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Detailed analysis of a new translocation in pig

NM Astachova*, LV Vysotskaya, AS Graphodatsky

Institute of Cytology and Genetics, Academy of Sciences of the USSR, Siberian Division, Novosibirsk-90, USSR

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reciprocal translocation / swine

INTRODUCTION

Today the necessity of cytogenetic control methods in farm animal breeding has become evident. Using animals with chromosome aberrations causes economic losses, much greater than those spent on chromosome analysis (Fechheimer, 1979; Gustavsson, 1980; Popescu and Tixier, 1984). Reciprocal translocations are the most widespread chromosome aberrations in domestic pigs.

MATERIALS AND METHODS

The laboratory pigs, obtained by reproductive interbreeding of Swedish *Landrace*, Vietnamese *Black* swine, Central Asian and Central European wild boars, were bred at the experimental farm in the Siberian Department of the USSR Academy of Sciences (Tikhonov and Panarina, 1980). A boar with a chromosome abnormality was identified among these animals during a routine cytogenetic study. The boar, issued from hybrid parents, was heterozygous for Robertsonian translocation 16/17.

The phenotype and sexual behavior of the boar were normal. He was used to cover 12 sows, but no offspring were obtained. Practically no sperm were observed in the seminal fluid. The absence of normal sperm in seminal canal apertures was confirmed by histological analysis of seminal tissue. Blood samples were used to prepare prometaphase chromosomes using Ikeuchi's technique (Ikeuchi, 1984) and to obtain high-resolution differential staining. In addition to seminal cell analysis, light microscope preparations of synaptonemal complexes were studied. They were identified on nitric oxide–silver-stained light microscope preparations of 'thrown flat' nuclei (Dresser and Moses, 1979).

* *Author to whom all correspondence should be addressed.*

RESULTS AND DISCUSSION

G- and C-banded karyotypes of the boar carrying the translocation are presented in figures 1 and 2. Analyzing the chromosomes of this boar, one can see an

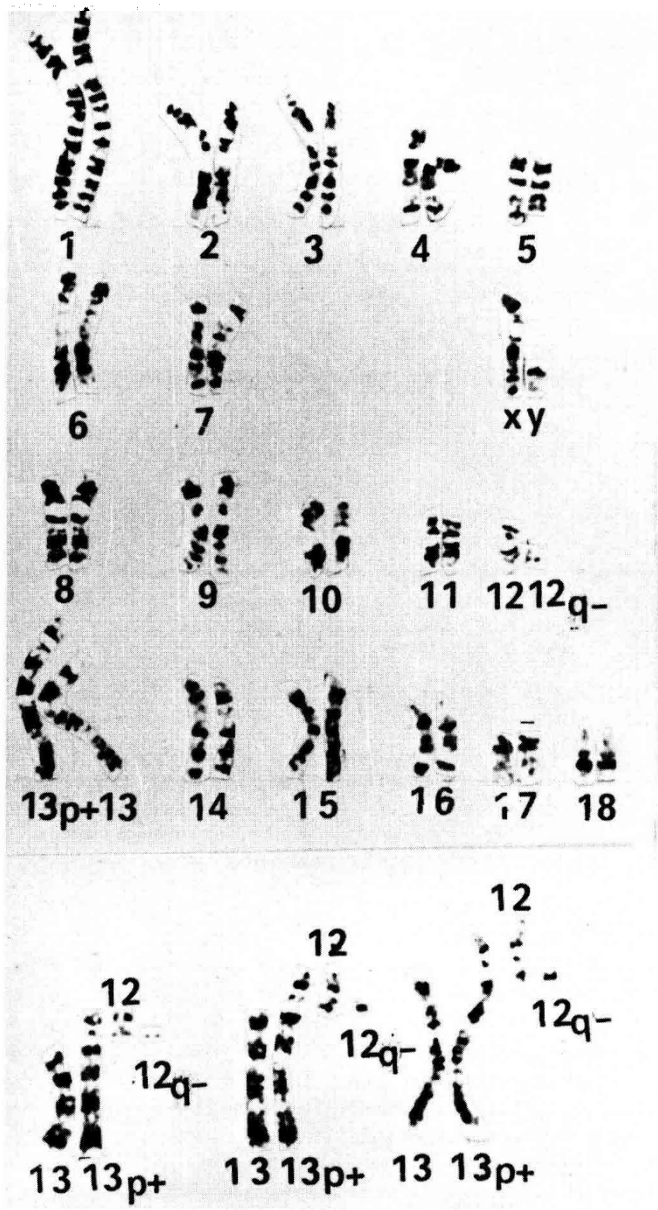

Fig 1. a. G-banded karyotype of the boar-carrier of the 12/13 translocation. **b.** G-banded chromosomes of pairs 12 and 13 with different levels of resolution.

additional arm on the largest acrocentric no 13 and a small element. Our analysis revealed that the p and, apparently, a small part of the q arm of one of the no 12 chromosomes had been translocated to one of the no 13 chromosomes. Figure 1b shows chromosomes 12 and 13 from three cells at different stages of condensation; only high-resolution banding patterns enable analysis of the identified aberration in detail. The conservation of the centromere region of the chromosome 12 that participates in the aberration was confirmed by C-banding of chromosomes (fig 2).

