fore representative of the better submerged-body propulsors). The highest values of P_A were 229, 254, and 278 kg m sec^{-1}.

By use of the average measured value for D', values for P_D were calculated and added to each corresponding value of P_A to obtain total power output at each instant during acceleration. The total maximum power outputs were 387, 419, and 461 kg m sec^{-1} (5.08, 5.51, and 6.05 hp) for the three fastest runs. The calculated peak power output generally occurred about 1.5 seconds after the initial acceleration and about 0.5 seconds before top speed was reached; this fact suggests that the initial maximum power burst can last only 1.5 seconds. During the earlier stages of acceleration, the power output increased gradually from around 250 or 300 kg m sec^{-1} to the maximum reported power. Results show that maximum power output per unit body weight is about 1.5 times the maximum (3.6 kg m sec^{-1} per kilogram) reported (*4*) for a human athlete. Since human athletes can produce such power only during a single jerking movement of arms and legs, a crude extrapolation from the power data presented by Wilkie (*4*) suggests that, for a 1.5-second period, the power output by a porpoise may be greater than that by a human athlete by a factor of 2.5. Such greater power might be explained by one or more of these factors: greater ratio of muscle weight to body weight, better distribution of muscles, or greater oxygen content in the blood.

Maximum speed recorded during the tests was 11.05 m/sec (21.4 knots). Speed generally decreased by 10 to 20 percent soon after top speed had been reached, although the animals appeared to continue swimming fast. The calculated power output at top speed was 292 kg m sec^{-1}, 30 percent below the maximum power output for that run. The next highest speeds were 10.35 and 10.30 m/sec (20.1 and 20.0 knots). There is evidence that the top speed of one other run, not reduced because of poor camera exposure, was in the region of 11 m/sec. Distance from the start to the point of top speed generally varied from 13 to 17 m—7 to 9 body lengths; time to top speed averaged 2.0 seconds.

Detailed analysis of the speed-versus-time data tend to support the assumption that the porpoise's drag coefficient when swimming is approximately the same as when coasting; if it were significantly less when swimming, the calculated power output at top speed would be much lower than the calculated power peak that occurs during acceleration. On the other hand, if it were much greater when swimming, the power calculated in the early stages of acceleration would be much lower than the power calculated near top speed. Both these extremes appear unlikely from the training and physiological viewpoints.

An alternate check of top speed was made in an oceanaruim at Sea Life Park, Oahu, where two other specimens of *S. attenuata* were trained with four spinner porpoises to swim at high speed around a 70-m path circling a small island in a tank. The animals appeared to travel at extremely high speed, but reduction of data showed top speeds of only 7.7 to 8.3 m/sec, 2 to 3 seconds after the start.

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11. Supported by the Bureau of Naval Weapons Task Assignments (RRRE 04001/216-1/R009-01-01 and R360-FR106/216-1/R011-01-01). We thank Philip Helfrich, W. B. McLean, and T. A. Pryor for management and technical assistance, K. S. Norris for reviewing the manuscript, J. C. McCarthy for operations supervision, Patrick Quealy and Randy Lewis for aid in training, Walter Aymond, John Hayward, Jr., and P. S. Lang for operational assistance, William Schevill for additional references, and the University of Hawaii Marine Biological Laboratory for use of facilities. Contribution No. 5, Oceanic Institute, Makapuu Point, Oahu, Hawaii.

25 February 1966

Pteridines in the Fat Body of a Mutant of Drosophila melanogaster

Abstract. *A mutant,* white-mottled orange, *which deposits less than wild-type amounts of drosopterins in the eyes, has been found in* Drosophila melanogaster. *Variations in other pteridines were also observed. The mutant accumulates and later loses drosopterins in the abdominal fat body of adults.*

A spontaneous eye color mutant, *white-mottled orange* (w^{mo}), was found in a mass mating of *Drosophila melanogaster* where females were attached-X and males *maroon-like (ma-l).* When crossed with *yellow, scute, miniature, forked,* it was located at 1.5 ± units from the distal end of the X chromosome. The mutant was removed from *ma-l* stock and the phenotype proved to be mottled. A stock was established, but it still appears to be somewhat variable in phenotype. Generally, eyes of both males and females are light, mottled orange in color on emergence. During the first 3 or 4 days of adult life, when raised at 25°C, the color of the male's eyes becomes almost brown, while the color of the female's eyes remains considerably lighter.

