CYTOGENETIC ANALYSIS OF EARLY DEAD EMBRYOS IN CHICKEN BREEDING STOCKS

ANÁLISIS CITOGENÉTICO DE EMBRIONES PRECOZMENTE MUERTOS EN LOTES DE CRÍA DE POLLOS

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Palabras clave adicionales

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SUMMARY

Chromosomal aberrations in poultry embryos leading to abortions cause variable decrease in hatchability. Occurrence of chromosomal abnormalities was investigated in the early dead chicken embryos candled out on the 5th day of incubation. Ratio of heteroploidy in the dead embryos and their accumulation was investigated in the families. Our findings suggest, that not only the frequency but also the weight of chromosome abnormalities in early wastage can be quite different between families of the same line. Earlier observations showed that certain females may produce more chromosomally abnormal embryos than others. In our investigation aberrations seemed to accumulate in families of some cocks also.

RESUMEN

Las aberraciones cromosómicas que conducen a la muerte de embriones de pollo originan pérdidas variables de incubabilidad. Se investigó la aparición de anomalías cromosómicas en embriones de pollo que habían sufrido muerte precoz y habían sido detectados mediante transiluminador en el quinto día de incubación. La ratio de heteroploidía en los embriones muertos y su acumulación fue investigada en las familias. Nuestros hallazgos sugieren que no sólo la frecuencia, sino también la importancia relativa de las anomalías cromosómicas como causa de estas pérdidas tempranas pueden ser bastante diferentes entre familias de la misma línea. Observaciones anteriores mostraban que ciertas hembras pueden producir más embriones cromosómicamente anormales que otras. En nuestra investigación, las aberraciones cromosómicas parecieron acumularse también en las familias de determinados gallos.

INTRODUCTION

Chromosomal abnormalities in avian embryos may be responsible for variable losses during hatching. These mostly numerical abnormalities are resulted from errors in meiosis, fertilization or early cleavages. Differences in their occurrence (1-14 p. cent) are known when breeds, lines (Reddy and Siegel 1977), families or individual hens (Snyder *et al.*, 1975; de la Sena *et al.*,

1992) are investigated which refers to the genetic background of this phenomenon. Consequently, elimination of hens or families with high proportion of embryonic chromosome abnormalities from breeding stocks might be of practical importance (Szalay 1989).

Since these abnormalities generally lead to early embryonic death, early candling and cytogenetic analysis of abortive embryos from registered eggs may characterise families of a breeding stock.

MATERIAL AND METHOD

Two lines (A, B) of a layer hybrid were examined with 149 and 169 families. Incubated eggs in three consecutive series were marked with a number (family or cock) and a letter (hen). On the 5th day of incubation *empty* eggs were removed with candling and early dead embryos were selected with opening the egg shell at the air chamber. Eggs were injected with Vinblastine for mitotic arrest and incubated for a further hour. Dead embryos were phenotypically classified according to Abbott and Yee (1975) and membranes were processed for cytogenetic analysis.

RESULTS AND DISCUSSION

Among 482 (A line) and 572 (B line) karyotyped dead embryos 114 (24 p. cent) and 117 (20 p. cent) showed chromosome abnormalities.

Clustering of chromosome abnormalities was observed in some families **(table I)** in number and/or proportion in the dead embryos.

In some cases the abnormalities originated from certain hens (in families 299 and 367 of the A line, and 110 from the B line). These hens produced several karyotypically normal dead embryos as well. This might be due to the lack of the abnormal cell line in the investigated sample. The type of chromosome abnormality did not seem to be characteristic for the hens except for the D hen from the family No. 367 of the A line with a tendency of producing diploid/poliploid mosaics. The interesting in this phenomenon that types of chromosome abnormalities have quite different origin: haploidy: sperm cells, triploidy: mostly suppression of formation of the second polar body, tetraploidy and poliploidy: endomitosis during very early development (Fechheimer and Jaap 1980; Fechheimer 1981). Therefore, it looks strange if a hen producing often chromosomally abnormal embryos may have all the types of aberrations in their embryos (299 C from the A line). It may suggest the frequent existence of different cell lines (including numerical chromosome alterations) at the very early development and somehow only one will eventually form the embryo in most of the cases. The lines with chromosome alterations might reach a detectable proportion in the case of disturbed normal diploid line development (simultaneous embryo malformation). It might be also supported by the fact of very high ratio of diploid/aberrant (haploid, triploid, poliploid) chimeras in chicken embryos. The predisposition for abnormal diploid cell line development can also be suggested by the higher number of karyotypically normal early dead embryos (like in case of all the mentioned hens).

Table I. *Detailed karyotype and phenotype data of selected families with clustered embryonic chromosome abnormalities.* (Datos detallados de cariotipo y fenotipo de familias seleccionadas con anomalías cromosómicas en embriones).

A line				B line			
Family	Karyotype	Hen	Embryo phenotype	Family	Karyotype	Hen	Embryo phenotype
232	haploid/diploid	H	D1	30	haploid/diploid	Q	D1
	diploid/an uploid	K	PD		haploídJdiploid	R	D1
	diploid/poliploid	D	D1		haploid/diploid	D	BWE
	diploid	D	PD	42	diploid/poliploid	N	PD
312	diploid/triploid	H	D2		diploid/poliploid	E	BWE
	diploid/poliploid	E	D3	110	haploid	K	PD
	diploid/triploid/polliploid	B	D3		triploid	T	EABN
	diploid	D	D2		diploid/poliploid	T	D3
297	haploid/diploid	M	BWE		diploid	T	PD
	tetraploíd	K	BWE		diploid	K	BWE
	diploid/tetraploid	Q	D3		diploid	T	BWE
	diploid	K	PD		diploid	T	D1
	diploid	A	D1		diploid	N	BWE
213	haploid	M	PD				
	díploid/poliploid	N	PD				
245	diploid/poliploid	D	BWE				
	diploid/tetraploid	T	D2				
274	haploid/diploid/triploid	K	PD				
	diploid/poliploid	C	BWE				
299	haploid/diploid	C	D4				
	diploid/poliploid	C	D1				
	diploid/triploid	C	BWE				
	diploid/poliploid	N	BWE				
	diploid	B	BWE				
	diploid	C	D1				
	diploid	C	D1				
367	haploid/diploid/poliploid	C	BWE				
	diploid/poliploid	D	PD				
	diploid/poliploid	D	BWE				
	diploid/poliploid	D	BWE				
	aneuploid	D	D1				
	diploid	D	D2				
	diploid	D	BWE				
	diploid	E	PD				
	diploid	D	BWE				
	diploid	D	BWE				
	diploid	C	D2				
	diploid	C	BWE				

Phenotypic categories:
PD: positive development
BWE: blastoderrn without embryo
D1-4: embryos died at the 1-4 day stages
EABN: malformed living embryo

In cases of families where different hens produced chromosomally abnormal dead embryos (like families No. 232, 312 from the A line, and 30 from the B line) the role of the cock is suspected, which has been neglected. One of them (No. 30) showed consequent occurrence of haploid/diploid chimerism in different hens, which might be characteristic for the cock (having sperm cells with extreme ability to go into mitosis?). The others did not show specific type of abnormality, which may lead speculation similar to the case of the hens (disturbed development of karyotypically normal cell line).

Therefore, besides disposition of the hens for producing certain type of chromosomal abnormalities, other factors (genetic) can also be considered in development of abnormal cell lines of early embryos.

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