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Phillip J. Reitan
Luther College

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An Analysis of the Developmental Effects of the Embryonic Lethal Mutation X-23 in *Drosophila*¹

PHILLIP J. REITAN²

Abstract. The developmental abnormalities resulting from a recessive, second chromosome, X-ray induced lethal mutation acting during the embryonic period of *Drosophila melanogaster* are described. Embryos of 18-22 hours of development were studied. The pattern of abnormalities indicates that the lethal effect was due to a general cessation of development during the period from 9-14 hours. There is a general failure of mid-gut and muscle development. The hypoderm, foregut and hindgut structures appear to have developed normally. In the absence of muscle development, hypodermal differentiation produces deep furrows which distort the structure of the embryo. Variation in the expression of the lethal genotype produces some differences in the morphogenetic movements of this period. Germ band shortening occurs regularly, dorsal closure is sometimes present and head involution is seen in one embryo. These differences are related to the degree of hypodermal differentiation and muscle development which occurs. This is interpreted to mean that the force generated by the thinning and spreading of the hypoderm is responsible for these movements. In the course of normal development this force is controlled by the concurrent differentiation of the musculature.

This paper is a report on analysis of the developmental effects of a second chromosome, X-ray induced, recessive, embryonic, lethal mutation in *Drosophila melanogaster*. Our principle interest in this problem is the study of the nature of the normal developmental processes. Complete descriptive information is available from the work of Sonnenblick (1950), Poulson (1950) and Ede and Counce (1956). However, there is relatively little information on the relationships existing between the developing parts of the embryo. The structure of the egg is such that the classical techniques of extirpation, vital staining, transplantation and blastomere separation can not be used. The egg is enclosed within a tough chorionic membrane, contains a large amount of centrally placed yolk and features superficial cleavage and mosaic development. Some recent work using ultrasonic treatments (Counce and Selman, 1955) and ultra violet light (Hathaway and Selman, 1961) has been attempted to selectively destroy specific cells at specific times. But perhaps the most handy device for the production of developmental abnormalities has been the

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² Department of Biology, Luther College, Decorah, Iowa.

lethal mutation. In addition to contributing to the growing volume of literature concerning the relationship between the gene and development, genetically based abnormalities provide for a large number of specific effects occurring at specific times. Sufficient descriptive data may be obtained so that an analysis may provide some good guesses as to the normal sequence of events and their interrelationships is possible. No one embryo or group of embryos can be expected to provide very much information, but a comparison of abnormalities of several types and several sources may be expected to provide a much more comprehensive picture of *Drosophila* development.

MATERIALS AND METHODS

Source of Mutant Stock. The stock of X-23 mutants used in this work was obtained from Dr. Frank Seto who isolated the mutant, determined its location on the second chromosome and assigned the name (Seto, 1954). The stocks were maintained in a curly-balanced system. Curly is also a recessive lethal, but it has no morphological effect in the embryonic period (Hadorn, 1951).

Technique of Collection and Preparation of Embryos. Eggs were collected from four- to five-day-old females that had been mated 16 hours prior to the collection time. Collections were made only from rapidly laying individuals and were allowed to develop 18-22 hours before being fixed and stained for microscopic examination. Fixation was in Carnoy's fluid; staining was with Heidenhain's iron hematoxylin. Serial sections were cut at 10 microns. Eggs, prior to embedding in paraffin, were stained with eosin so that they could be seen readily. The embryos were oriented in the paraffin in rows so that up to 10 could be cut simultaneously and in the same plane.

Preliminary Analysis of Sectioned Material. A preliminary analysis of sectioned material was made to determine if the lethal mutation had a specific morphological effect and would be useful for this kind of study. One-hundred-eighteen eggs were examined. Of these, 74 had well developed embryos. Twenty-four percent (18) of these showed a consistent pattern of developmental abnormality. The large number (44) of undeveloped eggs probably reflects the high degree of egg inviability common in these stocks.

DESCRIPTION OF THE MUTANT EMBRYOS

Time of Lethality. A relatively consistent syndrome of effects indicates a general cessation of development between 9-14 hours. Normally during this period the morphogenetic movements of germ band shortening, dorsal closure, and head involution take place. Germ band shortening begins at about 9 hours, following

the formation of distinct hypodermal segments. As the germ band shortens the lateral hypoderm spreads dorsally to enclose the embryo. As dorsal closure is completed, head involution begins and is completed by 14 hours. (See Fig. 1.) All mutant embryos but one show developmental arrest to follow segmentation and precede head involution. In the single exception, head involution had occurred. However, all other aspects of the development of this embryo are consistent with 9-14 hour time. (See diagrams Fig. 4.)

Morphogenesis in the Mutants. In the 18 mutant embryos studied all but two show germ band shortening, seven had completed dorsal closure and one, head involution. In all but two, the hypoderm is characterized by a series of deep folds (Figure 2), two very prominent ones plus a number of small erratic infoldings. These furrows appear to result from uncontrolled segmentation movements.

Organogenesis. In normal development during this period, midgut development and muscle differentiation occur. Hypodermal differentiation and nerve cord formation are essentially complete. Nerve cord condensation is completed during the following period from 14-18 hours.

