Materials and methods. Mitosis was examined mainly in orcein stained cells from the cerebral ganglia of 3rd instar larvae, and meiosis in the developing testes of puparia. The Giemsa staining technique was identical to that used on the chromosomes of Orthopterans[2] and *Aedes aegypti*[3].

Mitosis. The somatic pairing commonly found in most Dipterans is a feature of this species, and homologous autosomes invariably emerge from interphase already associated as somatic bivalents. However, the sex chromosomes are an exception, for neither the two X chromosomes in females nor the X and Y in males ever appear intimately associated (Figure 2). As the autosomes contract during prophase the close association tends to lapse and the only point of contact is restricted to a region surrounding homologous centromeres. By metaphase even this link is lost. The remainder of mitosis is unexceptional, sister chromatids move to opposite poles at anaphase, and following cleavage of the cytoplasm to form two new cells, they despiralize to an amorphous nuclear mass.

In both sexes the diploid chromosome complement in $2n = 10 + XX/XY$, and the male karyotype is illustrated in the idiogram (Figure 1). The largest and 2 shortest autosomal pairs are almost metacentric whereas pairs 2 and 3 are noticeably submetacentric. Both the X and Y chromosomes are acrocentrics although the short arm of the Y is appreciably smaller than that of the X. In overall length the X resembles that of chromsosome 1 whereas the Y is slightly shorter than chromosome 5.

The C-banding technique was used to discover whether there are any additional cytological markers[3] which might prove useful in research involving chromosome mutations. In this respect the method proved disappointing, for similar sized segments on each side of the centromere in all the autosomes stained with equal intensity, and there were no intercalary bands (Figure 2). However, the entire short arms of the X and Y chromosomes stain, together with a substantial segment proximal to the centromere in their long arms.

Meiosis. Cells in meiosis have been identified in 3rd instar larvae, pupae and adults together with a substantial number of mitotically dividing cells. The onset of meiosis is heralded by the appearance in the nucleus of a dark staining heterochromatic mass (Figure 3) which corresponds to the precociously condensed and already associated X and Y chromosomes. Like other Dipteran species where pronounced somatic pairing is a characteristic feature there is no classical leptotene or zygotene[4–6]. The first recognizable stage is pachytene; the autosomes are closely synapsed and only in occasional cells can the duplicate nature of a chromosome be identified. Autosomal bivalents continue to contract (Figure 4) until a point is reached when the intimate pairing tends to lapse. This is particularly noticeable in procentric regions but can also be detected at other points along the bivalents (Figure 5). It may be significant that there is no diplotene stage of the type found, for example, in the *Culicini*, for these bivalents more closely resemble diakinesis or prometaphase[7]. This may well reflect an achiasmate mode of meiosis although structures have been observed which could be interpreted as cross-overs (Figures 5, 6 and 7). It seems almost certain that if there is chiasma formation on the male side then only genetical methods will provide the answer, for the bivalents are so small that normal cytological techniques are inadequate.

Throughout first prophase the heteromorphic sex bivalent remains highly condensed and in the majority of cells it is difficult to establish the pairing relationship of the X and Y. However, it is tentatively suggested that there is an end-to-end association of the long and short arms of the 2 chromosomes (Figures 5 and 6). Their behaviour at anaphase I is unpredictable for they may move to opposite poles in concert with the autosomes, they may separate precociously, or they may exhibit delay.

Following cytokinesis at the end of the first sequence meiosis, the chromosomes retire briefly into an interphase stage, and when they reappear at second prophase contract rapidly to metaphase II. Precocious condensation is again displayed by both the X and Y members during these stages (Figures 8 and 9) and is still evident at anaphase II (Figure 10). After the chromatids have arrived at the poles in telophase II the autosomes gradually despiralize to form a reticulate mass in the spermatid nuclei but both the X and Y chromosomes remain as heterochromatic bodies.

[2] A. GALLAGHER, G. M. HEWITT and I. GIBSON, Chromosoma *40*, 167 (1973).
[3] M. E. NEWTON, D. I. SOUTHERN and R. J. WOOD, Chromosoma *49*, 41 (1974).
[4] S. A. L. MESCHER and K. S. RAI, Mosquito News *26*, 45 (1966).
[5] S. M. ASMAN, J. med. Ent. *11*, 375 (1974).
[6] D. I. SOUTHERN and P. E. PELL, Chromosoma *44*, 319 (1973).
[7] D. I. SOUTHERN, T. A. CRAIG-CAMERON and P. E. PELL, Trans. R. Soc. trop. Med. Hyg. *66*, 145 (1972).

