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A MOSAICISM WITH KARYOTYPE DESIGNATION OF 59. X0/60, XX/ 61, XXX IN RED PIED HEIFER (Part III)

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A Red Pied heifer with a history of anestrus and the diagnosis of gonadal dysgenesis was cytogenetically investigated. The chromosome complement was found to be 59. XO/60.XX/61.XXX. Besides the finding of numerical changes in the karyotype of the investigated animal, a high per centage of structural chromosomal aberrations was observed too. Based on these results it can be concluded that the observed mosaicism originated from a failure of sister-chromatids of the X chromosome to separate during mitotic division in the early stages of embryogenesis. The finding of structural chromosomal aberrations can not be neglected because the investigated heifer arose from an area where pesticides and other agricultural and industrial pollutants were widely used.

Key words: Mosaicism, karyotype, red pied heifer, cytogenetics

INTRODUCTION

Different numerical and structural chromosome aberrations have been observed in cattle. Many such chromosome anomalies are responsible for reproductive failure. Among them, XX/XY chimerisms, trisomy X, different types of karyotype mosaicism and chromosome polymorphism, polyploids, Robertsonian translocations, tandem fusions and etc., have been frequently described (Basrur et al. 1966; Dunn et al. 1968; Basrur et al., 1969; Halnan, 1975; Dunn et al., 1979; Soldatović and Zimonjić, 1986; Soldatović et al., 1986a; 1986b; 1986c, Soldatović et al., 1989 etc.). These karyotypic changes could be the causes of various reproductive inefficiencies in heifers, too. However, the finding of a mix oploid X chromosome variant with a chromosome complement of 59 XO/60 XX/61 XXX was a very rare instance. Thus it was found in a Pinzgauer X Angus heifer by Swartz and Vogt (1983). In human females, a similar type of mixoploid (45, XO/46, XX/47 XXX) has been reported (Simpson, 1986). The consequences of this karyotypic change in humans, as well as in animals, are related with the diagnosis of gonadal dysgenesis. Under this desig-

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nation can be considered phenotypic female animals with developmental anomalies of primary or secondary sexual characteristics.

MATERIAL AND METHODS

A phenotypically normal Red Pied heifer was submitted to cytogenetic analysis due a history of anestrus. It was 22 months old. The heifer had normal external genitalia. It was well fed and weighed 424 kg. Underdeveloped internal genitalia were palpable with difficulty on rectal inspection. After the sacrifice of this heifer, small underdeveloped ovaries without follicules and no gonadal activity were observed the Fallopian tubes were like pearly ribbons.

The karyotype test employed short term cultures of lymphocytes from the peripheral blood (Moorhead et al., 1960) and monolayer cultures of fibroblasts from the spleen (Ford et al., 1956; Evans et al., 1964). A great number of metaphases was examined and the best samples were chosen for the karyotypic test (400 metaphases).

RESULTS AND DISCUSSION

The results of chromosomal analyses in the investigated heifer are given in Table 1.

Table 1.	Karvotypic	changes	of	chromosome	number	in	the	mosaic	Red	Pier	heifer	
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The state of		ber of	Numeric changes of sex-chromosomes							
Type of cells		erved phases	59	. XO	60	. XX	61. XXX			
	N°	%	N°	%	No	%	No	%		
Lymphocytes	150	100,00	30	20,00	81	54,00	39	26,00		
Fibroblasts	250	100,00	39	15,60	137	54,80	74	29,60		
Σ	400	100,00	69	17,25	218	54,50	113	28,25		

Of 400 metaphase cells analyzed (150 lymphocytes and 250 fibroblasts), 69 (17,25%) were 59, XO; 218 (53.50%) were 60, XX and 113 (28.25%) were 61, XXX.

As Swartz and Vogt (1983) suggested, such a finding of mixoploid chromosome complement may have resulted from mitotic X-chromosome non-disjunction early in embryogenesis. According to these authors that event would produce complementary cell lines of 59, XO and 61, XXX from a single 60, XX cell. The nearly equal proportion of normal vs. abnormal cell lines suggested that this mitotic X-chromosome nondisjunction accurred early in embryogenesis (Figure 1.).

In this study, monosomic cells (2n=59, X) were observed in a lower per centage (17.25%) than trisomic cells (28.25%) It may be consider ed that monosomic cells survive with greater difficulty in organisms than trisomic cells.

