was confirmed by an analysis of the left tibia of the bird, which 40 days after feeding, showed about 7% of the phosphorus fed.

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Some Observations on the Larval Growth Rate and Viability of Two Tumor Strains of *Drosophila melanogaster*

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In a recent paper (*3*) it was pointed out that spontaneous tumors occurring in several genetic tumor strains of *Drosophila melanogaster* showed marked decrease in incidence if the cultures were maintained at a temperature of 30° C. While at lower temperatures responses in terms of incidence varied considerably in the several strains studied, the decrease in incidence at the high end of the range was uniform for all.

The nature of this decrease in incidence is of considerable interest, since the temperature change, like any environmental change, must produce its effect through the physiology of the individual. In their publications dealing with viability and survival of strains of *D. pseudoobscura* (*1*), Dobzhansky and Spassky point out that the survival values of strains homozygous for various chromosomes including wild types may vary considerably with temperature change, even approaching a lethal or semilethal condition at temperatures which might not normally be considered extreme. This being the case, one may wonder if the decrease in tumor incidence noted above is the direct result of such a semilethal or other similar effect related to the chromosomal complement of the tumor strains, which manifests itself at 30° C. On the other hand, it could result from a factor such as an altered growth rate at higher temperature, which does not affect the viability of the culture to any great degree, but may affect the expression of certain genetic characters (*2*).

The paucity of studies dealing with temperature effects on larval growth and survival in tumor strains has made it necessary to probe a few fundamental points before undertaking any detailed investigation of growth rate or survival in relation to the tumor problem.

First, we should have some idea of how the range of 20°–30° C affects the viability of the tumor strains. Second, it would seem important to know if there is any significant variation of growth rate in tumor strains which results from altered temperatures within this range. Finally, if there is variation, does each strain have its own level of reaction, or do they all respond in about the same way? Some preliminary answers to these questions may be found in the data presented in this report. These data are admittedly far from quantitative, since they are derived from experiments set up only as precursors to more thorough studies. They are published now largely as a supplement to the paper mentioned above (*3*).

Two of the tumor strains used in the previous investigation were selected as the basis for this study. These were of the genetic make-up bw tu (a strain showing high incidence of tumors at room temperature as well as low constant temperature, but showing sharp decline at 30° C), and st sr e^s ro ca, tu-36a (a strain low in incidence at 20° C and at 30° C, but showing increased incidence between these extremes). A Burlington, Vermont, wild strain was selected as a control. All three strains were fairly closely inbred, but could not be considered isogenic.

Cultures of these stocks were put in half-pint bottles which were placed in incubators set at 20° ± .5° C, 25° ± .5° C, and 30° ± .5° C. From these cultures eggs were collected on yeast-seeded molasses agar blocks, mounted on the inner surface of the bottle cap (*4*). Eggs were collected over 4-hr periods and, after collection, were transferred to 4″ Petri dishes containing molasses agar well inoculated with yeast. Since the number of eggs placed in any one dish was limited to 150, the number of dishes representing each collecting "run" varied, depending on the number of eggs collected during the period. Five hundred eggs were considered a minimum for study of each strain at each value. In some cases several collecting "runs" were necessary to gather this minimum number; in other cases all eggs were gathered in a single 4-hr period. After transfer of the eggs to the Petri dishes, they were replaced in the incubator, allowed to hatch, and the larvae allowed to develop. Fresh yeast was added from time to time to insure optimum food conditions in all cultures.

Since larval growth only was of interest, the time of pupation, marking the end of the larval period, was used as the basis for comparison of growth rate. The end point arbitrarily selected was the time when the number of pupae equaled half the number of eggs started. In timing the "runs," the 4-hr collection period was not included, the start of the "run" being set as the time of transfer of eggs into the Petri dishes.

Table 1 sets forth the data for the three strains at the three temperature values. Since the cultures were not under constant observation, but were examined at intervals during the day, the end point of 50% pupation is not exact, and the allowance for error in terms of elapsed time between the final reading and the one previous to it is stated. In a few cases this interval was overnight, and so is quite large. However, in these cases it will be noted that this large interval does not

confuse the results obtained. The number of pupae present at the time of final reading is also stated.

