addition, we failed to alter the genotype. *White* is believed to alter the permeability of the eye to eye pigment precursors[7].

Females heterozygous for the X linked allele *w* were mated with males containing a *Bar Stone* (B^S) allele translocation on their Y chromosome. The B^S eye phenotype is shown in figure 1. The F_1 progeny of this cross consist of phenotypically normal females and 2 types of males: white bar eye and red bar eye. The F_1 larvae were collected within ± 1 h of hatching and placed on yeast seeded cream of wheat medium[8] in 100×25 mm petri dishes. Between 50 and 55 h after hatching larvae were transferred to identical medium (control) or medium containing 3% lactamide w/w. After the adults had emerged, the petri dishes were frozen. For scanning electron microscopy (SEM) specimens were fixed in 2% glutaraldehyde, rinsed and freeze dried prior to gold coating. Photographs were taken with a Cambridge SEM at an accelerating voltage of 18 kV.

As seen in figure 2 (red B^S) and figure 3 (white B^S), lactamide treatment increased eye facet number in genetically B^S eyes to about 1/3 that of controls (fig. 4). The qualitative effects of lactamide were not altered by the presence of a *w* allele in the genotype. In addition to its effects on eyes, lactamide also affected the wings and/or legs of a majority of both males and females. In some flies, the 3rd leg was swollen and shortened showing segmentation abnormalities (fig. 5) compared to controls (fig. 6). Abnormalities were most pronounced in 3rd legs, with the proximal segments (femur and tibia) appearing more abnormal than distal segments (tarsi). Pupae unable to eclose often showed extensive abnormalities in all 3 pairs of legs. Wing abnormalities consisted of a notch in the distal region of the wing (fig. 7) as compared to controls (fig. 8). Of 71 adults treated with lactamide as larvae, 65% showed wing and/or leg defects whereas 56 untreated individuals were normal.

Our results show that lactamide is not organ-specific but may have morphological effects on both cephalic and thoracic structures. It is tempting to speculate on mechanisms of lactamide action which could account for its effect on increasing eye facet number in *B* eyes as well as producing short swollen legs and notched wings. It has been suggested[9] that the *B* phenotype results from cell death due to excessive lysosome production. It has also been shown that acetamide (an amide with morphological effects similar to lactamide) reduces the number of lysosomes in *Bar* flies[9]. If lactamide interferes with lysosome synthesis in other organs it may impede cell degradation processes which are necessary for normal morphogenesis. For example, ultrastructural evidence suggests that basal regions of the prepupal leg epithelium are broken down prior to completion of leg elongation[10]. Thus lactamide's effects on various organs may all be a consequence of its effects on lysosomes.

1 Acknowledgments. I thank M.E. Kennedy for technical assistance, S. Corff for comments on the manuscript and the personnel of the Electron Microscope Facility in the Department of Zoology, University of Toronto, for help with specimen preparation and SEM. This work was supported by NSERC (Canada).

2 A. Abd-el-Wahab, J. Genet. *56*, 475 (1959).
3 A. Abd-el-Wahab, J. Genet. *56*, 437 (1959).
4 S. Kaji, Mem. Kon. Univ., Sci. series *4*, 1 (1960).
5 C.A. Poodry and H.A. Schneiderman, Wilhelm Roux Arch. *166*, 1 (1970).
6 W.D. Seybold and D.T. Sullivan, Devl Biol. *65*, 69 (1978).
7 D.T. Sullivan and M.C. Sullivan, Biochem. Genet. *13*, 603 (1975).
8 E. Rapport and C.S. Sing, Can. J. Genet. *13*, 822 (1971).
9 M. Michinomae, Jap. J. Genet. *51*, 315 (1976).
10 E. Rapport, in preparation.

Variable positions of nucleolus organizer regions in Bovidae

B. Mayr and R. Czaker

Institut für Tierzucht und Genetik, Veterinärmedizinische Universität Wien, Linke Bahngasse 11, A-1030 Wien, and Histologisch-Embryologisches Institut der Universität Wien, Schwarzspanierstrasse 17, A-1090 Wien (Austria), 21 August 1980

