Evolution, 38(2), 1984, pp. 300–313

MEASURING THE COST OF REPRODUCTION. I. THE CORRELATION STRUCTURE OF THE LIFE TABLE OF A PLANKTON ROTIFER

GRAHAM BELL
Biology Department, McGill University, Montreal, Québec H3A 1B1, Canada

Received December 27, 1982. Revised June 22, 1983

The Cost Hypothesis

The foundation of the modern theory of life histories is the hypothesis of reproductive cost. If an increment in present reproduction is associated with a decrement in the expectation of future reproduction, because either future survival or future fecundity is reduced, then age-specific reproduction can be optimized under natural selection. In the absence of such an association, selection will merely favor the maximization both of reproduction and of survival. Reproductive cost is not a sufficient condition for the evolution of intermediate levels of reproduction, since some cost curves favor the greatest possible rate of reproduction or of survival. However, it is a necessary condition: it is not possible to optimize the life history unless some cost of reproduction exists. The first clear enunciation of this principle by G. C. Williams (1966*a*, 1966*b*) was followed by a surge of theoretical work in which a negative relationship between present and future reproduction was used to compute optimal values of reproduction and survival under different circumstances (e.g., Gadgil and Bossert, 1970; Schaffer, 1974; Charlesworth and Leon, 1976; Bell, 1980).

While cost-based theories have been rather successful in predicting patterns among life histories, their foundation has received little rigorous investigation: the cost principle is so eminently plausible that its status as a scientific hypothesis—as the specification for the design of an experiment whose result cannot be foreseen with certainty—seems almost to have dropped out of sight. It is only recently that reports of experiments designed specifically to detect reproductive cost have begun to appear in substantial numbers, and the results of these experiments have been by no means unequivocal.

There are several categories of evidence, on the face of it offering support for the cost hypothesis, which must be rejected because they do not bear on the question, or bear only indirectly. In the first place, the hypothesis posits a negative relationship between present and future reproduction, between individuals within a population (or equivalently between the effects of alternative genes). Among the predictions of the theory based on this premise may be a negative relationship between, say, the fecundity and survivorship of different species or of different populations of the same species. But if these predictions are used as the assumptions on which the theory is built, the argument becomes circular. The common observation that fecundity and survivorship are negatively related when different species (e.g., Tinkle, 1969) or populations (e.g., Leggett and Carscadden, 1978) are compared cannot, therefore, be used as evidence supporting the cost hypothesis itself. Secondly, the cost hypothesis requires only a direct relationship between present and future reproduction. The causation behind this relationship is doubtless indirect, but it is the existence and not the mechanism of cost which is directly at issue. The many studies which demonstrate, for example, an association between increased reproduction and decreased growth, however suggestive, are not directly rel-

TABLE 1. A digest of attempts to test the cost hypothesis directly in *Drosophila*.

Variable 1	Variable 2	Correlation	Notes	Authority
Longevity	early fecundity	<0	large cage popn.	Rose and Charlesworth, 1981*a*
	later fecundity	variable	large cage popn.	Rose and Charlesworth, 1981*a*
Longevity	fecundity	<0	inbred line	Gowen and Johnson, 1946
Longevity	fecundity	$\gg 0$	highly inbred	Giesel, 1979
Longevity	male mating activity	<0	manipulative, new strain	Partridge and Farquhar, 1980
Viability	fecundity	>0	involved complete sterility	Temin, 1966
Viability	fecundity	>0	new mutations	Simmons et al., 1980
Viability	development rate	~ 0	new mutations	Mukai and Yamazaki, 1971
Fecundity	development rate	<0	for high-fitness chromosomes	Hiraizumi, 1961
		>0	for low-fitness chromosomes	

evant, even though decreased growth might be held a priori to imply decreased future fecundity. Moreover, it is crucial that causality should flow from present to future reproduction, rather than that some common factor should cause both present and future reproduction. It is very easy to keep one culture on the laboratory bench and another in the refrigerator, observing that those in the refrigerator reproduce less and survive better; this is not evidence for reproductive cost. Experiments such as those described by Calow and Woollhead (1977) or by Hirshfield (1980), in which a negative effect of reproduction on survival was mediated by temperature or ration, do not, therefore, provide the definitive evidence we seek.

