen or so percent (1, 7). Karyotype analysis made in two-direction hybrids of Rhode Island Red and Sussex proved higher level of chromosome aberrations at the early embryo stages among progeny after parents with inheritance of embryo disturbances as compared to those without such inheritance (4).

The aim of the present studies was to estimate the influence of frequency of chromosome abnormalities occurring at the early embryo stages among families with the presence or the lack of developmental spine disturbances originated from groups of two-direction selection on embryo anomalies of skeleton.

MATERIAL AND METHODS

Embryos from three generations of Rhode Island Red (RIR) and Sussex (Sx) chicken lines from the farm Kossów of the Institute of Genetic and Animal Breeding of the Polish Academy of Sciences,

originated from mating within selection towards embryo abnormalities of skeleton were the research material. Hens for mating were kept in individual cages and inseminated every week. Chromosome aberrations presence (ABR) was recorded after 18-20 hrs of incubation.

Karyotype investigations were carried out using eggs incubated at 38°C for 16-18 hrs. Isolated germinal disks were then incubated at the same temperature on a medium RPMI-1640 with colchicine addition, and after 2 hrs treated with hypotonical NaCl solution (0.8%). Fixation and staining the preparations was done according to Zartman and Jaszcak's method (9). Cytogenetical analysis included 8 pairs of the largest macrochromosomes and pairs of sex chromosomes in 10-20 metaphases of each embryo.

Totally, the material consisted of 1210 early embryo stages being 25 paternal and 161 maternal progenies.

Karyotype studies were connected with groups of full families with the division into (L) without anomalies – 104 groups, and 57 groups, where cases of significant deformations or vertebra relocations within thoracic or lumbar fragment of spine occurred (H). Due to the fact that there were no differences of chromosome aberration frequencies between RIR and Sx chicken embryos, the data obtained were analyzed together.

Numerical data were tested using χ^2 criterion with Yates' correction for small population in a sample.

RESULTS AND DISCUSSION

Cases of chromosome abnormalities occurrence among embryos under study were of slight frequency (3.6%) (Table 1). General percentage of embryos with chromosome aberrations in particular generations was close (from 3.2% to 4.1%) and it did not differ from that in laying hens (1, 2, 3). However, the percentage of embryos with aberrations in families where spine abnormalities were not found amounted to about 2.5% and it was lower than such embryos contribution in families with spine anomalies in every following generation (statistically significant in the last generation – 6.9%).

Table 1. Number and frequency (%) of chromosome abnormalities (ABR) among families without (L) and with anomalies of spine (H)

Years (generation)	1995		1996		1997		Total	
Families	ABR	N	ABR	N	ABR	N	ABR	N
L	6 (2,4%)	252	6 (2,3%)	261	6 (2,5%)	244	18 (2,4%)	757
H	10 (5,1%)	197	6 (5,4%)	112	10 * (6,9%)	144	26 * (5,7%)	453
Total	16 (3,6%)	449	12 (3,2%)	373	16 (4,1%)	388	44 (3,6%)	1210

*P ≤ 0.05.

Table 2. Chromosome composition of abnormalities in early embryos in L and H families

Families	L	H	Total
Aberration			
Haploid 1A	Z (3)	Z (5)	8
Haploid-euploid 1A/2A	Z/ZZ (4); Z/ZW (5)	Z/ZZ (6); Z/ZW (4)	
1A/3A	Z/ZZW (1)	—	20
Diploid-polioid 2A/3A	—	ZW/ZZW (1) ZW/ZWW (1)	
2A/4A	ZZ/ZZZZ (3) ZW/ZZWW (2)	ZZ/ZZZZ (1) ZW/ZZWW (2)	10
Triploid 3A	—	ZZZ (1); ZZW -1 (1)	2
Tetraploid 4A	—	ZZZZ (1)	1
Pentaploid 5A	—	ZZZWW (1)	1
Aneuploid	—	ZW -1 (1)	1
Struktural	—	ZZ (t3q ⁻ ; Z ⁺) (1)	1

() – embryo number.

No matter what the family was (Table 2), among chromosome abnormalities, haploid-euploid chimeras (45.5%), haploids (18.2%) and diploid-polyploid mosaics (22.7%) were observed most often. In families (H), aberrations that were not present in families without skeleton abnormalities were about 23% abnormalities found. They included 4 polyploids, 1 aneuploid and 1 translocation.

Numerous chromosome abnormalities such as haploids and haploid-euploid chimeras, diploid-tetraploid mosaics, polyploids and aneuploids resulting from errors during meiosis, conception and early stages of cleavage are in general of lethal character in the first stage of embryos development or they are connected with developmental disturbances (1, 8). Also structural abnormalities of translocation or inversion type can disturb the right development of their owners.

