

## RESOURCE AVAILABILITY, MATERNAL EFFECTS, AND LONGEVITY

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**Abstract**—Experiments with a clone of the cladoceran *Daphnia pulex* indicate that the nutritional conditions of the maternal environment play a major role in determining the progeny's phenotype. Apparently, by influencing the physiology and/or morphology of individuals during early development, maternal investment not only enhances juvenile survival but has long-lasting, favorable effects on the progeny's ability to convert resources into growth and reproduction as well as negative repercussions for adult survival of the progeny. Life-span may also be radically altered by modifying food schedules within an individual's life. Neither reproductive effort nor rate of living hypotheses can explain longevity variation within *Daphnia* clones; rather the onset of senescence appears to be associated with a general breakdown in the ability to incorporate energy into biomass. Analysis of our results as well as earlier data with a "rate of aging-threshold vitality" model suggests that increasing the availability of food to an individual increases the rate of aging while decreasing the threshold vitality necessary for survival and that increasing maternal investment increases both the vitality at birth and the rate of aging of the progeny.

### INTRODUCTION

PARENTAL EFFECTS, especially maternal effects (i.e., the influence of the maternal environment on the phenotypic expression of progeny), are well-documented in a diversity of animals (David, 1961; Falconer, 1981) and have recently attracted a great deal of attention from experimental gerontologists (Lints, 1978). However, with the notable exception of David's (1961) thorough investigation with *Drosophila*, almost all existing work on parental effects has focused on maternal age effects, i.e., the influence of maternal age on offspring quality, and the interpretation of many of these studies is open to criticism (Lints, 1978). Except for Robertson and Salt, 1981, very little consideration has been given to the progeny character that would seem most relevant to gerontologists—longevity.

Yet since a diversity of characters including egg size (David, 1962) and nucleic acid content (Tsien and Wattiaux, 1971), developmental rate (Delcour, 1969), body size (Falconer, 1981), and genotype (Bridges, 1929; Valentin, 1973; Kram and Schneider, 1978) are known to be influenced by maternal effects, and since these traits are often correlated with longevity (Comfort, 1979; Lamb, 1977; Lints, 1978), it is not unreasonable to expect early maternal influences (operating solely through the egg) to be important determinants of an individual's life-span. The demonstration of Lansing effects, cumulative and reversible parental age effects, on the life-span of a diversity of organisms (Lansing, 1947;

Ashby and Wangermann, 1954; Murphy and Davidoff, 1972) is further supportive of such an expectation.

We have investigated this problem at an unparalleled level of simplicity using a single clone of the parthenogenetic cladoceran, *Daphnia pulex*. In the following pages we demonstrate not only that the nutritional environment of the mother has multiple and long-lasting effects on the phenotypes of her progeny, but also that these effects are highly dependent on the environment of her offspring. In addition, we consider our results in light of recent speculations on the mechanisms of aging and examine a general model that may be of utility in explaining life-span variation among individuals under controlled environmental settings.

## METHODS

All *Daphnia* used in this experiment were third generation descendents of a single parthenogenetic female drawn from a laboratory stock of an obligately unisexual strain of the Group A composite genotype (Lynch, 1983); as parthenogenesis in *Daphnia* is of the ameiotic type, all experimental individuals were genetically identical, barring mutations. The progeny from the first clutch of the stem mother (the grandparental generation) were separated into high and low food treatments until they released their first clutch, the members of which (the parental generation) were treated in an identical manner as their parents (Fig. 1). These two preliminary generations insured that all experimental individuals within lines would have identical past histories.

The first clutch progeny of the parental generation on high food were randomly divided into high (HH) and low (HL) food lines; similarly, progeny from low food lines were separated into high (LH) and low (LL) food lines. Thirty individuals were exposed to each treatment. In the absence of maternal effects the HH and LH lines should be identical to each other, as should the HL and LL lines.

The food supply consisted of a 5:1 cell density ratio of pure cultures of *Scenedesmus dimorphus* and *Chlamydomonas reinhardtii*: high food - 80,000 and 16,000 cells/ml, and low food - 8,000 and 1,600 cells/ml, respectively. The algae were grown on a medium modified from Guillard and Lorenzen, 1972 and supplemented with vitamins; new cultures were inoculated every 7 days, insuring that algae used as food always came from cultures aged 1-2 weeks. Cell densities were determined with a hemacytometer, centrifuged from the algal medium, and then diluted in a defined zooplankton medium (modified from Murphy, 1970) to the appropriate density.

Algae and *Daphnia* were grown in a Percival incubator at 20°C on a 12:12 hr light:dark cycle. *Daphnia* were maintained individually in 40 ml of medium, with fresh medium being substituted every other day. Every in-

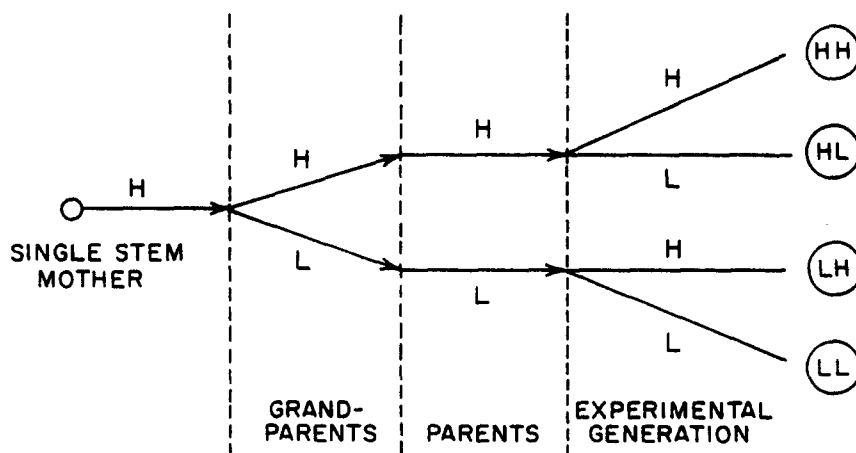


FIG. 1. The experimental design. Three preliminary generations were completed prior to the experimental generation. H and L refer to high and low food levels. The four experimental lines are HH, HL, LH, and LL, the first letter referring to the conditions under which the mother was raised, the second to the conditions under which the measured individual was raised.

dividual was examined daily for survival and measured for growth (total length) and reproduction (size of clutch carried, and/or number and size of new offspring released) under a Wild M-8 dissecting microscope.