The best single source of directly relevant evidence is the *Drosophila* literature, from which I have abstracted a number of studies in Table 1. The best of these studies has been published recently by Rose and Charlesworth (1981*a*), who found a negative correlation between longevity and early fecundity, although the correlation between longevity and later fecundity is not consistently either positive or negative. An associated selection experiment (Rose and Charlesworth, 1981*b*) is difficult to interpret, but provides some indirect evidence of a negative response of longevity to selection for increased fecundity. Gowen and Johnson (1946) also found a negative correlation between survival and fecundity, but Temin (1966), Giesel (1979) and Simmons et al. (1980) all found that survival and fecundity were positively correlated. Rose and Charlesworth (1981*a*) point out that these latter studies used new mutations (Simmons et al., 1980) or highly inbred lines (Giesel), or else involved complete sterility (Temin). They may therefore reveal little more than the existence of unconditionally disadvantageous alleles, while experiments with larger or less inbred cultures generally support the cost hypothesis. It may be particularly significant that Hiraizumi (1961) found a quadratic relationship between fecundity and development rate, with high-fitness chromosomes showing a negative and low-fitness chromosomes a positive correlation between the two variables. However, the difficulties of interpreting such experiments mirror the difficulties encountered in experiments where the fitnesses of chromosomal

homozygotes and heterozygotes are compared (review in Lewontin, 1974), and are unlikely to be resolved in any simple fashion.

Several papers have appeared recently in which the effect of fecundity on survival has been studied in natural populations of birds. Bryant (1979) found that the survival rate of single-brooded female *Delichon urbica* exceeded that of double-brooded females, though there was no effect among the males. In this, as in most other bird studies, "survival" is measured as a return rate, which is satisfactory only to the extent that the birds are known to return faithfully to the same breeding site. In *D. urbica,* according to Bryant, males appear to be faithful but females may not be, and this difference might contribute to his result. Hogstedt (1981) found that the survival of *Pica pica* (males and females pooled), measured as the number of breeding seasons completed, actually increased with mean brood size. However, the obvious explanation of his data, that brood size increases with age, was ruled out on the basis of a sample of only seven individuals. Neither Kluijver nor Perrins (both cited by Lack, 1966) found any relationship between brood size and subsequent female survival in *Parus major.* The most satisfactory data of this kind was obtained by Smith (1981) for *Melospiza melodia* on Mandarte Island, B.C., where the population is known to be almost wholly faithful. He found that surviving females made the same number of nesting attempts and reared larger clutches than females which subsequently died, in direct contradiction to the predictions of the cost hypothesis.

There appear to be very few comparable studies of mammals. Fairbairn (1977) has shown that female deer-mice (*Peromyscus maniculatus*) which breed early in the spring have much greater mortality immediately afterwards than females which delay reproduction until the summer.

Other studies have manipulated the clutch size of birds in order to demonstrate a cost. In *Ficedula hypoleuca,* Askenmo (1979) found that males rearing experimentally enlarged clutches survived less well than controls. The result was not quite unequivocal, however, because of the wide variation in the survival of controls in different years, and because it is not clear whether the controls received the same handling as the experimental broods. (Campbell, in Lack, 1966, found that brood size and the survival rate of females were uncorrelated in unmanipulated populations of this species). De Steven found no correlation between reproduction and subsequent survival in a similar experiment with *Iridoprocne bicolor,* but her sample size was very small. Kluijver (1971) removed a large proportion of fledgling *Parus major* from a population during a series of years, and observed a subsequent increase in the survival of females. At the same time, however, management techniques were changing his study sites drastically, and such unreplicated, uncontrolled experiments involving confounding temporal heterogeneity are impossible to interpret rigorously.

The evidence from birds is summarized in Table 2. Clearly, it does not constitute solid, unarguable support for the cost hypothesis.