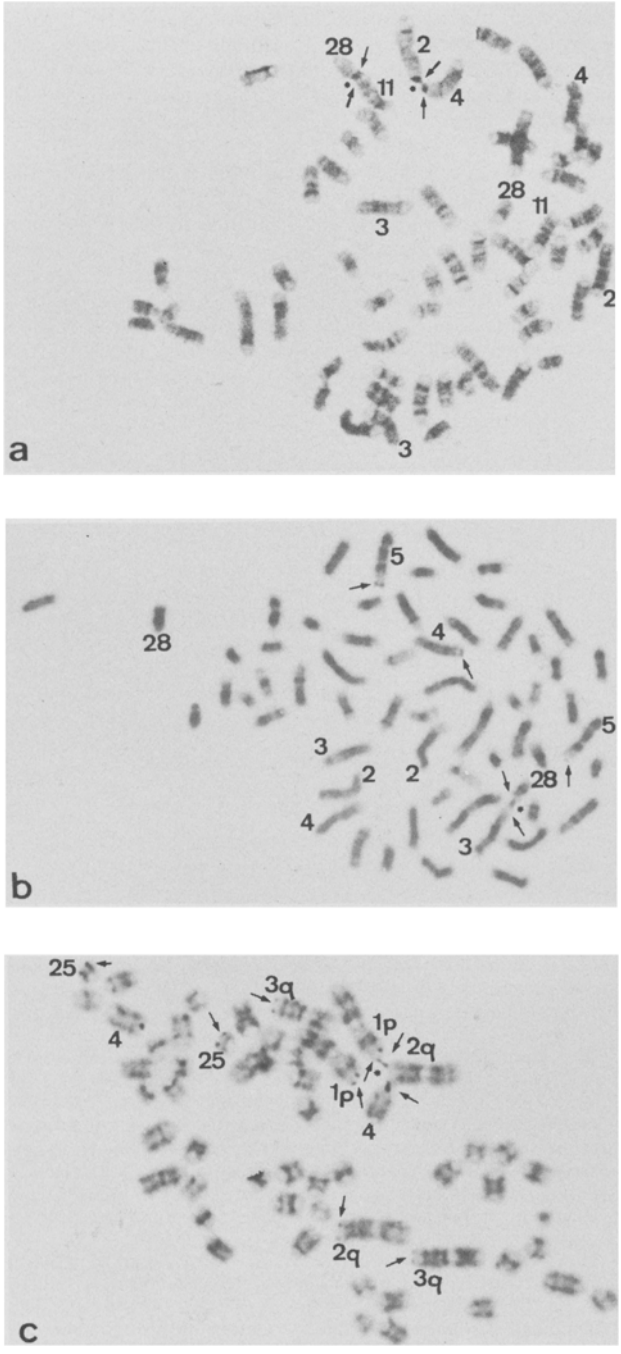
Summary. Silver NOR staining has been applied to cattle (*Bos taurus* L.), goat (*Capra hircus* L.) and sheep (*Ovis aries* L.) chromosomes. The sites of silver NORs showed variation within the family Bovidae probably due to a reciprocal translocation event.

Due to Robertsonian translocation, which for the species of the family Bovidae proves to be the primary source of interspecific karyotype evolution[1], there exists a striking constancy in the number of chromosome arms and conservation of banding pattern[2-4]. All but 2 of the autosomes in cattle were reported to show a considerable degree of homology of banding pattern with goat and sheep equivalents[2,4]. Recently in Bovidae silver staining techniques were applied for the differential staining of NORs in cattle, sheep and goats[5]. In that study it was suggested that the NORs of these species occur on chromosomes which have homologous banding patterns and the conclusion was drawn that there had been a conservation of the number and location of NORs during the evolution of these members of the family Bovidae. In the present paper variation in the sites of silver NORs within the family Bovidae is demonstrated.

Materials and methods. Chromosomes of 10 Fleckvieh cattle (5 male, 5 female), 10 Saanen goats (2 male, 8 female) and 10 Österreichische Stein sheep (4 male, 6 female) were prepared from phytohaemagglutinin stimulated lymphocytes following standard short term cultures. Slides were stained using a combined Ag-Giemsa technique[6] for demonstrating both NORs and G-bands. Chromosome identification followed the Reading-system[7].