The relative amounts of resources allocated to growth and reproduction by the different lines were estimated from dry weight measures. Since eggs and somatic tissues in cladocerans are approximately calorically equivalent on a dry weight basis (Schindler, 1968; Snow 1972), proportional investments calculated in terms of dry weight should be very similar to ratios based on calories.

The amount of dry weight invested in growth ( $\mu\text{g}/\text{day}$ ) was derived by converting changes in length between instars to dry weight by the length-weight regression appropriate to the food supply and dividing by the instar duration. The length-weight relation was determined by drying individuals of various lengths for 48 hrs at  $60^\circ\text{C}$  and then weighing them to the nearest microgram on a Mettler M22 electrobalance. Of the adult females weighed, only those that had recently emptied their ovaries by producing a clutch were utilized, the clutch being removed prior to weighing; this insures that estimated rates of investment in growth are not biased by changes in ovary or progeny weights. Logarithmic regressions of dry weight ( $\mu\text{g}$ ) on length (mm) were highly significant ( $p < 0.0001$ ):  $W = 11.04L^{2.05}$  for individuals grown on high food, and  $W = 7.81L^{2.16}$  for individuals grown on low food.

The amount of dry weight invested in reproduction ( $\mu\text{g}/\text{day}$ ) was estimated as (number of eggs produced/instar  $\times$  dry weight/egg  $\div$  instar duration). Mean dry weights for freshly deposited eggs are 2.11 and 1.73  $\mu\text{g}$  under high and low food conditions.

## RESULTS

The growth, survivorship, and reproductive schedules for the four cohorts are illustrated in Fig. 2. Significant maternal effects with respect to growth existed for the individuals grown on low food to those individuals whose mothers were grown on high food (HL) consistently having larger sizes than the LL control lines. Although the effects are not as pronounced, the maternal environment also influenced the growth of the high food lines. As the growth curves within food treatments are essentially parallel after 10 days, most of the maternal influence on growth must have been expressed early in life.

The maternal environment had no influence on the reproductive schedule of the high food lines, but there were pronounced differences between the low food lines. Until about the twelfth day of life (through the second clutch), the HL individuals carried clutches that were substantially larger than those of LL females and nearly identical to those of the high food lines; immediately thereafter, the low food lines converged and remained similar with respect to reproduction. The important point is that early in life HL individuals invest nearly as much in reproduction (on an absolute scale) as would be the case if they were growing on high food.

Thus, maternal investment has a major influence on the progeny's ability to harvest energy at relatively low food concentrations. When the HL and LL lines are compared, it becomes clear that a 0.38  $\mu\text{g}$  dry wt. (22%) additional investment per egg is converted into an  $\sim 100\%$  increase in the progeny's ability to incorporate energy into growth and reproduction (F) early in life (Fig. 3). Such an increase in net energy intake implies that maternal effects alter the physiology and/or morphology of individuals during development in ways that generate very major phenotypic changes later in life.

Two differences of importance with respect to survivorship also arose between the lines (Fig. 2, Table 1). Early survivorship was significantly lower for the LL than for the other lines. Being raised on high food or descending from a mother raised on high food nearly guaranteed survival to maturity. However, individuals grown on low food but derived from high food mothers (HL) experienced a very significant and precipitous increase in mortality after 40 days; no HL individual lived beyond 50 days. The three remaining lines exhibited very similar  $l_x$  schedules after 50 days, with  $\sim 30\%$  of the individuals surviving to 50 days and several surviving as long as 70 days.

One characteristic for which the lines were remarkably constant was the proportion of

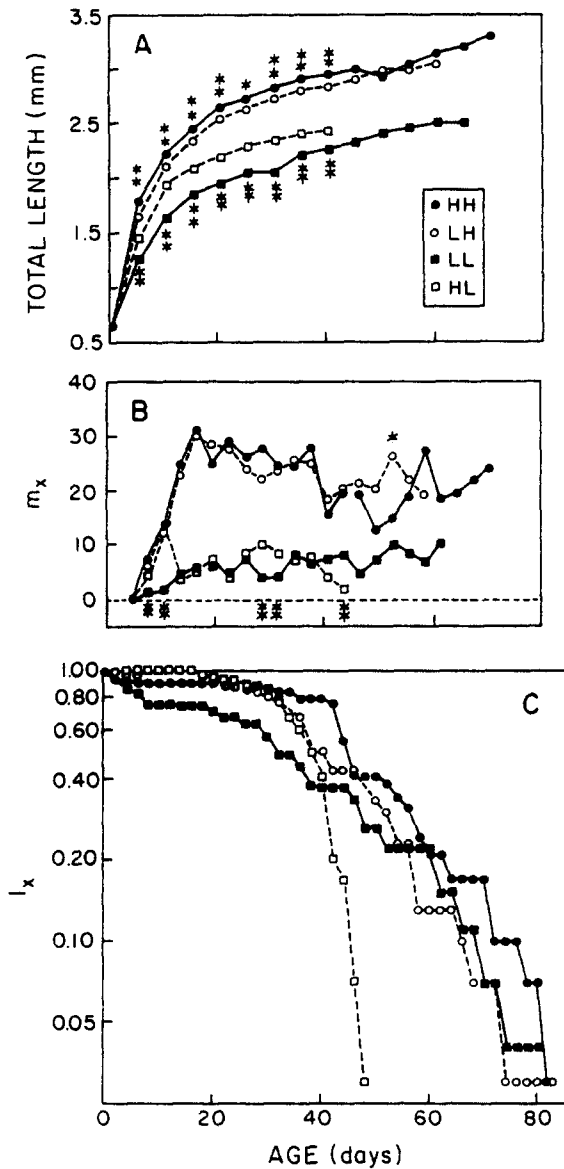


FIG. 2. Growth, reproduction, and survivorship schedules for the four lines. (Data for body size and  $m_x$  are only plotted when the sample size  $\geq 5$ .)

A. Significant body size differences between high (HH, LH) and between low (HL, LL) lines are denoted by \* ( $p < 0.05$ ) or \*\* ( $p < 0.01$ ) (t test with unequal sample sizes); all comparisons across high and low food treatments were significant.

B.  $m_x$  is the average number of offspring released over a three-day interval; significant differences between low and between high food lines are indicated by \* ( $p < 0.05$ ) or \*\* ( $p < 0.01$ ) (t test with unequal sample sizes). The reproductive output of HL individuals was not significantly different from that of LH individuals at either 7.5 or 10.5 days or from that of HH individuals at 10.5 days, but HH and HL individuals were significantly different ( $p < 0.05$ ) at 7.5 days. All other comparisons between high (HH, LH) and low (HL, LL) lines exhibited significant differences.