Among a few works that connect the chromosome abnormalities with skeleton developmental disturbances occurrence in chickens, inversion in the second pair chromosome responsible for reducing the chicken's leg (5) and found high percentage of haploid and aneuploid cells (42.4%) in medulla of transgenic chickens with abnormal skeleton development of no-tail type (6) can be cited. Moreover, among embryos with chromosome aberrations, such anomalies as: size reduc-

tion or lack of a head, lack or limiting the eye development, vertebra deformations or duplication of embryos around one spine were observed (8).

Studies were connected with the problem of the influence of mating the fertile poultry on the initial stages of embryos development where the absence or presence of spine developmental abnormalities were found without direct estimation of developmental deformations of embryos. Results obtained can point to the opportunity for chromosome abnormalities appearance among matings leading to spine developmental anomalies. They are confirmed by researches (4) carried out using RIR and Sx line poultry estimated on the basis of the frequencies of spine deformations at the embryo stage, as well as using hybrids (RIR x Sx and Sx x RIR) within (H) and (L) groups (10.2% to 4.5%). Therefore, some per cent of the early mortality of embryos that can result from the specific mating between poultry inherited with spine developmental abnormalities, cannot be excluded.

CONCLUSIONS

1. Chromosome abnormalities among embryos under study occurred with slight frequency (3.6%) at the absence of significant differences between generations.
2. The percentage of embryos with chromosome aberrations in families where the anomalies were not found (2.4%) was significantly lower than that in families with spine abnormalities (5.7%).
3. It was found out that the following aberrations took place only in families with spine disturbances: triploids, tetraploids, pentaploids, aneuploids and translocation.

REFERENCES

1. Bloom S. E.: Chromosomal Abnormalities in Chicken (*Gallus domesticus*) Embryos: Types, Frequencies and Phenotypic Effect. *Chromosoma* 37, 309-326, 1972.
2. Fechheimer N.S., Jaap R. G.: Origins of Euploid Chimerism in Embryos of *Gallus domesticus*. *Genetica*, 52/53, 69-72, 1980.
3. Jaszcak K., Jaszcak J.: Chromosomal Abnormalities in Early Embryos and Hatchability in Reproduction Flocks of Layer and Broiler Hens. *Genetica Polonica*, 34, 287-293, 1993.
4. Jaszcak K., Pryszcz W., Zieba G.: Karyotype Analysis of Chicken Embryos in Families With High and Low Level of Skeletal Defects. *Proc. 11th Current Problems In Avian Genetics*, 63-65, Balice (Poland) 1995.
5. Langhorst L.J., Fechheimer N. S.: Shankless, a New Mutation on Chromosome 2 in the Chicken. *Heredity*, 76, 182-186, 1985.
6. Mateescu V.: Compatibility of Completely Rumpless Chick Ontogenic Development with Some Types of Genome Chromosome Abnormalities. *Proc. Worlds Poultry Congress*, 4, 5, New Delhi (India) 1996.

7. Snyder M. D., Fecheimer N. S., Jaap R. G.: Incidence and Origin of Heteroploidy Especially Haploidy in Chick Embryos from Intraline and Interline Matings. *Cytogenetics and Cell Genetics*, 48, 130-136, 1975.
8. Thorne M. H., Collins R. K., Sheldon B. L.: Chromosome Analysis of Early Embryonic Mortality in Layer and Broiler Chickens. *British Poultry Science*, 32, 711-722, 1991.
9. Zartman D. L., Jaszczyk K.: A Procedure for Chromosome Preparations from Early Chick Embryos. *Prace i Materiały Zootechniczne*, 23, 115-118, 1979.

STRESZCZENIE

Wśród 1210 wczesnych stadiów zarodkowych kur Rhode Island Red oraz Sussex, pochodzących z kojarzeń ptaków selekcjonowanych przez trzy pokolenia na zarodkowe wady kośćca, określono częstość występowania nieprawidłowości chromosomowych w rodzinach, w których nie stwierdzano (L) oraz w rodzinach z wadami kręgosłupa (H). Nieprawidłowości chromosomowe występowały z nieznaczną częstością (3,6%), przy braku istotnych różnic między pokoleniami. Udział zarodków z aberracjami chromosomowymi w rodzinach, w których nie stwierdzono wad (2,4%), był istotnie mniejszy niż w rodzinach z wadami kręgosłupa (5,7%). Stwierdzono ponadto, że następujące aberracje: triploidia, triploidia z aneuploidią, tetraploidia, pentaploidia, aneuploidia oraz translokacja, związana z przemieszczeniem części długiego odcinka chromosomu 3 pary do chromosomu Z, wystąpiły jedynie w rodzinach z wadami kręgosłupa. Nie można wykluczyć, że kojarzenia ptaków, przyczyniające się do powstawania wad rozwojowych kośćca, mogły być jedną z przyczyn powstawania niektórych nieprawidłowości chromosomowych, a tym samym mogły wpłynąć na poziom wczesnej zamieralności zarodków w rodzinach obciążonych wadami kośćca.

MATERIAL AND METHODS