Usually there is a great quantity of sperm in the seminal cell preparations. Having analyzed about 200 preparations from the animal under study, we identified no more than 10 mature spermatozoa. Normal animals have many cells at the diakinesis stage. The boar carrying the translocation had only four degraded diakinetic sperm and many pachytene cells. Figure 3 shows synaptonemal complexes; the chromosome 12 and 13 quadrivalent is characteristic of reciprocal translocation (Switonski and Gustavsson, 1986).

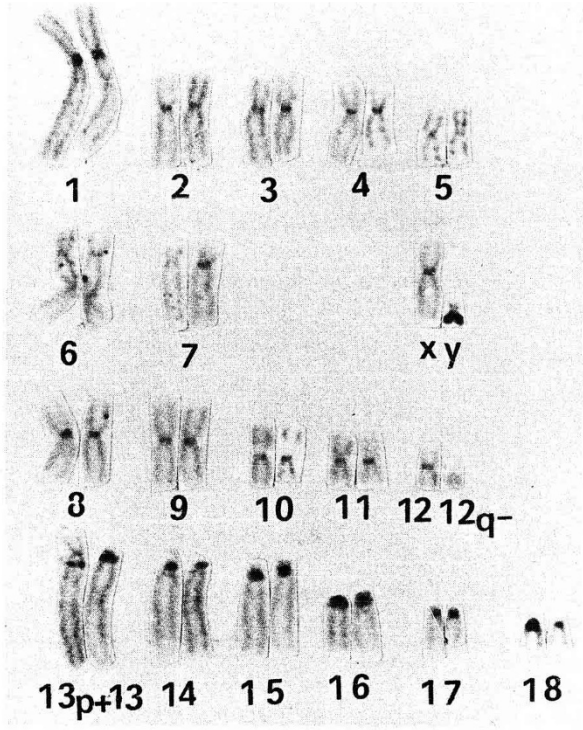

Fig 2. C-banded karyotype of the boar-carrier of the 12/13 translocation.

Today, the data concerning chromosome aberrations in domestic pigs are numerous enough, and most of these aberrations are non-Robertsonian translocations (Gustavsson *et al*, 1982; Popescu *et al*, 1984). Aberrations were mostly identified in animals with normal phenotype and normal sexual behavior, but whose number of live offspring was reduced 25–100% as a result of unbalanced gametes. Only two

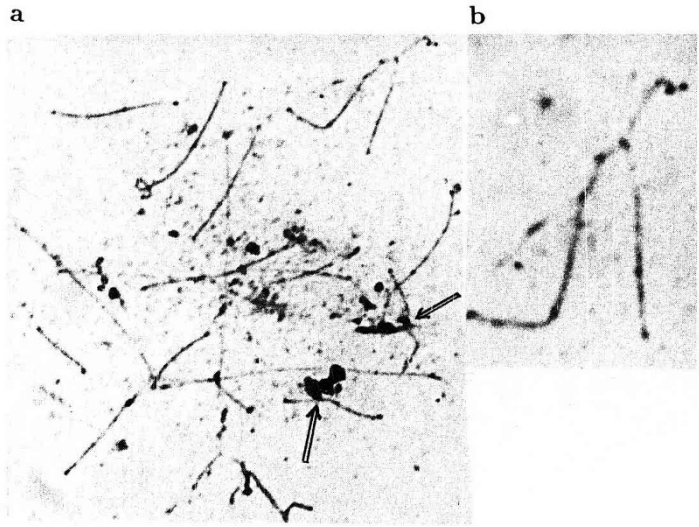

Fig 3. Synaptonemal complex of the pachytene stage. **a.** Bivalents formed by 8 and 10 nucleolar organizer region (NOR)-carrier chromosomes are shown (arrows) **b.** Quadrivalent formed by the rearranged and normal chromosomes of pairs 12 and 13.

of the translocation-carrier boars were absolutely sterile: rcp 6p+/15q– (Bouters *et al*, 1974) and rcp 12q–/13p+. In the latter case, the number of diakinetic cells and sperm was essentially reduced. It is possible that some genotype component important for normal meiosis was affected by the aberration of chromosomes 12 and 13.

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