C. Comparisons of the survivorship ( $l_x$ ) schedules are provided in Table 1.

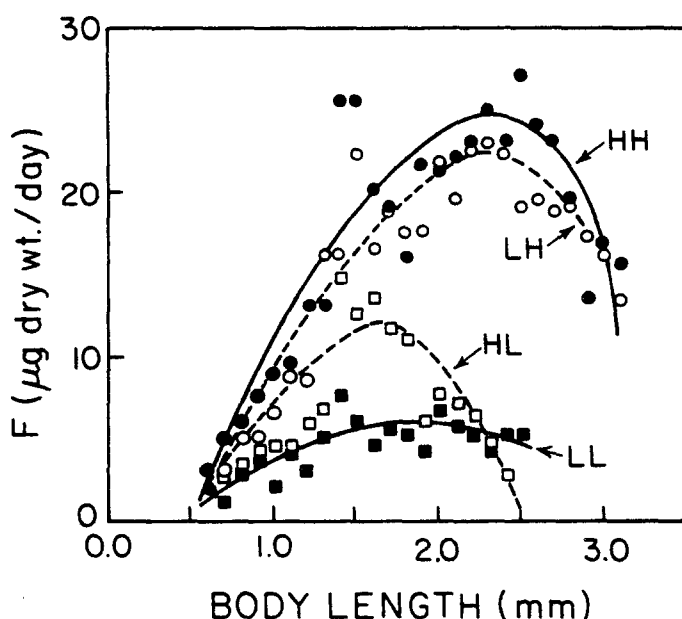


FIG. 3. Mean values of net energy intake,  $F$ , for 0.1 mm size classes for the four lines fitted by eye.

energy intake invested in reproduction by individuals of different sizes. Defining reproductive effort (RE) as the dry weight invested in eggs per instar + the total investment in eggs and growth per instar, the size-specific patterns are virtually identical for all four treatments (Fig. 4). Divergences from the general pattern only appear in those large size classes that occur late in life when survivorship is falling.

These results suggest that size-specific, relative allocation of resources to reproduction is an extremely conservative character for this *Daphnia pulex* clone. However, while reproductive effort has almost always been defined as above or in a very similar manner by ecologists, such a definition does not adequately portray the costs of reproduction for organisms with different energy intakes. A 50% investment in reproduction has much

TABLE 1. CHI-SQUARE COMPARISON OF AGE-SPECIFIC SURVIVAL.

| Age<br>(days) | LL vs. HL | HH vs. LH | HL vs. LH | LL vs. HH |
|---------------|-----------|-----------|-----------|-----------|
| 10            | 0.01      | NS        | NS        | 0.01      |
| 20            | 0.01      | NS        | NS        | 0.01      |
| 30            | 0.01      | NS        | NS        | 0.01      |
| 40            | NS        | 0.01      | NS        | 0.01      |
| 50            | 0.01      | NS        | 0.01      | 0.01      |
| 60            | —         | NS        | —         | NS        |
| 70            | —         | NS        | —         | NS        |
| 80            | —         | NS        | —         | NS        |

Chi-square comparison of  $l_x$  between various treatments. NS indicates  $p > 0.05$ .

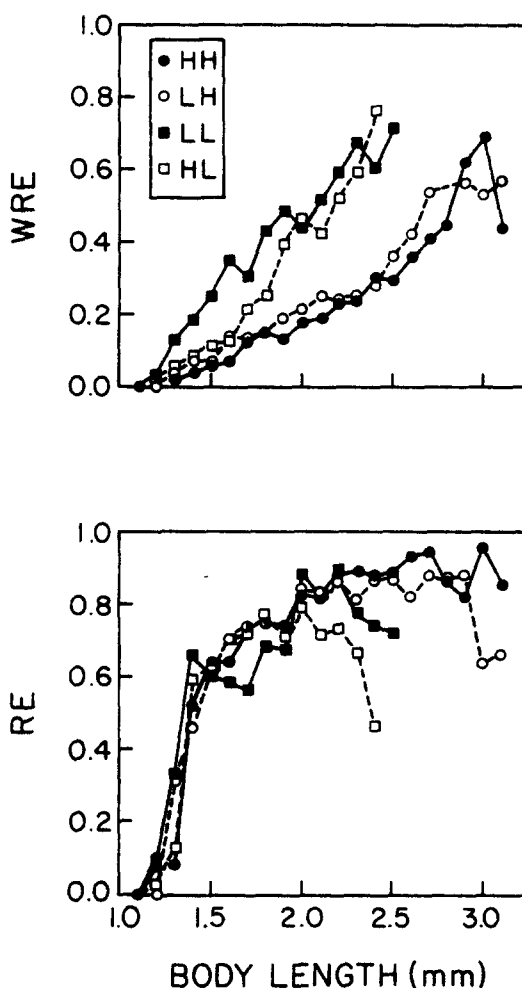


FIG. 4. Mean values of reproductive effort (RE) and weighted reproductive effort (WRE) for 0.1 mm size classes for the four lines.

more significant consequences for the growth and maintenance of a poorly nourished individual than for a well-fed individual (for whom the 50% remaining for growth and maintenance will be much more substantial in absolute terms). This interpretative problem can be rectified by weighting the standard reproductive effort expression (RE) by the absolute investment in growth so that the new measure will give progressively lower values for individuals with equivalent RE but increasing total energy intakes,

$$\text{WRE} = \frac{R}{(1 + G)(R + G)} = \frac{1}{1 + G} \cdot \text{RE}$$

where G and R represent the dry weight investments in growth and reproduction per unit time. Note that WRE = RE only when G = 0.

When viewed in this manner, it becomes apparent that the costs of reproduction are much more severe for the low food lines and become increasingly more so as the individuals get larger (Fig. 4). Although the weighted reproductive effort function is virtually identical for both high food lines irrespective of maternal environment, there are important differences between the low food lines. While WRE was consistently higher for the LL line than for the high food lines, the HL line started out life with low WRE values similar to the high food lines, and then after the formation of the first two clutches rapidly converged on (but did not surpass) the high WRE values of the LL line. Thus, neither the RE nor WRE sets of data support the hypothesis that the elevated mortality of the HL line is a consequence of an elevated cost of reproduction. Moreover, the general absence of significantly negative correlations between WRE and longevity of individuals within lines (Table 2) indicates that variation in life-span within lines is not a function of variation in reproductive effort.