Mutant w^{mo} was tested in combination with several other mutants. With *ma-l* both males and females have the same eye color on emergence as the mutant without *ma-l,* but the eyes remain much lighter when *ma-l* is present and no mottling is present with *ma-l.* The new mutant, heterozygous with *white,* has very dark eyes on emergence—about the color of older *ma-l* alone—which do not appear mottled. The mutant in combination with *white-Brownex* is lighter in color than when combined with *white* and again does not appear mottled. Interaction between *white-apricot* and w^{mo} causes about the same coloration as the *white-Brownex* combination does, but the eyes appear mottled. The new mutant in combination with *scarlet, vermillion,* or *brown* gives colors slightly lighter than the mutant alone and the colors are not mottled. None of these combinations darkens with age as w^{mo} alone does.

In order to survey eye pteridines, single heads of the mutant w^{mo} were mashed directly on Whatman No. 1 chromatographic paper. The chromatograms were developed at 18°C in dark-

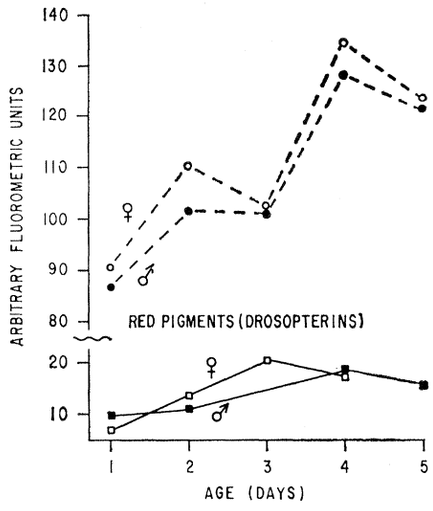

Fig. 1. Quantities of drosopterins in the heads of adult wild-type and w^{mo} individuals. Dotted lines represent wild type and solid lines, w^{mo}. Open symbols, females; solid symbols, males.

ness by the method of Hadorn and Mitchell (*1*)—double, one-dimensional, ascending paper chromatography that employs a mixture of *n*-propanol and 5 percent ammonia (2 :1), and thoroughly dries the paper between runs. Each paper usually contained 14 separate heads. The strips of the various pigments (as viewed under an SL 3660 ultraviolet Mineralight) were cut out, and the intensity of the fluorescing color was measured with a Turner model 111 fluorometer. The secondary filters we used were as follows (numbers indicate those supplied by G. K. Turner): for drosopterins, 2A and 23A; for xanthopterin and isoxanthopterin, 2A, 58, and 1-60; for sepiapteridine, 2A and 58; and for HB compounds, 2A and 75. The development of the drosopterins has been plotted in Fig. 1 so that males and females of both wild type and w^{mo} may be compared. (Each point in all figures represents a mean value derived from a minimum of 28 measurements.) Although the amount of drosopterins in heads of wild-type females is consistently higher than that in the male, this difference does not appear significant.

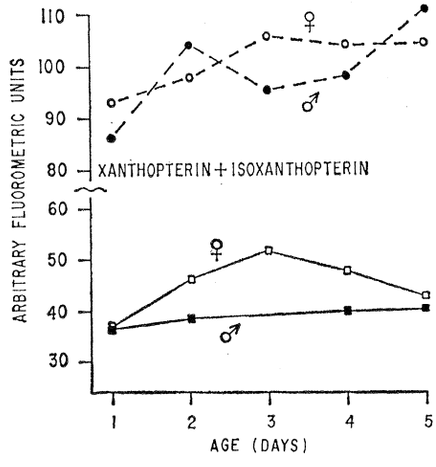

Fig. 2. Quantities of xanthopterin and isoxanthopterin in the heads of adults wild-type and w^{mo} individuals. Symbols same as in Fig. 1.

Change in the amount of drosopterins in heads of w^{mo} individuals is very slight over the 5 days of test. This would probably indicate that darkening of the eyes is not due to a further accumulation of red pigments, but is a result of further deposition of ommochromes, melanin, or sepiapteridine. However, the ommochromes have not been measured. The amount of red pigments increases in wild-type heads during the first 5 days of imaginal life. The two decreases in rate of accumulation during the 3rd and 5th days cannot be explained at the present time.

