N-acetyl-mannosamine or mannose, or (ii) to the action of periodate on the glycol bonds of more complex carbohydrate structures in carbohydrates similar to these or to those reported by Boyd.

The failure of similar eluates from cells treated with the viruses of influenza and Newcastle disease to inhibit may be due to their more vigorous enzymatic action. The importance of linkage is emphasized by the unpublished results (*12*) obtained in this laboratory, in which human urinary mucoprotein inhibited Rh antibody slightly after incubation with mumps virus, but not after incubation with influenza, NDV, or RDE; stronger inhibition occurred after incubation with trypsin.

MATTHEW C. DODD
NANCY J. BIGLEY
VIRGINIA B. GEYER
Department of Bacteriology, Ohio State University, Columbus

References and Notes

1. E. Hackel, R. E. Smolker, S. A. Fenske, *Vox Sanguinis* **3**, 402 (1958).
2. W. C. Boyd, M. H. McMaster, E. Waszczenko-Zacharczenko, *Nature* **184**, 989 (1959).
3. N. J. Bigley, R. W. Chandler, M. C. Dodd, *J. Immunol.* **80**, 85 (1958).
4. Anti-e sera were obtained from the Knickerbocker Blood Bank, New York, N.Y. All other sera were obtained from the Ortho Pharmaceutical Corporation, Raritan, N.J., and from Dr. Warren E. Wheeler, Children's Hospital, Columbus, Ohio.
5. Obtained from Dr. Robert M. McCluer, Research Biochemist, Research Division, Columbus State Institute of Psychiatry.
6. Obtained from Dr. Charles W. DeWitt, Research Division, Upjohn Co., Kalamazoo, Mich.
7. Obtained from Dr. Chester I. Randles, Department of Bacteriology, Ohio State University, Columbus.
8. O. Mäkelä, *Ann. Med. Exptl. et Biol. Fenniae, Helsinki* **35**, suppl. 11, 1 (1957).
9. G. Blix and L. Odin, *Acta. Chem. Scand.* **9**, 1235 (1955); Z. Klenk and K. Lauenstein, *Z. Physiol. Chem. Hoppe-Seyler's* **295**, 164 (1953).
10. H. Faillard, *Z. Physiol. Chem. Hoppe-Seyler's* **307**, 62 (1957).
11. A. Gottschalk, *Advances in Enzymol.* **20**, 135 (1958).
12. H. M. Johnson, N. J. Bigley, M. C. Dodd, unpublished data.

2 August 1960

Venation Polymorphism and Genetic Variability in Drosophila melanogaster Loew

Abstract. Experimental evidence indicates that phenocopy production may provide an inflated estimate of the importance of genetic variability and recombination in the production of venation phenodeviants.

Some phenotypic characteristics may be misleading indicators of genetic variability. Wing venation variants in *Drosophila melanogaster* Loew are particularly suspect. Milkman (*1*) has recently reported data which he interprets as supporting the notion that a pair of individuals carries "to a large degree the potential variability of the population." He used the first 1000 F_2 progeny of wild inseminated females as samples, and surmises that the appearance of a phenodeviant not present in the female grandparent is an indication of genetic recombination and production of "rare" genotypes.

The presence of four alleles for each locus among the offspring of a mating pair obviously leads to a great number of possible genotypes in the F_2 progeny. This number is not necessarily a large fraction of the total available to the species if many large multiple allelic series exist. This also seems trivial, since with even four alleles at each of five loci (*1*) there are 10^5 possible genotypes, a number far greater than the size of most F_2's examined.

Recombination within and between loci need not be the only source of phenodeviants. Indeed, the possibility exists that among near-homozygous inbred lines environmental variations, even within one culture, could produce such effects.

Thus we have at least two possible explanations for phenodeviants: (i) rare recombinants from highly heterozygous populations, or (ii) theshold effects common to many genotypes, leading to production of phenodeviants in some environments.

An experiment was performed with highly inbred sublines of Canton-S and Oregon-R. The flies were raised at 25 ± 1°C on agar-yeast-sugar–propionic acid food media. The parent stocks were examined each generation during the experiment, as were reciprocal F_1's and F_2's. The wing venation anomalies were recorded and in most cases sketched, and the flies were preserved.

The experiment was repeated by three individuals. Reported variants included extra or missing anterior and posterior cross veins, bifurcations of vein tips, extra venation from the center of cross veins, and other extra venation. Table 1 indicates the overall frequency of venation phenodeviants in each class. Chi-square analysis yielded probabilities between .99 and .30 that the observed differences were due to chance alone.