Ethanol as a 'Food' for *Drosophila melanogaster*: Influence of the *Ebony* Gene

M. LIBION-MANNAERT, J. DELCOUR[1], M. C. DELTOMBE-LIETAERT, N. LENELLE-MONTFORT and A. ELENS

Département Génétique et Physiologie Cellulaire, Facultés Universitaires N. D. de la Paix, rue de Bruxelles 61, B–5000 Namur (Belgium), 2 July 1975.

Summary. The survival time of adult *Drosophila melanogaster* flies without food is greater in the presence of ethanol, especially for flies of strains or lines with a higher alcohol dehydrogenase activity. It seems that the *ebony* gene can act in some populations as a selective factor favoring the ADH^F allele, as well as the minor genes enhancing the alcohol dehydrogenase activity level.

In adapting itself to temperate climates, *Drosophila melanogaster* seems to have modified its ecological niche in order to exploit food sources characterized by a higher alcohol concentration: European strains are much more ethanol-tolerant than African or American tropical strains[2]. Such an adaptation must be in some way related to the genes controlling the alcohol dehydrogenase isozymic pattern, as the ratio of the ADH^F to the ADH^S allelic frequencies varies in the Eastern United States according to a clinal pattern from 1 to 9 in the South to about 1 to 1 in the North[3]. Under laboratory conditions, addition of alcohol to the culture medium shifts the ADH^F allelic frequency to higher values[4]. Enzymatic activity is usually higher in ADH^F/ADH^F than in ADH^S/ADH^S

strains, but in both cases it is possible to select substrains with higher or lower ADH activity levels, indicating that ADH genes are also under the control of polygenic modifiers[5].

The present experiments show that the *ebony* gene is, in laboratory conditions at least, an internal selective factor favoring the ADH^F allele as well as the modifiers enhancing the alcohol dehydrogenase activity.

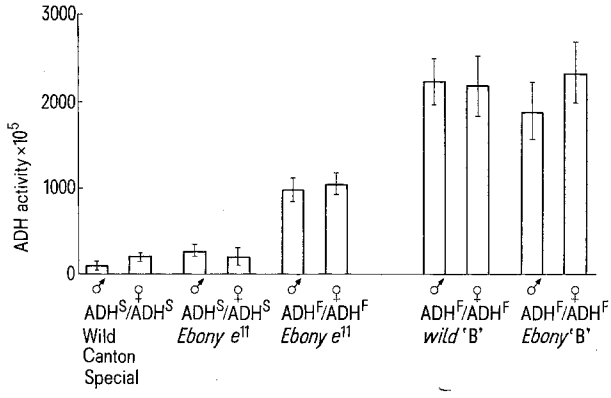

Fig. 1. Mean ADH activity of 10 day-old flies. Assays were performed according to Sofer and Ursprung[16]. Each value (95% confidence intervals) is based on 10 samples (5 flies each). Ordinate: unites of ADH activity $\times 10^5$. ADH genotypes have been determined by acrylamide electrophoresis of single fly homogenates, according to Ursprung and Leone[17].

An *ebony* e^{11} strain from our laboratory stocks has been shown to have a very high level of alcohol dehydrogenase, as compared with a Canton Special *wild* control[6]. Moreover, the survival time of adult flies kept in vials containing a medium made of agar and water is higher in the presence of ethanol, the difference being much greater for the *ebony* than for the *wild* Canton Special flies[7]. As our *ebony* e^{11} strain turned out to be polymorphic for the ADH system, it was easy to select substrains homozygous for either the ADH^S or ADH^F allele, having a lower or a higher alcohol dehydrogenase activity respectively. This suggests that the high enzymatic activity previously found in our *ebony* e^{11} strain simply reflects the high frequency of the ADH^F allele in that population (about 70%).

In order to obtain *wild* and *ebony* substrains with the same genetical background, males randomly taken from the e^{11} stock have been crossed with *wild* Canton Special

[1] Chargé de Recherches au F. N. R. S.
[2] J. David and C. Bocquet, C. r. Acad. Sci., Paris, D, *279*, 1385 (1974).
[3] C. L. Vigue and F. M. Johnson, Biochem. Genet. *9*, 213 (1973).
[4] J. Gibson, Nature, Lond. *227*, 959 (1970).
[5] R. D. Ward and P. D. N. Hebert, Nature New Biol. *236*, 243 (1972).
[6] M. Libion-Mannaert and A. Elens, *Drosophila Inform. Serv. 49*, 77 (1972).
[7] M. Libion-Mannaert, M. C. Deltombe-Lietaert and A. Elens, Abstracts 4th Eur. Drosoph. Res. Conf., Umea, Sweden (1974).