A closer examination of the total size-specific rates of investment in growth and reproduction suggests an alternative mechanism.  $F$  increases and then decreases with size (and age) in all four lines, but the peak comes exceptionally early and the subsequent decline is exceptionally severe in the HL line (Fig. 3). A negative energy budget is indicated for HL individuals larger than 2.5 mm, precisely the size at which the precipitous increase in mortality occurred in this line. This suggests that rather than being a consequence of reproductive effort, the reduced longevity of the HL line resulted from a general breakdown in the physiological capabilities of large individuals. Similar but later and less severe declines in  $F$  for the three longer-lived lines suggests that this may be a general mechanism of senescence in this organism.

Further evidence that the reduced longevity of the HL line is related to its early decline in net energy intake derives from the strong positive phenotypic correlations between  $F$  in instars 6–10 (~13–24 days) and 11–15 (~25–36 days) and longevity of individuals within this line (Table 2). These correlations were not significant for any of the other lines, but a significant positive correlation did exist between longevity and  $F$  at a later age (instars 16–20, ~37–48 days) in the HH line. The failure of the LL and LH lines to show similar correlations through the 20th instar does not appear to be an artifact of small sample sizes since these were similar to those for the HH line. However, the decline in  $F$  was mildest for these two lines, and it is possible that the correspondence between  $F$  and longevity does not arise until later in life when our sample sizes were prohibitively small.

Several differences in phenotypic correlations between the lines indicate that slight differences in parental investment at the egg stage alone can have profound and long-lasting effects on the organization of genome expression in these organisms. For instance, there is a highly significant negative correlation between age at maturity and longevity and positive relation between juvenile growth rate and longevity in the HL line, but no such significant correlations in the other lines (Table 2). Only the LL line exhibits a significant (positive) correlation between progeny production per day and longevity, and only individuals whose mothers were raised on low food exhibit a correlation between size at birth and longevity.

A major question that remains concerns the extent to which the mortality schedules of the different lines are set by events early in post-embryonic development. In particular, are the HL lines committed to an early death by the time juvenile development has been completed because of irreversible physiological and morphological modifications of the phenotype, or can subsequent modification of the environment promote greater longevity?

TABLE 2. CORRELATION WITH LONGEVITY.

| Line | Size at Birth | Age at Maturity | Juvenile Growth Rate | Net Energy Intake |        |        |        | Weighted Repro. Effort |       |       |       |
|------|---------------|-----------------|----------------------|-------------------|--------|--------|--------|------------------------|-------|-------|-------|
|      |               |                 |                      | 1                 | 2      | 3      | 4      | 1                      | 2     | 3     | 4     |
| HH   | 0.11          | 0.17            | -0.23                | -0.17             | 0.08   | 0.28   | 0.65** | -0.39*                 | -0.33 | -0.22 | -0.12 |
| HL   | 0.26          | -0.47**         | 0.53**               | -0.20             | 0.64** | 0.59** | -0.23  | -0.29                  | 0.43* | 0.05  | 0.58  |
| LL   | 0.49**        | -0.16           | 0.17                 | 0.39              | 0.40   | 0.30   | 0.18   | 0.11                   | 0.23  | -0.01 | 0.37  |
| LH   | 0.43*         | -0.02           | 0.05                 | 0.13              | -0.19  | 0.07   | -0.34  | 0.10                   | -0.13 | -0.31 | -0.30 |

Correlation coefficients (r) between life span and other life history parameters within the four lines. \*\* and \* indicate significance at the 0.01 and 0.05 levels. Net energy intake and weighted reproductive effort values were squared prior to the regression analysis in order to normalize them.



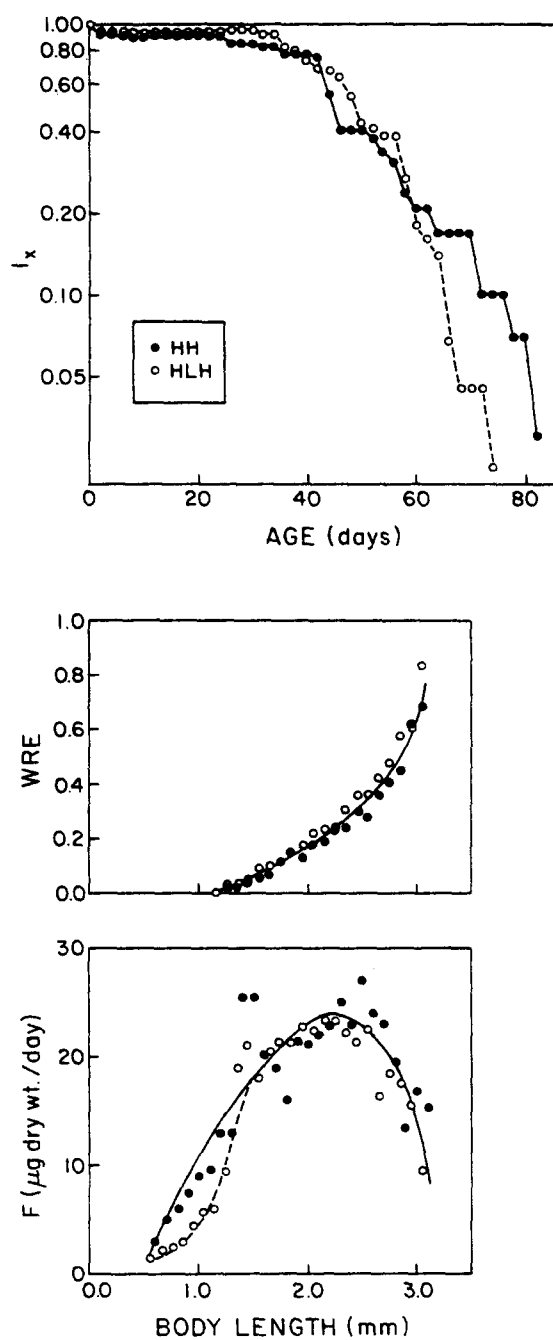


FIG. 5. A comparison of the HLH and HH lines for age-specific survival ( $l_x$ ) and size-specific weighted reproductive effort (WRE) and net energy intake ( $F$ ). Chi-square analysis revealed that there were no significant differences in survivorship at the 0.05 level.