Neither the combined results nor any of the three replications gave indication of a significant difference in frequency between classes. The most surprising (though a nonsignificant) finding was the low rate of phenodeviants among the F_2's as compared to the other classes. This could indicate that flies in a recombinant population may be less variable in this respect than inbred flies.

In addition it was noted that phenodeviants tended to come in groups from individual culture vials, in any of the classes. This lends some support to the notion that this is a threshold phenomenon requiring particular environmental conditions in addition to genetic conditions.

Table 1. Frequency of venation phenodeviants.

Stock Tested	*N*	Abnormal wing venation (*N*)	Frequency (%)
Canton-S	6,322	30	0.475
Oregon-R	5,676	24	0.423
Canton-S × Oregon-R sum reciprocal F_1's	3,557	17	0.478
Canton-S × Oregon-R sum reciprocal F_2's	12,734	37	0.2905
Sum Oregon-R and Canton-S	11,998	54	0.4501

The data presented indicate that the incidence of venation phenodeviants is not significantly different in the inbred lines, the hybrids, and the F_2 recombinants. If this is the case, then we fail to see how incidence of this type of variation in F_2's of wild inseminated females provides evidence either for or against the existence of "a large degree" of variability in the individuals Milkman (*1*) tested. In a number of our cases the venation phenodeviants were inbred, 50 to 100 offspring were observed, and no phenodeviants were noted. Thus it is unlikely that one or two loci were particularly involved, and it seems very likely that each phenodeviant represented a developmental accident.

Our experiments were carried out under conditions which Milkman (*1*) has indicated are unfavorable to the production of the cross-veinless phenotype. The incidence was actually very low: 0.00017 in the Oregon-R and Canton-S stocks (one sample in each stock). No cross-veinless phenotypes were found among 16,291 F_1's and F_2's examined. Thus, although the cross-veinless phenodeviants could and did appear in the inbred lines, they did not appear among the recombinants. If recombination were to produce this sort of variability, then we would have expected to find more cross-veinless phenotypes among the F_2's than among the parental stocks.

Studies by Dubinin, Dobzhansky, Spencer, and many others have already clearly shown the amazing variability of wild populations. We feel that studies based on such labile characteristics as wing venation anomalies are unlikely to provide more reliable estimates unless an effort is made to distinguish the variability assignable to developmental accidents. The experiment reported

here indicates that this type of wing venation variant is relatively common, even in inbred lines, and that F_2 data unsupported by control information of this sort are likely to be misleading.

JACK BENNETT, RONALD L. CAPEK,
THOMAS R. KALLSTEDT,
ROBERT E. MOISAND
Department of Biological Sciences, Northern Illinois University, DeKalb

Reference

1. R. Milkman, *Science* **131**, 225 (1960).

8 June 1960

In my paper (*1*) I reported examining 1000 flies in the F_2 of each of 21 wild inseminated *Drosophila melanogaster* females. Of these 21 F_2's, 11 contained flies with defective posterior crossveins. There were 119 such flies among the 21,000 examined, and their distribution among the 11 F_2's was far from random. I cited this distribution, in the light of previous information, as evidence for the abundance of genes in natural populations which, in rare combinations, would greatly increase the probability of a fly's having defective posterior crossveins.

Bennett *et al.* (*2*) cite the morphological variation observed in a highly inbred (and ostensibly isogenic) strain to emphasize the point that morphological variation is not necessarily a reflection of underlying genetic variation. This is, of course, true; the question of cause must be put to any such observation.

I should like to confirm my conclusions with more recent information. I should then like to make some comments on the paper by Bennett *et al.*

First, a repeat of the experiment on later generations of the 21 strains gave good agreement, pointing to the persistence of differences among the strains. Second, I have been able so far to obtain a true-breeding, polygenic, crossveinless (*cve*) strain from each of two of the original strains (*3*). In the absence of intrastrain heterozygosity of *cve* genes, this would of course have been impossible. I should mention, in addition, that the crossvein defects of some of the strains were distinguishable from one another, and that this distinction was the same in both experiments.

Now I should like to discuss certain of the statements in the paper by Bennett *et al.* In the abstract the word *phenocopy* is used. Later, the implication is maintained that the only major alternative to genetic variation, as a cause of phenotypic variation, is environmental variation. In many cases, and very probably in this case of venation variation, a third force is extremely important. Wright (*4*) and Reeve and Robertson (*5*) call it "chance variation," and Waddington (*6*) calls it "developmental noise." Chance variation comprises the indeterminate events with developmental consequences. Although these authors discuss chance variation in terms of later development, it must be of equal importance from the start, for even genetically identical eggs are known to vary in size, content, and maternal environment, and adult structures are not independent of such variation.