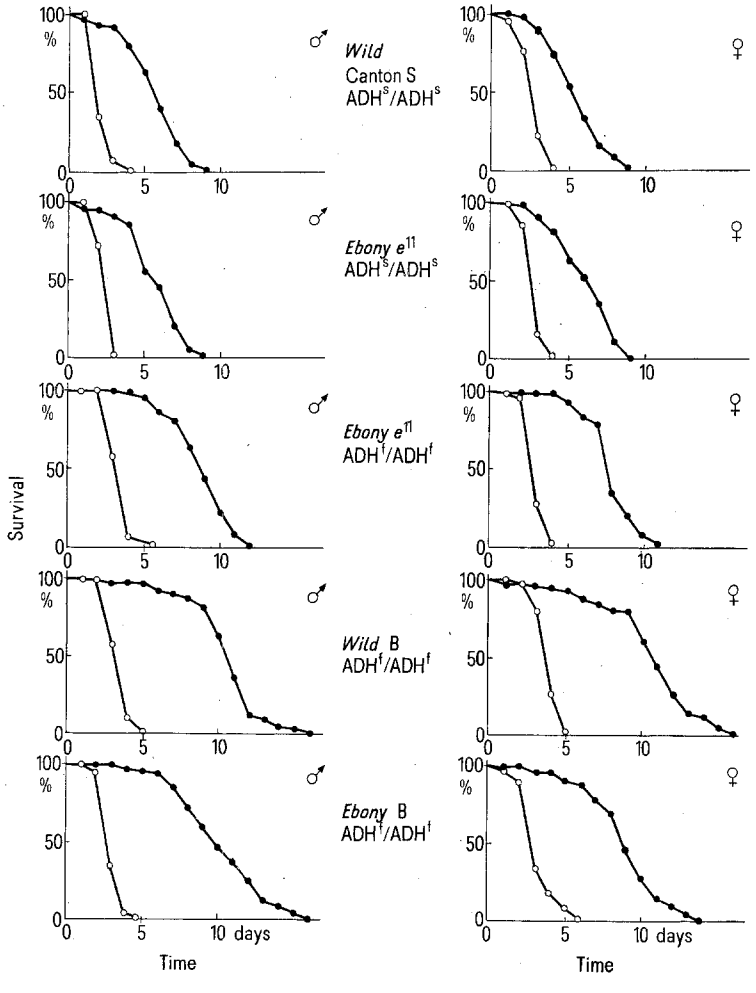

Fig. 2. Survival time (ordinate: % survival; abscissa: time in days) of starving flies in the presence of (closed symbols) or in the absence (open symbols) of ethanol. Groups of 10 individuals of the same sex (10 days old), were put in 100 cm^3 vials containing 20 g hydrated agar medium (7,5 g agar for 250 ml water) to which amphotericine had been added in order to avoid any growth of yeasts or other microorganisms. Except for the controls, 2 ml of ethanol was also added. Every day, the surviving flies were transferred to a new vial containing fresh medium. 10 repetitions, of the controls as well as of the experimental animals, have been done.

females (all homozygous ADH^S/ADH^S, as the Canton Special stock is monomorphic), and the F_1 has been self-crossed. From the resulting F_2 progeny, phenotypically *wild* females had been allowed to mate with *ebony* males, and this crossing scheme had been repeated for more than 50 generations, in order to eliminate the *wild* chromosomes. Thereafter, *ebony* and *wild* substrains (so-called *ebony* 'B' and *wild* 'B' in Figures 1 and 2) have been selected in 3 generations. When screened for ADH isozymic patterns and tested for alcohol dehydrogenase activity, both substrains turned out to be homozygous ADH^F/ADH^F and to have a very high dehydrogenase activity level, even higher than the ADH^F/ADH^F *ebony* e^{11} substrain (Figure 1).

When alcohol is the only source of energy available, the survival time is greater for flies of all the 5 substrains: *wild* 'B' and *ebony* 'B' have the highest record, followed by *ebony* e^{11} ADH^F/ADH^F, then *ebony* e^{11} ADH^S/ADH^S, and finally *wild* Canton Special, also ADH^S/ADH^S (Figure 2).

According to JACOBS[8–10], a high concentration of hemolymphal β-alanine is responsible not only for the melanic phenotype of the *ebony* flies, but also for the very poor coordination of their movements as well as for a deficiency in carbohydrate catabolism. A better tolerance for alcohol, and some capacity to use it as an energy source, may palliate in some way the behavioural and metabolic anomalies of the *ebony* flies; it could also provide some selective advantage in the competition with *non-ebony* flies, especially if the food sources are richer in alcohol, as, for instance in environments where fermentation takes place. Such characteristics may help to explain why the frequency of the *e* genes remains so high in populations where it is in competition with its wild type allele[11–14], especially in the colder environments which are known to favor the ADH^F allele.

Moreover, it is known that sexual activity in *ebony* e^{11} substrains is higher for ADH^F/ADH^F than for ADH^S/ADH^S flies[15]. In each of the 50 generations leading to 'B' substrains, *ebony* males are back-crossed with heterozygous females, and therefore sexual selection favors the ADH^F allele: this would explain why both *ebony* and *wild* 'B' substrains are homozygous ADH^F/ADH^F.