We examined this problem by initiating another HL line under identical conditions to the first except for switching all individuals back to high food on their seventh day (approximately their age at maturity).

Figure 5 illustrates that the survivorship schedule of this new line (designated HLH) is statistically indistinguishable from that of the HH line. The size-specific patterns of WRE for the HLH and HH lines are also indistinguishable, and the patterns of net energy intake for the two lines converge following the switch in food regimes for the HLH individuals. Thus, the manifestation of a maternal influence is not simply a function of an individual's setting during its immature phase; it may be highly modified by events occurring after maturation. At the very least then, any prediction of the life-span of an individual requires information on its maternal, pre-reproductive and reproductive environments.

## DISCUSSION

We have not established the molecular basis of the maternal effects observed in this study. Goulden and Hornig, 1980 have pointed out the beneficial effects that lipid storage may have on a daphnid's ability to survive during periods of low food availability and have suggested that transfer of lipid stores through eggs may extend the benefit to the progeny. However, our results indicate that maternal stores do much more than simply enhance juvenile survival. Under limiting food conditions, individuals that are endowed with a supplemental store of energy and/or nutrients at the egg stage exhibit an enhanced incorporation of energy into early growth and reproduction far in excess of what can be directly attributed to maternal stores. Moreover, the physiological and/or morphological consequences of maternal investment that increase progeny fitness early in life can be associated with a substantial decrease in survivorship of those same progeny later in life (the HL line). Thus, depending on the environmental setting of the progeny, a small increment in maternal investment at the egg stage alone can result in a radical modification of offspring phenotype.

Since all of the *Daphnia* in this study shared identical genomes, it seems most likely that the observed differences between the four lines are products of the same gene complexes, the outward expressions of which are molded by environmental circumstances. It is possible that the divergence between the HL and LL lines resulted from the expression of different genes whose translation or transcription depended upon their early (embryonic) environmental setting, but the extreme similarity of life histories of the HH and LH lines argues against this. Therefore, our discussion will focus on nongenetic causes of variation in longevity.

Our results are clearly inconsistent with the prominent "cost of reproduction" hypothesis for longevity (Williams, 1966; Calow, 1979). The projected cost of reproduction was exceptionally high for the LL line throughout life, yet its maximal life-span was identical to that of the HH and LH lines. The cost of reproduction for the HL line was relatively low early in life and only approximated that of the LL line later in life, but at the point when all of the HL individuals had died, 25% of the LL individuals were still alive. Finally, of 16 regressions between age-specific WRE and life-span made within lines, only two are significant, one of which is positive (Table 2).

As it is rather loosely defined, the "rate of living" hypothesis for aging does not adequately explain our results either. First suggested by Pearl (1928), the idea that an en-

vironmental reduction of the growth and/or maturation rate will increase longevity is consistent with numerous studies (Ingle, 1933; McCay and Crowell, 1934; McCay *et al.* 1939, Lints and Lints, 1971; Kent, 1981), although cause and effect have not been discriminated in most cases. In our study, however, the relation between juvenile growth rate and life-span within the lines was insignificant except in the case of line HL for which there was a significantly positive correlation (Table 2). Moreover, when all four lines are compared, a strong positive, rather than negative, correlation between developmental rate and mean longevity is revealed (Fig. 6), a pattern which nonetheless can be completely obviated by modification of the environment following maturation (cf. the HLH line).

Our results are of particular interest when compared to a similarly designed experiment performed by Ingle *et al.*, 1937. They determined life tables for a clone of *Daphnia laevis* on a manure infusion medium (their "well-fed" conditions) and on the same medium diluted 36-fold with pond water (their "starvation" conditions). (Their low-food individuals were actually reproductively active and not really starving to death.) Since both lines were started from "large broods produced by healthy vigorous mothers" (presumably under "well-fed" conditions), they would seem to be analogous to our HL and HH treatments. Yet contrary to our results, Ingle *et al.*'s, 1937 "HL" lines survived longer than their "HH" lines both in terms of mean (38 vs. 28 days) and maximum (51 vs. 46 days) longevity (Fig. 8). A similarly designed experiment later performed with the same clone (Fig. 8) gave comparable results (Dunham, 1938).

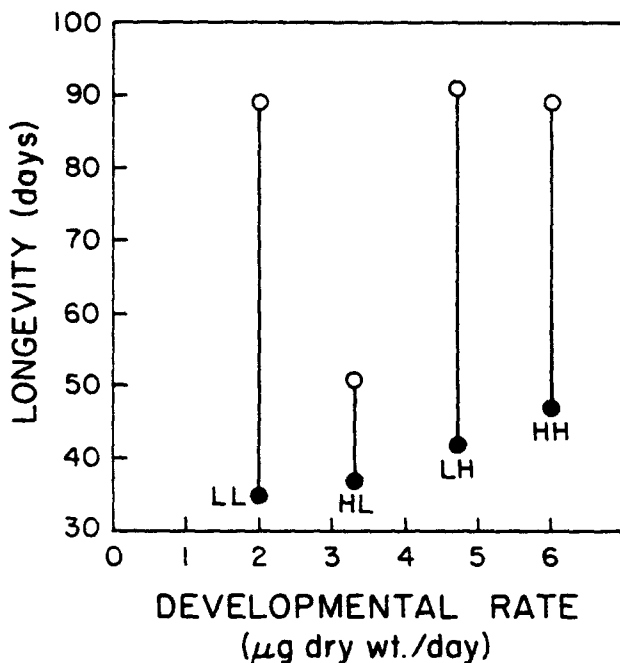


FIG. 6. Mean (solid points) and maximum (open points) life spans and developmental rates for the four lines. Maximum life span was taken to be the age at which  $I_x = 0.01$ . Developmental rate is equal to the difference in dry wt. between the sizes at maturity and birth divided by the age at maturity.