Because of poor separation, concentrations of xanthopterin and isoxanthopterin were measured together (Fig. 2) as a single area of the chromatogram. These amounts seem to remain approximately the same during the 5 days, although w^{mo} contains less than half that of wild type. No explanation is now offered for the difference between wild type and mutant.

Concentrations of sepiapteridine and HB ("himmelblau" compounds including 2-amino-4-hydroxypteridine and biopterin) were added after measurement (Fig. 3). Sepiapteridine and biopterin are apparently very closely related in a common metabolic pathway (*2*). The combined concentrations increase approximately twofold during the first 5 days in w^{mo}, while there is a dip during the first 3 days in wild type followed by a slight increase. In both this mutant and *sepia*, biosynthesis or deposition of red pigments is interrupted with a concomitant increase in the amount of sepiapteridine (*3*). Eyes of the mutant *sepia* are very dark brown, owing to the accumulation of sepiapteridine, and some of the eye darkening in w^{mo} may be explained by accumulation of this compound. However, the differential between males and females cannot be accounted for on this basis.

Before the mutant was isolated from *ma-l*, it was noted that some of the males had an unusual orange-red color in their abdomens. Upon dissection, this color was seen in the abdominal

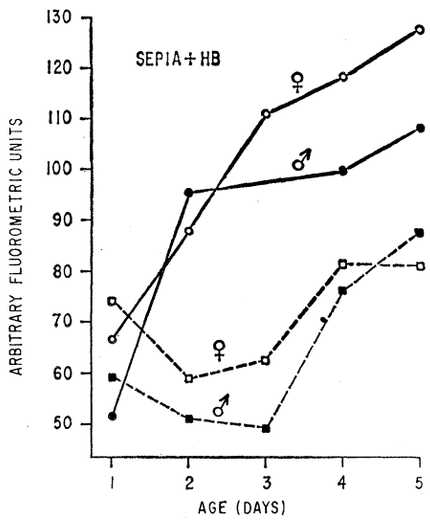

Fig. 3. Quantities of sepiapteridine and HB in heads of adult wild-type and w^{mo} individuals. Quantities of the two chemicals added together after measurement. Symbols same as in Fig. 1.

fat bodies, and, upon chromatography by the method of Hadorn and Mitchell (*1*), the color appeared to be attributable to drosopterins. Red pigment of the fat bodies separated into two vague fluorescent components with very low R_F values (0.05 to 0.1) and nothing was left at the origin. Consequently, there appears to be no large amount of ommochromes contributing to the color of the fat bodies. A small amount of HB compounds was also present in the fat bodies.

In establishing the stock of w^{mo} an attempt was made to determine whether accumulation of red pigments in abdominal fat bodies was also a result of

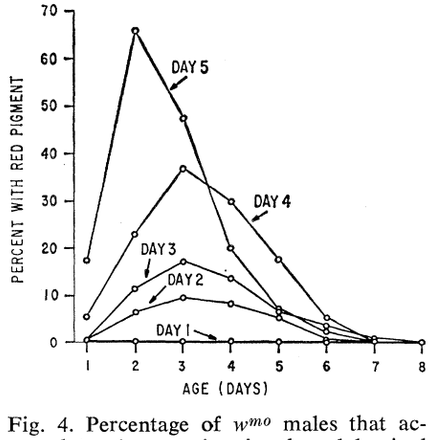

Fig. 4. Percentage of w^{mo} males that accumulate drosopterins in the abdominal fat body. Each line represents those flies that emerged on the designated day. They were checked daily for the appearance and disappearance of drosopterins (abscissa).

white mutation. None of the several thousand females that we examined ever exhibited this abnormal accumulation of drosopterins, and furthermore, even some of the males did not exhibit the phenotype. However, outcrosses to various wild stocks, including Oregon-R and Canton-S, failed to remove the red fat body phenotype. Accumulation of various pteridines in the testes, but not in the ovaries, has been discussed as a secondary sex characteristic in *Drosophila* (*2*).

