phoretic alleles have been considered. The samples were from natural populations of *Drosophila melanogaster* collected in September and October in various localities of Italy in different years (table). The table shows that the configuration of allelic frequencies at the *Pgm* locus is strikingly similar from locality to locality and in various years. In particular, a) all the populations examined are polymorphic for the $Pgm^{1.00}$ and $Pgm^{0.70}$ electrophoretic alleles, and b) the $Pgm^{1.00}$ allele is the most commonly found throughout the populations and in different years. Similar observations have been made by other authors in many species for various systems[9-15]. However, strikingly similar results are often interpreted from an evolutionary genetics viewpoint in a completely different way. Some authors have as a matter of fact interpreted their data as proof of the action of natural selection[9-11], others, considering the different alleles to be neutral, have argued that these polymorphisms are mainly the consequence of a process of random sampling of the alleles, if there is a certain degree of migration between neighbouring populations[12-15]. However, since in *Drosophila willistoni*[16] and *Drosophila subobscura*[12-15], chromosomal polymorphisms, unlike enzyme polymorphisms, are very different from one locality to the next, different local populations should not represent samples from 1 single panmictic population. Also populations of *Drosophila melanogaster* from different localities can be considered to be different populations, but in this case because of the differences in frequency of *SD* (segregation distorter) chromosomes, 2nd chromosomes sensitive to *SD* and 3rd chromosomes carrying modifiers of the *SD* phenomenon[18,19]. It remains to be seen whether random genetic drift alone can explain the similarity of patterns observed. That is, it is necessary to estimate the size of a population (N_e) during the bottle-neck period. This must be large enough to cover the differences in gene frequencies found from one year to the next. In this case it must be recognized that all these populations derive from a single native population, or at least that they all started out with the same gene frequency. In our calculations we have neglected the mutation rate which for electrophoretically detectable alleles has been estimated as 10^{-8} per locus per generation[14]. We have calculated the probable value of the effective breeding size N_e during the bottle-neck period in the populations examined[20]. The best estimate of N_e is about 5000. If this value is confirmed by field studies (an excellent estimation technique of the population size seems to be that of the mark-release-recapture analysis[21]), then it could be concluded with relative confidence that the PGM electrophoretic polymorphism is maintained in the natural populations studied by random genetic drift. Otherwise we could conclude that besides random genetic drift some form of selection (or co-selection due to linkage disequilibrium effect) is operating on this system.

It is worth emphasizing that a stabilizing selection would make the formal N_e appear larger than the 'true' one by buffering genetic drift. Disruptive selection would lead to an underestimation of N_e. Studies using different markers would make it possible to compare the various N_e values and evaluate the importance of genetic drift and natural selection in maintaining protein polymorphisms in natural populations.

1 I am grateful to Prof. D. Sperlich and Prof. W. Seyffert for their kind hospitality during my sojourn as Research Fellow of the Alexander von Humboldt Foundation in the Institute of Biology II at the University of Tübingen (FRG) where the final version of this paper has been done. The technical assistance of Mr A. Micheli is also acknowledged.

2 R.C. Lewontin, The Genetic Basis of Evolutionary Change. Columbia University Press, New York 1974.
3 M. Nei, Molecular Population Genetics and Evolution. American Elsevier, New York 1975.
4 H. Harris, The principles of Human Biochemical Genetics. North-Holland Publ., Amsterdam/London 1975.
5 T. Dobzhansky, F.J. Ayala, G.L. Stebbins and J.W. Valentine, Evolution. Freeman and Co., San Francisco 1977.
6 G. Trippa, C. Santolamazza and R. Scozzari, Biochem. Genet. *4*, 665 (1970).
7 G. Trippa, G.A. Danieli, R. Costa and R. Scozzari, Drosoph. Inf. Serv. *52*, 74 (1977).
8 G. Trippa, A. Loverre and A. Catamo, Nature *260*, 42 (1976).
9 F.J. Ayala, M.L. Tracey, L.G. Barr, J.F. Mc Donald and S. Perez-Salas, Genetics *77*, 343 (1974).
10 F.M. Johnson and P.M. Burrows, Biochem. Genet. *14*, 47 (1976).
11 D.A. Powers and A.R. Place, Biochem. Genet. *16*, 593 (1978).
12 C.B. Krimbas and S. Tsakas, Evolution *25*, 454 (1971).
13 W.H. Li and M. Nei, Genetics *86*, 901 (1977).
14 M. Kimura and T. Ohta, Nature *229*, 467 (1971).
15 P.A. Fuerst, R. Chakraborty and M. Nei, Genetics *86*, 455 (1977).
16 F.J. Ayala, J.R. Powell and T. Dobzhansky, Proc. natl Acad. Sci. USA *68*, 2480 (1971).
17 W. Pinsker, P. Lankinen and D. Sperlich, Genetica *48*, 207 (1978).
18 G. Trippa and A. Loverre, Genet. Res. *26*, 113 (1975).
19 G. Trippa, A. Loverre and R. Cicchetti, Genetics *95*, 399 (1980).
20 G. Trippa, R. Scozzari, G. Zei and G. Modiano, in preparation.
21 M. Begon, O. Milburn and D. Turner, J. nat. Hist. *9*, 315 (1975).