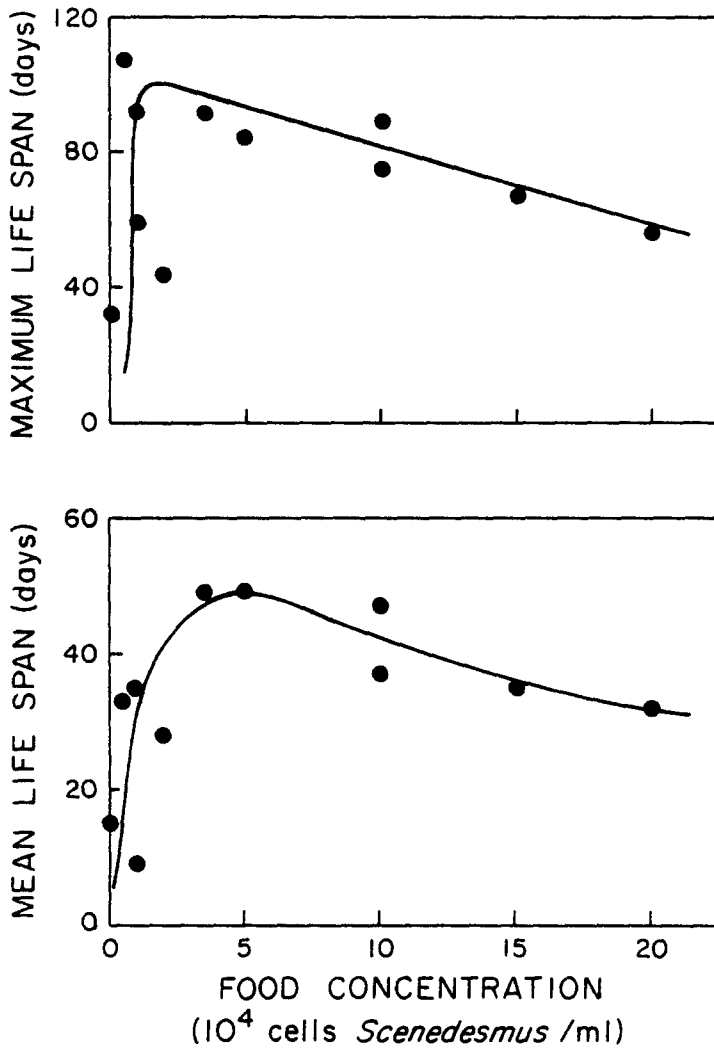


FIG. 7. Mean and maximum longevity as a function of food availability for the *Daphnia pulex* clone used in this study under conditions of identical maternal and progeny environments. Data points include the LL and HH lines of this study as well as nine other lines of 50 individuals each from earlier unpublished work. Maximum life span is the extrapolated age at which  $I_x = 0.01$ . Food concentration is given as cell density of *Scenedesmus*, but *Chlamydomonas* were also present at one-fifth the *Scenedesmus* level in all cases. Curves fitted by eye.

Rather than being a consequence of a true biological difference between experimental organisms, the discrepancy between our results and those of Ingle *et al.* and Dunham may be an artifact of scale reflecting the non-linear response of longevity to food concentration. In our *Daphnia pulex* clone both mean and maximum life span gradually increase with decreasing food availability until a critical food concentration is reached at which point longevity rapidly declines to zero (Fig. 7); data for other daphnids (Frank *et al.*, 1957; Vijverberg 1976; Porter and Orcutt, 1980) are consistent with this pattern. Thus, the relative life-spans of lines grown on different food concentrations depend on the positions

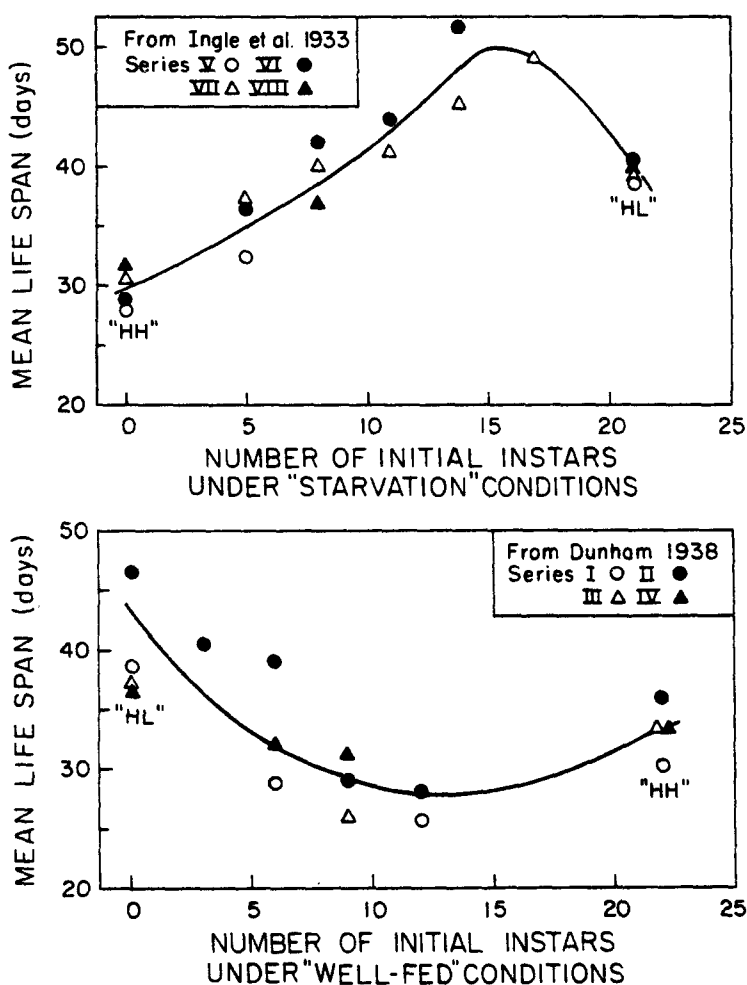


FIG. 8. Mean longevity for lines of a *Daphnia laevis* clone with various schedules of high and low food throughout life. Each point refers to a line of multiple individuals; different series refer to different sets of experiments. Curves fitted by eye.

of those concentrations relative to the optimum for longevity. Our low and high food concentrations straddled the optimum so that the LL line was low on the mean life-span scale relative to the HH line but nearly identical to the HH line on the maximum life-span scale. Although there is no way of translating the food of Ingle *et al.*, 1937 and Dunham, 1938 onto our scale, it is clear that if both their "well-fed" and "starvation" food conditions were in excess of the optimum, a comparison of "HH" and "LL" lines would have shown the latter to have greater longevity. They of course followed an "HL" and not an "LL" line, but the different responses to HL treatment that arose in our studies may be a consequence of the relative positions of H and L foods as well.