At first it appeared that those males that developed this pigment did so in the 2nd or 3rd day of adult life. To determine the time of pigment development the following was done: Culture bottles containing 30 pairs of w^{mo} adults were set up at 23°C, and adults were allowed to lay eggs for 1 day. (All flies were raised on cornmeal-yeast-agar medium.) Adults that emerged from these cultures within each 24-hour period were collected and observed daily. Any male that showed visible red pigment in the abdomen was removed from the collection and observed daily for disappearance of the pigment (Fig. 4). Males that emerged during the 1st day never accumulated drosopterins in the fat bodies; those that emerged later, however, did show red deposits in the abdomens. It seems that the later the adults emerged—the longer the developmental period—the sooner the males showed red pigment. The increased developmental period also corresponds with an increased frequency of phenotypic expression. Furthermore, the day-5 males lost the pigment earlier.

In many of the males that were dissected during the time of pigment loss, dark red-brown depositions were seen in the Malpighian tubules. Since the Malpighian tubules reputedly serve as excretory organs (*4*), it is possible that drosopterins or their breakdown products are being excreted through the intestinal tract (*5*). Furthermore, on emergence the fly contains two types of abdominal fat cells (*6*). Disappearance of the pigment corresponds quite well to disappearance of the "pupal-fat" cells.

Protein granules are apparently necessary for normal pigment deposition in the eyes of several insects (*2*). Goldsmith and Kramer (*7*) have described large protein granules in unpigmented *D. melanogaster* fat body, but these are much larger than those found in eyes. Rizki (*8*) has reported the accumulation of ommochromes in the anterior fat body of *Drosophila*. He did not, however, report finding protein granules in this organ. Phase microscopy of living fat body from w^{mo} individuals showed no granules in the cytoplasm.

Testes and Malpighian tubules accumulate drosopterins in some mutants and species of *Drosophila* (*9*). Fat bodies, whose function has been likened to vertebrate liver (*10*), may also play a role in pteridine metabolism.

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21 February 1966

Lymphocytes from Thymectomized Rats: Immunologic, Proliferative, and Metabolic Properties

Abstract. *Thoracic duct lymphocytes from neonatally thymectomized Lewis rats fail to produce runt disease in newborn Brown Norway rats when injected with up to ten times the number of normal lymphocytes needed to cause runting. The immunologically deficient lymphocytes appear, however, to confer tolerance, and at least some enlarge and divide when stimulated in vitro with phytohemagglutinin, or xenogeneic or allogeneic cells. Small lymphocytes from thymectomized animals have defective RNA metabolism as judged by a marked impairment in their ability to incorporate uridine-5-H^3 or cytidine-H^3 in vitro.*

The immunologic defects which develop in rats thymectomized at birth have been considered to arise from a failure of central immune mechanisms, such as cell proliferation and interaction, which may be dependent upon thymic function in vivo (*1*). However, it has also been demonstrated that isolated spleen cells from thymectomized mice are in themselves immunologically deficient, for they exhibit a decreased capacity to produce runt or graft-versus-host disease when transfused to normal, allogeneic recipients (*2*). Because the latter work indicated that lymphocytes from thymectomized rodents may be qualitatively as well as quantitatively deficient, it seemed of value to confirm and extend the study in the rat where techniques for lymphocyte culture might permit a further characterization of the nature of the cellular disorder. Accordingly, I describe attempts to induce runt disease and immunological tolerance in Brown Norway (BN) rats by injections of thoracic duct lymphocytes from neonatally thymectomized Lewis rats. Certain proliferative and metabolic characteristics of the immunologically deficient lymphocytes are described as they were observed in short-term tissue culture.

In studies such as this, where identification of the specific cell type involved in immune reactions is important, thoracic duct lymph offers an advantage over spleen or other lymphoid cell suspensions in that it contains lymphocytes in nearly pure population. Because of this, and because thoracic duct lymph yields immunologically active cells (*3*), it was collected either from the neck (*4*) or abdomen (*5*) of donor rats and used throughout the study. Lewis rats (less than 1 day old) were thymectomized by splitting the sternum and dissecting the thymus free after the animals had been chilled at −10°C until they were apneic (5 to 10 minutes). Control animals were either sham-operated or, more commonly, untreated. After serving as cell donors, all thymectomized rats were autopsied to assure the completeness of thymectomy.

The capacity of lymphocytes from neonatally thymectomized Lewis rats to induce runt disease in newborn BN animals was studied first. Thoracic duct cells from thymectomized donors were washed in Hanks balanced salt solution, and, after being resuspended in 0.1 to 0.3 ml, were administered in doses of 1 to 20 × 10^6 lymphocytes per recipient. All injections were given into