Direct observational evidence from organisms other than *Drosophila* and birds is very scanty, but two recent papers claim to provide it. In a widely-cited article, Snell and King (1977) show a negative relationship between survival and fecundity in the rotifer *Asplanchna brightwelli.* In particular, the regression of mean fecundity (at age x) and mean survivorship (from x to $x + 2$) has negative slope. The statistical significance of this effect seems doubtful, and its biological interpretation is complicated by the lack of any relationship between fecundity and survivorship between ages x and $x + 1$ or between ages x and $x + 3$. Moreover, the result refers only to an unspecified fraction of the data available from the experiment they performed, and although the authors comment that a

TABLE 2. A digest of attempts to test the cost hypothesis directly in birds.

Species	Sex	Correlation between survival and fecundity	Manipulation	Authority
Pica pica	M + F	>0	no	Hogstedt, 1981
Delichon urbica	M	~0	no	Bryant, 1979
	F	<0	no	Bryant, 1979
Ficedula hypoleuca	M	<0	yes	Askenmo, 1979
	F	~0	no	Campbell (in Lack, 1966)
Iridoprocne bicolor	F	~0	yes	De Steven, 1980
Parus major	F	~0	no	Kluijver (in Lack, 1966)
	F	? <0	yes	Kluijver, 1971
Melospiza melodia	M + F	>0	no	Smith, 1981

"similar but weak negative relationship was found in other age-classes and clones tested," no measures of significance are available. More generally, the partial correlation between longevity and the rate of production of offspring was found to be negative, with long-lived individuals having low age-specific fecundity. This effect can arise, however, as a consequence of the deterministic mode of reproduction general in rotifers, with the total number of oocytes being fixed early in development; if postreproductive life is short, as it is in *Asplanchna,* then longevity and the rate of egg production are negatively autocorrelated (see Bell, 1983*b*). Law (1979) found that increased present reproduction was associated with a decline in both subsequent survival and fecundity in the grass *Poa annua.* In both cases the regressions given have enormous scatter, but are likely to be formally significant (the statistics given by Law are unclear). Again, however, the result depends on the particular comparison made: survival is negatively related to the number of inflorescences produced during the first four to five months of life, but not to the number produced during the whole of the first year. Moreover, this is again an unreplicated and uncontrolled experiment performed in a varying environment. It is possible that the first year happened to suit one type of plant and the second year another, with both types reproducing to their limit in both years. It is also possible that plants which in any one year were investing less in sexual were investing more in asexual reproduction, and vice versa.

In recent years, a number of papers have described the effect on parental fitness of manipulating parental care or access to mates in invertebrates. Partridge and Farquhar (1981) showed that the survival of male *Drosophila* was reduced by exposure to virgin but not by exposure to inseminated females. Brown (1982) found no difference between the lifespans of mated and unmated *Artemia* at high food levels, but a greater mortality of the mated females (though not males) at low food levels. For the mated females there was a nonsignificant negative correlation between mean daily fecundity and lifespan at low food levels and a significant positive correlation at high food levels, possibly arising as an artifact of the increase in fecundity from the early to the middle part of reproductive life. Virgin female copepods (*Mesocyclops*) lived longer than mated individuals (Feifarek et al., 1983), though the correlation between longevity and total egg production or initial clutch size was not significant. Dean (1981) again found that virgin female *Melanoplus* lived longer than mated females, but in this grasshopper virgins have the same rate of egg production as mated females! It is possible that all these experiments demonstrate an effect of mating (see Daly, 1978) rather than of reproduction per se.

Tallamy and Denno (1982) manipu-

lated maternal care in the tingid bug *Gargaphia,* where continued oviposition and caring for eggs are mutually exclusive activities. They found that longevity increased as the period of maternal care permitted in the treatments increased, demonstrating that alternative modes of reproduction have different effects on mortality. Since females which care for their young produced more surviving offspring than those which do not, this result implies a positive correlation between components of fitness. Moreover, Tallamy and Denno found no relationship between increased egg production early in life and subsequent fecundity or survival in any of their treatments, except one which yielded a positive correlation between initial and subsequent clutch size.