Upon consideration of these data, the first fact that stands out is that neither tumor strain has even 50% viability at 30° C, since both fail to reach the end point. Whether this represents sterility induced by high temperature or a failure to develop which may be regarded as a lethal or semilethal condition is not at present determined. It is clear, however, that in these tumor strains the reduction in tumor incidence at 30° C is accompanied by reduced viability.

TABLE 1

GROWTH RATE OF LARVAE AT THREE CONSTANT TEMPERATURE LEVELS

Run No.	No. of eggs	No. of pupae at final reading	No. of hrs to final reading
		30° ± .5° C	
		st sr stock	
1	203	92 (total pupation for run)	144–4
2	105	39 " " " "	158–3
3	271	92 " " " "	143–4
		bw tu stock	
1	200	79 (total pupation for run)	160–14
2	240	104 " " " "	141–2
3	225	93 " " " "	147–3
		wild stock	
1	530	296	113–14
		25° ± .5° C	
		st sr stock	
1	60	31	138–3
2	90	46	150–4
3	220	111	160–14
4	200	109	147–4
		bw tu stock	
1	140	72	142–3
2	120	61	165–4
3	311	200	142–4
4	110	62	142–4
		wild stock	
1	625	313	122–4
		20° ± .5° C	
		st sr stock	
1	744	414	213–4
		bw tu stock	
1	355	190	192–4
2	304	155	200–4
		wild stock	
1	712	356	260–2

As a second point, it would seem that the developmental rate of larvae of all three strains is, as might be expected, markedly slower at 20° C than at 25° C or 30° C, the control showing the slowest development of the three at 20° C and the fastest at the other two levels. On the basis of the results obtained, a comparison of control and experimental strains at 30° C is not justified, since the figure noted under control represents pupation of 50% of the eggs started, while those noted for the tumor strains represent total pupation. This total is, of course, less than 50%. Therefore, restricting present consideration to the 20° C and 25° C levels, it is interesting that both tumor strains develop more slowly at the higher temperature than does the wild, but more rapidly at lower temperature. The bw tu strain is evidently the least retarded at 20° C.

Finally, as far as can be noted in this work, viability is no different in the three strains at the lower temperature levels. In terms of tumor incidence this is confusing, since bw tu shows high incidence at 20° C, while st sr shows much-reduced incidence at this temperature. The fact that viability is not reduced in st sr probably indicates that this reduction in tumor incidence is dependent on other factors than that noted in the two strains at 30° C. Some clue bearing on this may lie in the fact that these tumor strains do have different developmental rates at 20° C, and this is now being investigated along with other phases of the tumor problem.

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High Insulin Tolerance in an Inbred Strain of Mice

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A case of extreme tolerance to insulin has been found in an inbred strain of mice. It is inherited. Cases of high insulin resistance are of interest in connection with carbohydrate metabolism and the possible mechanism of insulin action, and inherited variations are important in view of the recognized procedure of assaying the potency of insulin by determining the convulsive dose in white mice (*5, 6, 9*). Although controls involving temperature and food consumption prior to injection are generally practiced, controls involving possible hereditary variations in these white mice are not considered. An hereditary difference of the magnitude found in our strain is also of interest in connection with the sort of physiological differences that may be inherited and the possible extent of such variations in man.

Differences in insulin tolerance have been described for some classes of vertebrates. Many birds (quail, pigeon, fowl, etc.) have a higher tolerance to insulin than do mammals (*2, 3, 4*). Likewise, certain differences in insulin tolerance have been noted within a single species. In humans, cases of extremely high insulin resistance have been observed among some diabetic patients (*8*). McIntyre and Burke (*7*) found in albino rats one strain which had a tolerance 10 times as high as the standard strains. In mice, Allen (*1*), using an unspecified number and type of mice, reported the dose necessary for beginning shock to be 1,000 units/kg. These results are con-

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