One point on which we are in agreement with Ingle *et al.*, 1937 and Dunham, 1938 as well as with other investigators (McCay 1952; Ross *et al.*, 1976; Driver and Cosopodiotis, 1979; Weindruch and Walford, 1982) is that the age of onset of senescence is highly sen-

sitive to modification by temporal patterns of nutritional conditions (not including starvation) within an individual's life. However, our finding that the longevity of an HL line can be elevated by returning the line to high food at an early age is not entirely consistent with Ingle *et al.*'s (1937) results from raising numerous "HLH" lines with the initial L phase of varying length. Their data show that any initial period on low food elevates longevity with respect to pure "HH" lines, but that only relatively late switches result in an enhancement over "HL" lines (Fig. 8). Again this discrepancy may be a consequence of the different relative food concentrations in our experiments.

Of additional interest is Dunham's, 1938 experimentation with "HHL" lines (Fig. 8). His results indicate that any early period of life spent on high food subtracts from longevity compared to an "HL" line, and suggest that the reduction is greatest at some intermediate number of initial instars spent on high food.

Similar complexities revealed by experimental manipulation of temperature schedules in *Drosophila subobscura* (Clarke and Maynard Smith, 1961) led Maynard Smith (1963) to modify the "rate of living" model to a threshold model. He suggested that the rate of aging (the decline in "vitality") is approximately independent of temperature and that variation in life-span is largely a consequence of variation in the environmentally-determined "threshold vitality" necessary for self-maintenance. Subsequent experimentation with *Drosophila* has yielded data that are not entirely consistent with the threshold model (Hollingsworth, 1969; Lamb, 1968). Moreover, a simple threshold model cannot explain why HLH lines can survive longer than either HL or HH lines or why HHL lines do not survive as long as HH or HL lines. Nor can it explain why longevity increases and then subsequently declines with increasing food availability.

Although neither the "rate of aging" nor "threshold" models are independently capable of explaining longevity variation, and although both concepts must be loosely defined in an absence of a detailed understanding of the mechanisms responsible for senescence at the organismal level, a joint consideration of the two ideas is conceptually useful enough to yield predictive models for life span (Lamb, 1977). In the following analyses, we consider the age at death of an individual to be a function of the level of vitality at birth ( $V_0$ ), the rate of decline in vitality (the rate of aging,  $m$ ), and the threshold vitality ( $V_T$ ). For a constant environment the aging function is defined as

$$v(t) = V_0 - mt$$

where  $v(t)$  is the vitality at time  $t$ . The predicted life span ( $T$ ) is the age at which  $v(t)$  declines below  $V_T$ ,

$$T = \frac{V_0 - V_T}{m} \quad (1).$$

All three terms on the right can be considered to be functions of maternal ( $f_m$ ) and/or offspring ( $f$ ) food supplies.

This simple model immediately helps clarify the conditions necessary to generate a convex relation between life-span and food availability (as in Fig. 7). In a constant environment, mothers and daughters are exposed to identical food concentrations, i.e.,  $f = f_m = \hat{f}$ ,

and the behavior of  $T(\hat{f})$  can be examined by taking the first derivative of (1),

$$\frac{dT}{d\hat{f}} = \frac{m \cdot \frac{dV_o}{d\hat{f}} + \left\{ -m \cdot \frac{dV_T}{d\hat{f}} - (V_o - V_T) \cdot \frac{dm}{d\hat{f}} \right\}}{m^2}$$

It seems reasonable to assume that vitality at birth is an increasing function of food availability, i.e.,  $dV_o/d\hat{f} > 0$ , particularly since egg volume increases with food availability. Thus, since both  $-m$  and  $-(V_o - V_T)$  must be negative, the only way for  $T$  to first increase and then decrease with  $\hat{f}$  (i.e., for  $dT/d\hat{f}$  to change sign) is for  $dV_T/d\hat{f}$  and  $dm/d\hat{f}$  to be of opposite sign. For  $T(\hat{f})$  to be convex, the rate of aging must increase and the threshold vitality decrease with increasing food level, or vice versa. We can eliminate those mechanisms that simultaneously increase or decrease  $m$  and  $V_T$  as primary determinants of  $T(\hat{f})$  in environments of constant food availability.

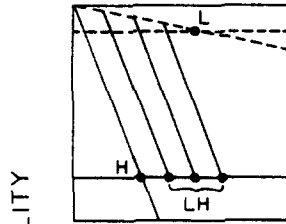
The question then remains as to whether it is the rate of aging or the threshold that increases with food level. Some insight into this problem can be gained from a comparison of the model's predictions with the results of Ingle *et al.* (1937) and Dunham (1938). Since all of their experimental lines were initiated with mothers grown under identical conditions,  $V_o$  is a constant, and the differences between lines are entirely functions of the progeny's food schedule, i.e., we can exclude maternal effects as a source of between-lines variance. Fig. 9 graphically portrays the expected patterns of longevity variation in "HLH" and "HHL" lines for the cases in which  $dm/df > 0$  and  $dV_T/df < 0$  (Model I) and  $dm/df < 0$  and  $dV_T/df > 0$  (Model II). We see that Model II predicts a decline and subsequent increase in mean longevity as the initial L phase is extended in HLH lines and an increase and subsequent crash in mean longevity as the initial H phase is extended in HHL lines; both predicted patterns are inconsistent with the data of Ingle *et al.* (1937) and Dunham (1938). Model I, on the other hand, generates expected patterns that are qualitatively similar to both sets of data in Fig. 8. This suggests that the rate of aging increases while the threshold vitality declines with increasing availability of food to individuals ( $f$ ). One possibility is that somatic mutations, metabolic oxidants, autoimmunity and/or some other precursor to cellular damage increases with increasing food availability and the resultant increase in cellular metabolism and mitotic activity, while the tolerance of the organism to specific levels of such damage (i.e., reductions in "vitality") also increases.

This analysis tells us nothing about the mechanistic relation between maternal food availability and an individual's longevity; we cannot simply assume the qualitative effects of  $f_m$  and  $f$  to be the same. Even our own data which clearly indicate that the maternal environment plays an important role in determining an individual's life-span are insufficient to verify the underlying mechanism. In fact, the patterns observed in Fig. 7 could actually be a consequence of  $dm/d\hat{f} < 0$  and  $dV_T/d\hat{f} > 0$  if the underlying effects of maternal investment were opposite to and of greater significance than those of the progeny environment. An examination of (1) demonstrates that  $\partial V_o/\partial f_m > 0$ ,  $\partial V_T/\partial f_m < 0$  and  $\partial m/\partial f_m < 0$  could all be involved in the enhancement of mean longevity that we found with increased maternal investment (Fig. 6). Indeed, one or two of these effects could actually be of opposite sign, and the pattern in Fig. 6 still emerge, if the remaining effects were of substantial magnitude and in the appropriate direction.