In the mutants both the foregut and hindgut structure are normal, but midgut development is not. Both anterior and posterior midgut rudiments are present, but they do not meet nor grow dorsally to enclose the yolk. In the single exception (Figure 3) the midgut is a complete sac, but the yolk still remains undigested.

Muscle differentiation is also incomplete. Normally myofibrils are present by 11 hours and muscular movements are begun by 14. In only one embryo (Fig. 3) is a developed somatic musculature present. In this one and one other, pharyngeal musculature is present. In other mutants the body wall musculature is seen as small strands without myofibrils or normal attachment to the hypoderm. Visceral musculature is largely absent.

Hypodermal derivatives such as chaetae and mouth hooks are seen in 4 of the mutant embryos. These structures usually appear at about 13 hours in normal embryos. Tracheae or tracheal rudiments are present in all embryos. These regularly appear prior to germ band shortening. The thinning of the hypoderm is evident in all of the embryos, except in the region of the head segments (Fig. 2). This hypodermal change is seen only in the one embryo in which head involution occurred.

The nervous system is evident. It is not condensed, and is often broken or displaced by the hypodermal furrows.

DISCUSSION

Nature of the Primary Disturbance. A morphological interpretation of the basic metabolic effect of a mutation is at best unsatisfactory. However, the general failure of midgut development and yolk utilization are suggestive of a lack of some basic activity leading to nutritive deprivation. This occurs in most embryos before muscle differentiation is completed. The hypoderm appears to be already past the point of no return and continues to differentiate. The presence of chaetae, mouth hooks and extreme hypodermal distortion in most embryos is evidence for this. The expression of the lethal is then consistent with the idea of "phase specificity" (Hadorn, 1951) in which specific genetic activity is expected to be unleashed at a specific time in development.

Causal Analysis of Abnormalities. Of particular interest to the morphologist is the analysis of those abnormalities which give some insight into the nature of normal development. In the mutant X-23 embryos, the principle cause of distortion appears to be the result of hypodermal differentiation. The deep furrows and irregular infoldings suggest that normal hypodermal thinning and spreading action is taking place, but the factors which normally control these movements are absent. This would suggest that muscle differentiation acts as the regulator of forces developing independently in the hypoderm. Indeed, the embryo in which somatic musculature is present is not distorted (Fig. 3). In the absence of muscle, germ band shortening and dorsal closure are frequently successfully completed. In a single embryo head involution occurs. The implication is that the normal morphogenetic movements of this period are all the result of hypodermal differentiation, but that these processes are directed by the developing musculature in normal embryos. Ede (1956 a, 1956b) in studying the mutants *Lff 11* and *X-2* reports similar hypodermal furrows and infoldings. He suggests from an analysis of these embryos that germ band shortening appears to result from hypodermal differentiation.

The appearance of X-23 mutants also indicates that hypodermal differentiation exists independently of muscle tissue. Ede (1956c) suggests that in his X-27 mutants and in insects in general this is the case. Earlier it had been suggested that perhaps an inductive relationship existed between muscle and hypoderm (Poulson, 1950) and that perhaps mosaicity was not complete in these embryos. Hathaway and Selman (1961), using an ultra violet microbeam for selective destruction of embryonic cells, report no departure from mosaicity. X-23 mutants do indicate that hypodermal differentiation is independent of muscle de-

velopment. Earlier inductive mechanisms are not, however, ruled out.

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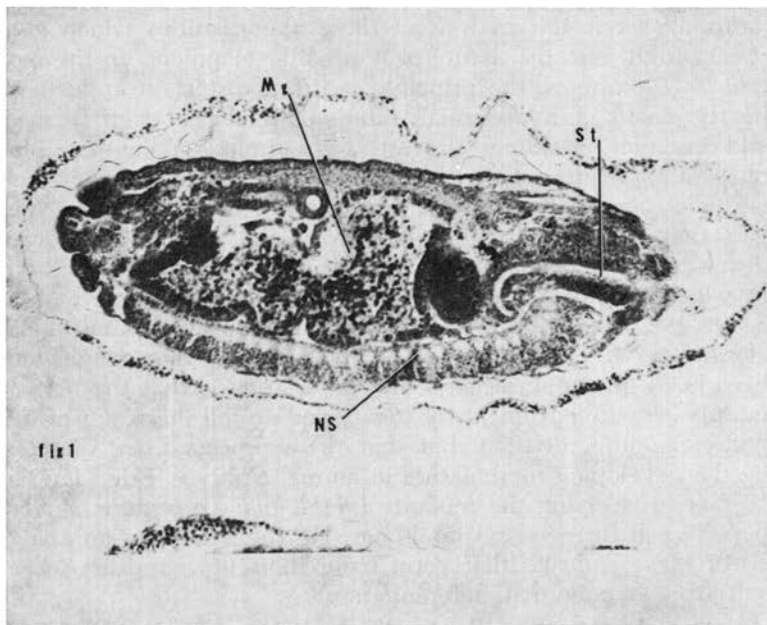


Figure 1. Normal embryo 14 hours.