The effect of lactamide on *Drosophila*

Ellen Rapport[1]

Department of Zoology, University of Toronto, 25 Harbord Street, Toronto (Ontario, Canada M5S 1A1), 4 November 1980

Summary. Lactamide fed to 3rd instar *Drosophila* larvae induces increased eye facet number in genetically *Bar* individuals and may also produce swollen, shortened legs and notched wings in *Bar* as well as non-*Bar* flies.

The effect of lactamide and related substances on eye facet number in various mutants has been studied extensively[2-4] with particular attention given to the increase in eye facet number caused by feeding lactamide to 3rd instar larvae of the *Bar* genotype; there are no reports, however, of lactamide effects on other organs. Since cells of larval eye imaginal discs are believed to be indistinguishable from cells of other discs both ultrastructurally[5] and biochemically[6] we wished to determine whether lactamide was acting in an organ-specific manner. In this report we show that lactamide can induce wing and leg abnormalities in addition to increasing eye facet number in the *Bar* mutant. In

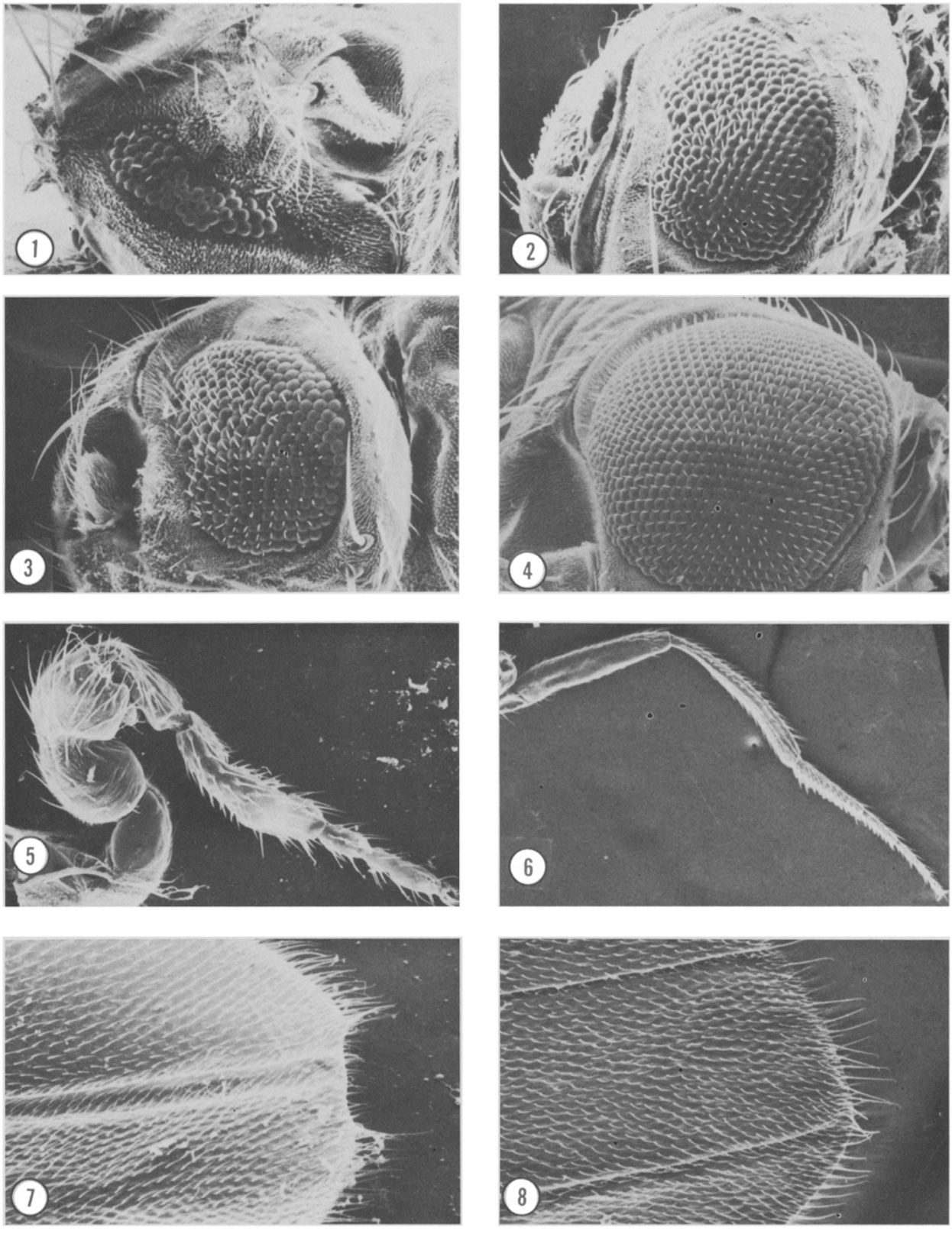

Fig. 1. B^S eye with less than 50 facets. × 164. Fig. 2. B^S eye enlarged as a result of lactamide treatment. × 164. Fig. 3. *w* B^S eye enlarged as a result of lactamide treatment. × 164. Fig. 4. Normal eye. × 164. Fig. 5. 3rd leg of a lactamide treated female showing abnormally small and swollen segments. × 146. Fig. 6. 3rd leg of an untreated female, the leg is thinner, longer and more clearly segmented than the leg in fig. 5. × 49. Fig. 7. Notched wing tip of a lactamide treated fly. × 219. Fig. 8. Normal wing tip. × 219.

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