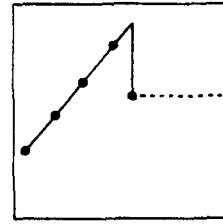
Some insight into this matter may be gained from considering the mean and maximum

HLH LINES

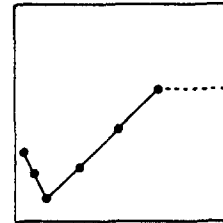
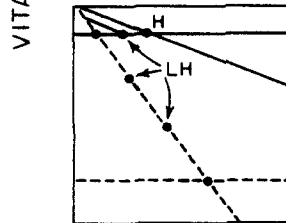
Model I:  
 $dm/df > 0$   
 $dV_T/df < 0$



LONGEVITY



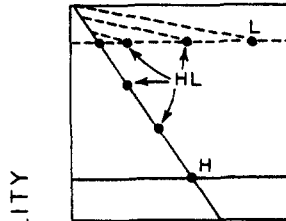
Model II:  
 $dm/df < 0$   
 $dV_T/df > 0$



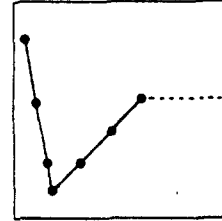
INSTARS

No. of Initial Instars  
on Low FoodHHL LINES

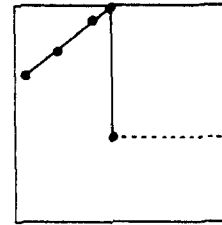
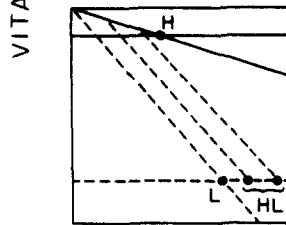
Model I:  
 $dm/df > 0$   
 $dV_T/df < 0$



LONGEVITY



Model II:  
 $dm/df < 0$   
 $dV_T/df > 0$



INSTARS

No. of Initial Instars  
on High Food

FIG. 9. Expected longevity for lines with various schedules of low and high foods during their lives graphically analyzed with a "rate of aging-threshold vitality" model. Two models are given as explained in the text, and the analysis is applied to lines that are grown on low food early in life and then switched to high food (HLH) and vice versa (HHL). Graphs on the left illustrate the analysis: diagonal lines represent the decline in vitality with age, horizontal lines represent the threshold vitality below which death occurs; dashed lines refer to low food conditions, solid lines to high, and solid points to age at death; L and H lines spend their entire lives on low and high foods respectively, LH and HL have food regimes altered at some point during their lives. Mortality occurs when vitality falls below the threshold for the environment in which the individual is currently living.



longevities of the four lines in our experiment (Fig. 6). For both the low and high lines, increased maternal investment increased the mean but decreased the maximum progeny longevity as a consequence of a decrease in the variance of longevity. An increase in  $m$  should tend to synchronize the age at death for a cohort of individuals as they will all cross the vitality threshold in a narrow range of time, and a simultaneous increase in the level of vitality at birth could increase the mean while decreasing the maximum longevity (Fig. 10). Although we are not yet able to determine whether the threshold vitality is also a function of  $f_m$ , we tentatively conclude that an increase in maternal investment enhances the quality of offspring at birth ( $V_0$ ) while simultaneously altering the rate of aging perhaps in a fundamentally similar manner as  $f$ .

It is important to keep in mind that the model presented above is intended to explain variation in physiologically determined longevity. It is not meant to explain the life-span of individuals in many natural populations that are exposed to selective agents (such as predators) that can impose mortality prior to the depression of "vitality" below the physiological threshold. Moreover, it is perhaps premature to generalize our experimental findings to other organisms. For instance, while the results of Robertson and Salt (1981) using the rotifer *Asplanchna girodi* are qualitatively similar to the results of Ingle *et al.* (1937) and Dunham (1938) for mid-life shifts in food availability, the results of their maternal effects experiment are fundamentally different from our own—an enriched maternal environment either had no effect or a deleterious effect on mean life-span. While ex-

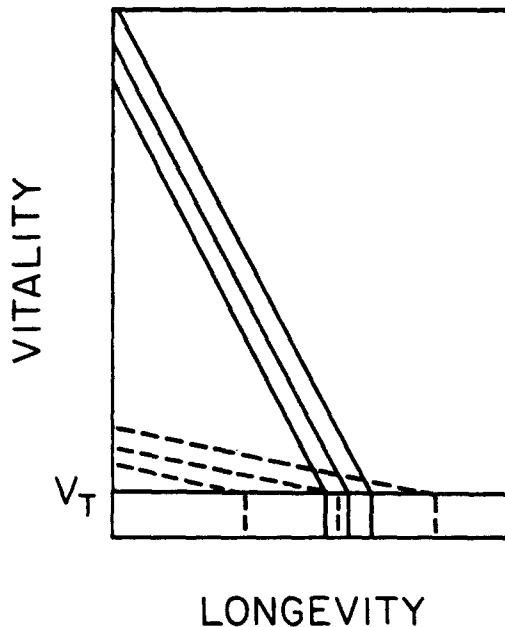


FIG. 10. Mean and range of expected life spans for two lines grown on identical food supplies but with different maternal effects. Dashed lines represent individuals whose maternal environment was low food, solid lines represent high food maternal environments. It is assumed that an increase in the maternal food supply improves the vitality at birth but increases the rate of ageing. An individual's life is terminated when its vitality declines below the threshold,  $V_T$ .

perimentation with rats, mice and hamsters (McCay 1952; Stuchlíková *et al.*, 1975) is in complete agreement with the *Daphnia* data that indicate that dietary restriction early in life followed by complete diet is especially conducive to long life-span, results with rodents also indicate that the initiation of dietary restriction late in life enhances longevity (Stuchlíková *et al.*, 1975, Weindruch and Walford, 1982) contrary to the results of Dunham, 1938. It remains to be seen whether these discrepancies are a consequence of variation in the mechanisms of aging and/or evolutionary strategies of phylogenetically diverse organisms or simply artifacts of employing food concentrations with fundamentally different locations with respect to the longevity-food availability relation. For instance, both of the food concentrations used by Robertson and Salt (1981) were to the right of the peak of  $T(\hat{f})$  while our two concentrations straddled the peak. Future work involving several food concentrations will help resolve this problem.

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