Such chance variation is what forces one to designate an array of phenotypes for a given genotype under well-controlled environmental conditions. Such morphological variation in spite of apparently uniform genotype and environment is discussed to some extent in several of the references cited in my paper. The problem, then, is to distinguish the causes of morphological variability.

This distinction can often not be made conclusively on the basis of simultaneous controls. In my experiments, I believe the evidence of genetic variability was good. I should have stated that among the vials of any single strain, *cve* flies appeared to be distributed randomly. Thus the nonrandom distribution of *cve* flies among strains meant that the strains were not identical. Conclusive evidence in such experiments comes, as Bennett *et al.* say, by a sorting out and identification of the factors involved. In the two *cve* strains obtained so far, there are apparently 3 and 2 *cve* genes, respectively. The 3 are each on a different autosome but have not been further localized yet. The 2 strains, which come from different grocery stores, seem to share at least one *cve* gene. None of the other 19 strains seems to have it, supporting the possibility (*1*) that many alternative combinations of genes for making *cve* flies exist.

As to the data reported by Bennett *et al.* I find more contrast than comparison with my own. They did not run 21 parallel lines. And, whereas I reported 119 *cve* flies from 21,000, they report only 2 from 28,000. It is difficult to comment on the exact numbers of *cve* flies to have been expected had they raised their animals at 18°C. Nevertheless, the frequency of all venation variants they report is within the range reported for some groups of wild flies in an extensive study by Dubinin (*7*). This supports Bennett *et al.*'s point that morphological variation, even under controlled environmental conditions, is not definitive proof of genetic variation. I have, incidentally, recorded other unusual forms of venation also. Some are strain-specific, which does point to a genetic basis.

I believe it is relevant to cite experiments conducted in parallel on wild and on inbred strains by Waddington (*8*). He produced a variable morphological response to a given type of heat shock in both, but only the wild strain responded to selection for susceptibility. Bateman has done the same thing (*9*).

One of Dubinin's most significant contributions to the defining of the genetic basis of natural variation was his work with 240 lines from wild inseminated females (*7*). Examination of successive generations led to the observation of venation deviants in 163 strains, the response of some of these strains to selection, and the genetic analysis of certain of the selected strains.

I believe we are in a position now to take a census of *cve* genes and thus begin to record the details of the genetic basis of a representative form of natural variation (*10*). I should be delighted to hear from anyone interested in participating.

ROGER MILKMAN*
Zoology Department, University of Michigan, Ann Arbor

References and Notes

1. R. Milkman, *Science* **131**, 225 (1960).
2. J. Bennett, R. L. Capek, T. R. Kallstedt, R. E. Moisand, *Science* this issue.
3. This work was supported by National Science Foundation grant G-9785, and done at The University of Michigan.
4. S. Wright, *Proc. Natl. Acad. Sci. U.S.* **6**, 320 (1920).
5. E. C. R. Reeve and F. W. Robertson, *Z. Induktive Abstammungs-u. Vererbungslehre* **86**, 269 (1954).
6. C. H. Waddington, *The Strategy of The Genes* (Macmillan, New York, 1957); see a→ A. I. Lansing, *Proc. Natl. Acad. Sci. U.S.* **34**, 304 (1948).
7. N. P. Dubinin, *Zhur. Obshchei. Biol.* **9**, 203 1948); translated by I. M. Lerner. A discussion of this paper may be found in Lerner's *Genetic Homeostasis* (Oliver and Boyd, Edinburgh, 1954).
8. C. H. Waddington, *Evolution* **7**, 118, (1933).
9. K. G. Bateman, *J. Genet.* **56**, 443 (1959).
10. R. D. Milkman, *Genetics* **45**, 35, 377 (1960).
* Present address: Zoology Department, Syracuse University, Syracuse, New York.

11 August 1960

Demonstration of Canonic Gonial Mitosis and Meiosis in Parascaris equorum

Abstract. It is shown that, contrary to what has been held, separate canonic chromosomes, without fusion into a chromatin mass, occur in the meiotic prophase of *Parascaris equorum*. In mitosis no club-shaped chromosome ends are visible. These results, obtained with a modified fixation procedure, which is described, have been checked by supravital observation.

Mitosis and meiosis in the horse ascaris, *Parascaris equorum* (old name, *Ascaris megalocephala*), have been reported to show several discrepancies in relation to findings in the great majority of animals and plants. Early authors (*1*), described extraordinary phenomena