Twenty- eight (7.00%) of all metaphases analyzed showed structural chromosome changes (Table 2.). Those changes were classified as chromatid

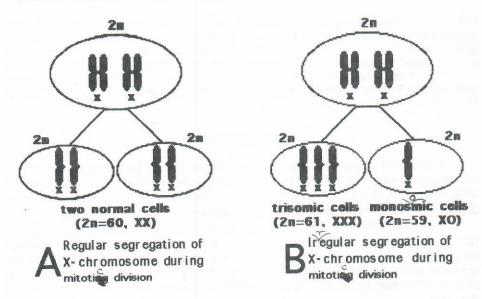


Figure 1. Regular (A) and irregular segregation (B) of sister - chromatids of the X-chromosome during mitotic division in early embriogenesis

and isochromatid breaks. Generally, they were located on the larger autosomes and the X- chromosome. The presence of structural chromosome aberrations was observed in nearly equal proportion in the three different cell lines However, cells from short term cultures of lymphocytes had a higher percentage (12.00%) of structural chromosome aberrations than cells from monolayer cultures of fibroblasts (4.00%). This can be explained by the selection effect, as well as, by the development requirements of monolayer cultures of fibroblasts. Fibroblasts take a longer period of time for their development in cultures than lymphocytes. Therefore, many aberrant fibroblasts may be unable to survive and die.

Table 2. Structural changes of chromosomes

Type of cells		of observed phases	Structural changes		
	N°	%	No	%	
Lymphocytes	150	100,00	18	12,00	
Fibroblasts	250	100,00	10	4,00	
Σ	400	100,00	28	7,00	

The finding of structural chromosome changes in 7.00% of all metaphases analyzed cannot be neglected The following facts should be considered. Firstly chromatid and isochromatid breaks and fragments can be the consequence of hormonal disorders (Jost et al., 1963; Jost et al., 1972; Swartz and Vogt, 1983). Secondly a number of different types of mutagens whose quantities in

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our environment frequently increase, may also cause chromatid and isochromatid damage. In this case, the investigated heifer came from an area where various agricultural and industrial pollutants are widely used. It could be possible that those pollutants caused structural changes of the chromosomes and hormonal disturbances which blocked numerous mechanisms and reactions responsible for their repair (Soldatović et al., 1986a; 1986b; Soldatović et al., 1989).

CONCLUSION

Based on the results obtained it can be concluded that the cytogenetically investigated heifer was mosaic with a karyotype designation of 59, X0/60, XX/61, XXX. Such karyotypic changes most likely resulted from X-chromosome non-disjunction in the early stages of embryogenesis.

It is possible that the observed structural changes of chromosomes (chromatid and isochromatid breaks) were the consequence of the permanent influence of various agricultural and industrial pollutants. At the same time, we consider that hormonal disturbances in this heifer may have caused the blockade of numerous mechanisms responsible for the repair of such chromosome damage.

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PRISUSTVO MOZAICIZMA U JUNICA RASE DOMAĆE ŠARENO GOVEČE SA KARIOTIPSKOM KARAKTERISTIKOM 2n=59, XO / 60, XX / 61, XXX

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SADRŽAJ

Junica rase domaće šareno goveče sa postavljenom dijagnozom Gonadal dysgenesis, kod koje se nije javljao estrus, podvrgnuta je citogenetičkom ispitivanju. Tom prilikom ustanovljeno je prisustvo mozaičnog kariotipa 2n=59.X0/60.XX/61.XXX. Pored numeričkih promena, u kariotipu ove junice uočen je i visok procenat strukturnih hromozomskih aberacija. Na osnovu citogenetičkih analiza može se zaključiti da otkriveni kariotipski poremećaj mozaicizam (mixoploidia) nastaje kao posledica nesposobnosti sestrinskih hromatida da se međusobno razdvoje u toku mitotičkih deoba, koje se obavljaju u ranim stadijumima embriogeneze. Nalaz strukturnih hromozomskih aberacija takođe se ne može zanemariti, pošto ispitivana junica potiče sa područja gde se nekontrolisano u velikim količinama koriste razni pesticidi, kao i drugi poljoprivredni i industrijski polutanti.