CHROMOSOMAL EVALUATION OF TWICE-OPEN HEIFERS

Evaluación cromosómica de vaquillas infecundas

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Introduction

Reproductive inefficiency is a widespread problem in all domestic animal species. Its causes are many. Aside from accidental causes these include anatomical defects of the reproductive system, disturbed endocrine function, nutritional imbalance or insufficiency, viral or bacterial infections, inherited lethal genes and chromosomal defects. The relative importance of these factors as causes of reproductive inefficiency is not yet known.

That chromosome abnormalities contribute to the problem of reproductive inefficiency is well documented. Case studies with cattle (Knudsen, 1958; Knudsen, 1961a; Knudsen, 1961b; Hansen, 1970; Popescu, 1972; Halnan, 1972; El-Nahass et al., 1974; Eldridge, 1974; Saldatovic, 1977), horses (Chandley et al., 1975; McFeely, 1975; Hughes and Trommershausen-Smith, 1977), sheep (Bruere, 1969), swine (Henricson and Backstrom, 1964; Akesson and Henricson, 1972; Locniskar, 1974; Vogt et al., 1974; Bouters et al., 1974) and humans (Fraccaro et al., 1973; Stengel-Rulkawski et al., 1976; Maeda and Ohno, 1976; Richer et al., 1977), clearly demonstrate the association between reduced fertility and chromosome abnormalities.

Few surveys have been conducted with domestic animals to determine the kinds and frequencies of chromosome defects among individuals classified as reproductively inefficient. A cytogenic survey by Gustavsson (1971) of 263 repeat-breeder heifers from Swedish herds showed that 30.8% carried a 1/29 translocation. It was also noted that daughters of sires carrying this translocation had lowered fertility. In a smaller study by Bongso and Basrur (1976) it was found that of 19 low fertility bulls in a Canadian A.I. Center, 5 (26%) were carrying chromosome defects.

Large cytogenetic surveys of men attending infertility or subfertility clinics have been conducted to assess the contribution to human infertility made by chromosomal factors (Kjessler, 1965; Kjessler, 1972; McIlree, 1966; Chandley, 1975; Faed, 1979). These surveys show that from 2.2% to 10.0% of the men attending these subfertility clinics have an abnormal chromosome pattern.

The purpose of this study was to compare the kinds and frequencies of chromosome defects in twice-open vs. contemporary fertile heifers. This information will help to clarify the extent of the contribution of chromosome defects to the overall problem of reproductive inefficiency.

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Materials and Methods

Experimental animals used in this study were located at the U.S. Meat Animal Research Center at Clay Center, Nebraska. Blood samples were collected from 71 twice-open (diagnosed not pregnant after each of the first two breeding seasons) and 71 contemporary fertile females following pregnancy determinations at the close of the 1980 and 1981 breeding seasons. Eleven breeds and various crossbred combinations were represented in this experimental group. Laboratory work and subsequent karyotype analyses were conducted at the University of Missouri in Columbia, Missouri.

Metaphase cells were obtained through 72-h cultures of heparinized whole blood. One ml of whole blood was added to 10 ml of culture media (medium 199) to which had been added 2 ml of inactivated fetal bovine serum, 0.15 ml of phytohemagglutinin (M form), 0.1 ml of penicillin-streptomycin, and 0.15 ml of sodium bicarbonate solution (7.5%). Cells were incubated at 38°C in 25-Cm^2 (surface area) tissue culture flasks. One hour before harvesting, 0.1 µg of colcemid/ml was added to each culture flask to arrest the cells at metaphase. At harvest, leukocytes were suspended in 0.075 M KCl hypotonic solution for 30 minutes. Cells were fixed in a freshly prepared 3:1 mixture of methanol and glacial acetic acid. Cells were affixed to slides by rapid air drying, stained with 10% Giemsa in buffer (pH 6.8), and mounted. Photomicrographs were taken and karyotypes subsequently constructed from 5 x 7 enlargements.

Results and Discussion

Thirteen of the 71 twice-open heifers were carrying chromosome abnormalities which likely were responsible for their failure to reproduce. Abnormalities observed include one tetraploid/diploid mosaic, five presumptive 1/29 translocations and seven involving the sex chromosomes. These are listed separately for each animal in table 1. No chromosome defects were observed among the 71 contemporary fertile females.

Table 1. Abnormal Karyotypes Observed Among 71 Twice-Open Heifers.

Animal no.	Breed type	Karyotype designation
1	Red Poll	59,XXt(1/29)*
2	Red Poll	59,XXt(1/29)*
3	Marc II	59,XXt(1/29)*
4	Simmental	59,XXt(1/29)*
5	Simmental	59,XXt(1/29)*
6	Pinzgauer	60,XXX
7	Red Poll	60,XXX
8	Limousin	60,XX/60,XY
9	Simmental	59,XO/60,XX
10	Hereford x Red Poll	59,XO/60,XX
11	Marc II	120,XXXX/60,XX
12	Pinzgauer x Angus	59,XO/60,XX/61,XXX
13	Charolais	59,XO/60,XX/61,XO

*Presumptive 1/29 translocation.

Familial relationships were noted among heifers carrying the presumptive 1/29 translocation. The two Red Poll heifers had a coefficient of relationship of 25%. The parents of one of these heifers were the grandparents of the other. The Marc II and the two Simmental heifers carrying the presumptive 1/29 translocation likewise were related. Their coefficients of relationship ranged from 0.78% to 3.125%. (Marc II animals are a composite population consisting of 1/4 Simmental, 1/4 Angus, 1/4 Hereford and 1/4 Gelbvieh.) The 1/29 translocation, then, may have been transmitted in each case from a common ancestor.

Conclusion

Findings in this study suggest that chromosome defects may be a significant cause of the reproductive inefficiency of heifers that fail to produce a calf following either of their first two breeding seasons.

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R E S U M E N

Los hallazgos del presente estudio sugieren que defectos cromosómicos puedan ser una causa significativa de la infecieiencia reproductora en novillas que fracasan en quedar gesrantes tras una de sus estaciones de reproduccion primeras.