Abbreviations used:

HG, hind gut; Hy, hypoderm; Mg, midgut; NS, nervous system; PM, pharyngeal musculature; PMgR, posterior midgut rudiment; St, stomodeum; Y, yolk.

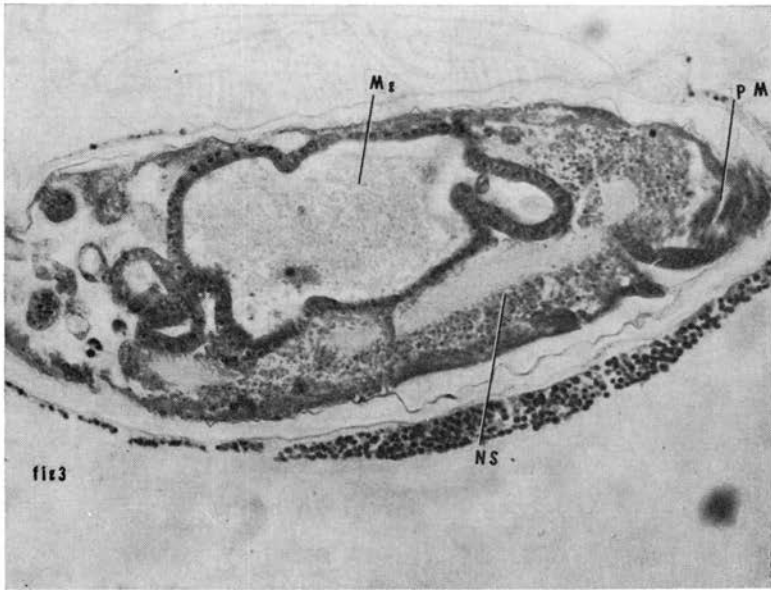


Figure 2. X-23 mutant—typical

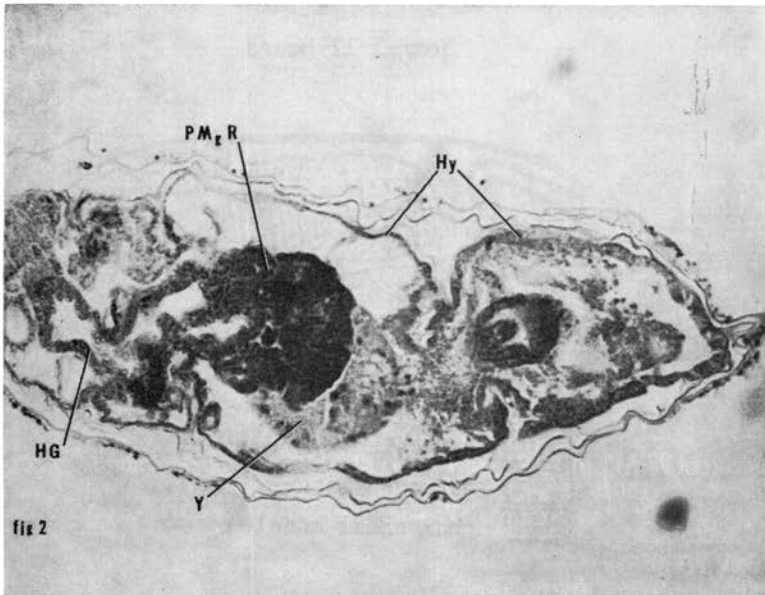
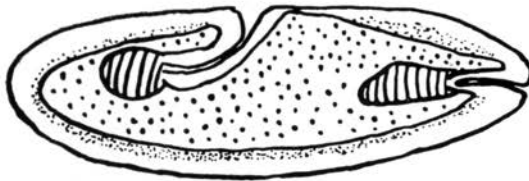
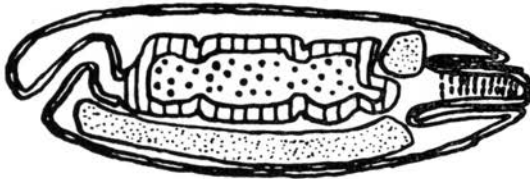


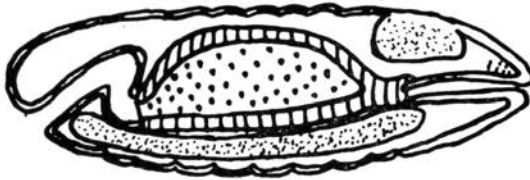
Figure 3. X- mutant—with musculature.



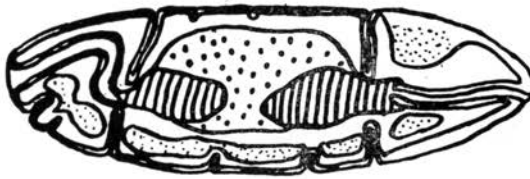
Normal 7 hours



Normal 14 hours



Normal 11 hours



Mutant X-23





-  yolk
-  mid-gut
-  nervous tissue
-  pharyngeal muscle

fig 4

Figure 4. Diagrammatic comparison of normal and X-23 embryos.