Results. In all 3 species cattle, goat and sheep silver stained NORs appeared as conspicuous black bodies. They were situated on the telomeric regions of several chromosomes. Often much smaller silver bodies were detected at the centromeric areas of some chromosomes. In the goat

(*Capra hircus* L., 2n = 60) 5 acroncentric chromosome pairs can be recognized presenting silver stained NORs in terminal positions. These are the chromosome pairs 2,3,4,5 and 28. In the complement of the domestic sheep (*Ovis aries* L., 2n = 54) NORs were apparent on chromosome arms 1p, 2q, 3q and on chromosomes 4 and 25. In cattle (*Bos taurus* L., 2n = 60) silver positive NORs are present in the chromosome pairs 2,3,4,11 and 28. The results indicate that there is no complete conservation of NOR regions during evolution of Bovidae. In cattle chromosome 11 is a silver NOR positive chromosome, while in goats chromosome 5 bears silver NORs. In the sheep the chromosome arm 3q which resembles goat and cattle chromosome 5 in G-band pattern bears NORs.



Ag-Giemsa stained metaphases of *a* cattle (*Bos taurus*, 2n = 60), *b* goat (*Capra hircus*, 2n = 60) and *c* sheep (*Ovis aries*, 2n = 54). Arrows indicate the sites of the NORs. Asterisks indicate associations between NOR-bearing chromosomes.

Discussion. The identity of Ag-As stained sites of those areas of the chromosomes coding for ribosomal RNA has been demonstrated by in situ hybridization with rRNA in various mammals[8,12]. This can also be extended to the species cattle, goat and sheep. G-banding studies revealed that each of the 23 different acrocentric autosomes of the domestic sheep was represented by an identical chromosome in the goat and that the arms of the sheep metacentric autosomes were identical matches with the remaining 6 goat acrocentrics[2,4]. A similar interspecies homology was evident for all but 2 autosomes in the ox[2,4]. According to the Reading nomenclature system[7], the acrocentric ox and goat equivalent arms of the metacentric chromosomes in the sheep are as follows: 1,3 and 1; 2,8 and 2; 3,11 and 5. The 2 pairs of autosomes which do not match in the 2 species[2,4] were karyotyped as chromosomes 9 and 14 in the Reading system[7]. However, it should be emphasized that G-banding pattern similarities do not in any way imply genetic homology. In the present study silver NORs occurred terminally on chromosomes 2,3,4,11 and 28 in cattle, 2,3,5,11 and 28 in the goat and 1p, 2q, 3p, 4 and 25 in the sheep. The variation of positions of silver NORs of chromosome 11 (cattle), 5 (goat) and 3q (sheep) indicates that the NOR sites were not completely conserved during evolution of the Bovidae. Our results suggest that the rDNA regions of chromosomes 5 and 11 in ox and goat might have been involved in a reciprocal translocation event which seems to have occurred before the 3 characteristic centric fusion events happened in domestic sheep evolution. On the other hand the possibility that the differences in distribution of the silver NORs among the 3 Bovidae subspecies could represent a variation in rRNA gene expression cannot be excluded definitively. Our findings of involvement of chromosome 11 as a NOR site in Fleckvieh cattle is in accordance with results in Holstein cattle[9] but is different from results on Friesian and Charolais cattle[5]. At present it is not clear whether the dichotomy in silver NORs of chromosomes 5 and 11 arose early or late in Bovidae evolution and if race differences still exist between cattle breeds. Similar differences in the localization of NORs have been observed between mice belonging to different subspecies[10,11] and between 5 species of primates[12].

1 D.H. Wurster and K. Benirschke, Chromosoma *25*, 152 (1968).
2 H.J. Evans, R.A. Buckland and A.T. Sumner, Chromosoma *42*, 383 (1973).
3 W. Schnedl and R. Czaker, Cytogenet. Cell Genet. *13*, 246 (1974).
4 R.A. Buckland and H.J. Evans, Cytogenet. Cell Genet. *21*, 42 (1978).
5 L.M. Henderson and A.N. Bruere, Can. J. Genet. Cytol. *21*, 1 (1979).
6 R. Czaker and B. Mayr, Experientia *36*, 625 (1980).
7 C.E. Ford, D.L. Pollock and I. Gustavsson, Hereditas *92*, 145 (1980).
8 C. Goodpasture and S.E. Bloom, Chromosoma *53*, 37 (1975).
9 D. di Berardino, F.E. Arrighi and N.M. Kieffer, J. Hered. *70*, 47 (1979).
10 H. Winking, K. Nielsen and A. Gropp, Cytogenet. Cell Genet. *26*, 158 (1980).
11 T.C. Hsu, A. Markvong and J.T. Marshall, Cytogenet. Cell Genet. *20*, 304 (1978).
12 T.C. Hsu, S.E. Spirito and M.L. Pardue, Chromosoma *53*, 25 (1975).