[8] M. E. JACOBS and K. K. BRUBACKER, Science *139*, 1282 (1963).
[9] M. E. JACOBS, Biochem. Genet. *1*, 267 (1968).
[10] M. E. JACOBS, J. Insect Physiol. *16*, 55 (1970).
[11] P. L'HÉRITIER and G. TEISSIER, C. r. Acad. Sci., Paris, Ser. Biol. *124*, 882 (1937).
[12] H. KALMUS, J. Genet. *47*, 58 (1945).
[13] A. ELENS, Experientia *14*, 274 (1958).
[14] M. E. JACOBS, Genetics *46*, 1089 (1961).
[15] J. VAN DEN HAUTE and A. ELENS, Drosophila Inform. Serv. *51*, in press (1975).
[16] W. SOFER and H. URSPRUNG, J. biol. Chem. *243*, 3110 (1968).
[17] H. URSPRUNG and L. CARLIN, Ann. N.Y. Acad. Sci. *151*, 456 (1968).

Autosomale Chromosomentranslokationen beim Fleck- und Braunvieh

Autosomal Chromosometranslocation of Piebald Cattle and Brown Cattle

G. F. STRANZINGER und M. FÖRSTER[1]

Lehrstuhl für Tierzucht an der Technischen Universität München, D-805 Freising-Weihenstephan (Bundesrepublik Deutschland, BRD), 16. Juli 1975.

Summary. Different autosomal chromosome fusions of piebald cattle and brown cattle are described. In randomly investigated breeds of cattle in Germany, we found 1/25 translocation in piebald cattle and a 1/29 Robertsonian fusion in brown cattle. Fertility differences were investigated.

Die umfangreichen Untersuchungen über die 1/29-Translokation beim Rind (GUSTAVSSON[2]; RIECK et al.[3]; QUEINNEC et al.[4]; POPESCU und BOSCHER[5]; HAGELTORN und GUSTAVSSON[6]) haben gezeigt, dass das Auftreten dieser Chromosomenanomalie in Europa gehäuft, jedoch nicht nur auf diese Länder beschränkt ist. Zusätzlich wurde erkannt, dass eine Prädisposition bestimmter Chromosomen zur Robertsonschen Fusion vorliegt, jedoch die Auswirkungen dieser Aberration auf phänotypische Merkmale von den verschiedenen Autoren unterschiedlich eingeschätzt und interpretiert werden.

In eigenen, stichprobenartigen Untersuchungen an ca. 200 Tieren aller in Deutschland vertretenen Rinderrassen konnten in zwei Rassen unterschiedliche, in heterozygoter Form auftretende Translokationen gefunden werden.

Da der erste aufgefundene Fall von einem weiblichen Tier ausging und die Nachkommen vor einem züchterischen Einsatz als heterozygot erkannt wurden, blieb die in den folgenden Abschnitten beschriebene Translokation auf wenige Tiere beschränkt. Der zweite aufgefundene Typ einer Translokation trat bei einem Besamungsstier mit einer grossen Nachkommenzahl auf, so dass die Verbreitung dieser Form bereits von Bedeutung ist.

Eine genaue zytogenetische Untersuchung durch spezielle Färbeverfahren an den Chromosomen der Translokationsträger ergab zwei verschiedene Fusionstypen.

Im ersten gefundenen Tier (Fleckvieh) war durch eine normale Anfärbung der Chromosomen der kurze Arm des Translokationschromosoms länger als das kleinste Chromosom Nr. 29. Durch eine Bänderungsfärbung (SEABRIGHT[7]) wurde die Möglichkeit geprüft, dass ein anderes als das 29. Chromosom an der Translokation be-

[1] Frau BECHER und Frau SPENGER möchten wir für die Unterstützung bei der Blutentnahme und Aufbereitung danken. Die Chromosomenuntersuchungen wurden aus Mitteln des Tiergesundheitsdienstes Grub (Dir. Dr. RICHTER) unterstützt, wofür an dieser Stelle der Dank ausgesprochen werden soll.
[2] I. GUSTAVSSON, Hereditas *63*, 68 (1969).
[3] G. W. RIECK, H. HÖHN und A. HERZOG, Zuchthygiene *3*, 177 (1968).
[4] G. QUEINNEC, R. DARRE, H. M. BERLAND und J. C. RAYNAUD, Symposium Madrid 1974, p. 131.
[5] C. P. POPESCU und J. BOSCHER, Symposium Madrid 1974, p. 165.
[6] M. HAGELTORN und I. GUSTAVSSON, Symposium Madrid 1974, p. 203.
[7] M. SEABRIGHT, Lancet *2*, 971 (1971).