The cost hypothesis is thus far less strongly supported than its importance, and its frequent assertion, make desirable. The present paper describes the first in a series of experiments designed to provide general and rigorous tests of the hypothesis.

Experimental Design

Experiments which are designed to detect a cost of reproduction may be passive or manipulative. The bulk of reports in the literature concern passive experiments, in which the present reproductive behavior of individuals is merely observed, and related to their subsequent expectation of reproduction. In manipulative experiments, such as Partridge and Farquhar's (1981) work with male *Drosophila,* the fate of a control group is compared with that of a group whose present reproduction has been altered in some specified way. Manipulative experiments are the more satisfactory, not least because the values of the independent variable can be preassigned, but are difficult to perform because it is necessary to perturb present reproduction without exerting any direct effect on future reproduction or survival. Passive experiments are much easier to perform, but require careful control of experimental conditions and—as discussed below—may prove difficult to interpret.

The experiment described here is passive, and thus involves measuring the future reproduction of individuals whose present reproduction varies. It is very desirable in such experiments to control both the genotype and the environment of the experimental organisms. The simplest and the most satisfactory way of controlling genotype is to use an asexual organism in which progeny can be assumed to be identical with one another and with their mother. I have chosen *Platyias patulus,* a medium-sized plankton rotifer whose life cycle comprises a sequence of asexual generations, during which eggs are produced mitotically, punctuated by occasional episodes of sexuality. Under the conditions of culture described below, about 7% of individuals produced sexual eggs; these individuals were excluded from all analyses.

Individual *P. patulus* were isolated from a collection taken from a small pond near St-Eustache, S. Québec, and placed in separate 3-ml cells in new, sterile tissue—culture plates (Linbro 76-033-05). The culture medium was a stock suspension of *Scenedesmus* standardized to 60% transmittance with a Bausch and Lomb Spec-20 spectrophotometer and changed every day. This medium contained bacteria and ciliates, besides larger organisms (a monogonont rotifer and a bdelloid rotifer) which were removed from the experimental cells. To detect any change in the medium during the course of the experiment, the longevity and fecundity of the rotifers were analyzed for the linear effect of calendar date independently of age; no effect significant at $P < .20$ was detected. The experiment was run on the laboratory bench at a temperature of 22 ± 2 C. Offspring of 15 wild-collected founding individuals were isolated as soon as possible (<24 h) after birth and cultured separately in new cells; in turn their progeny and grandprogeny were isolated, until a total of 24 individuals descending from the founding individual had been obtained. In a very few

cases parent and offspring could not be unequivocally identified; both were then discarded. Since the age of the founding individual was not known, it played no part in the experiment other than helping to create a clone of 24 individuals. The age of the founder might nevertheless create variance between clones, since the life histories of offspring are known to be affected by maternal age in rotifers (Jennings and Lynch, 1928; Lansing, 1948). However, only a few individuals in each clone were offspring of the founder itself, the rest being more distant descendents, and neither longevity nor total fecundity varied systematically with the number of generations separating individuals from the founder. Each of the experimental individuals was scrutinized every day and its reproduction and survival noted. The results of the experiment thus comprised the complete life history of 24 individuals (less a few sexual individuals, and a very few others lost during transfer to fresh medium) from each of 15 clones.

The experiment is designed to test the cost hypothesis, which states that present reproduction and expected future reproduction are negatively correlated. Expected future reproduction is determined jointly by future survival and by future fecundity, and we can therefore recognize two components of cost, the survival cost and the fecundity cost. For either component, any increment in present fecundity is held to cause a decrement in future performance. Such an hypothesis cannot be tested for individual organisms (since any individual must be either alive or dead at any given moment), but we can use the genetic system of the rotifer to replicate individuals by asexual reproduction. We can then predict that individuals within a clone which have greater present reproduction will have lower future survival or fecundity; I shall call these the "variable costs." It is conceivable, however, that a negative relationship of this sort could have evolved in the source population, through the selection of alleles expressing this sort of antagonistic pleiotropism. We might then observe no effect of present on future reproduction between individuals within a clone, but a negative relationship between clonal means; I shall call these the "acquired costs." The variable and acquired costs correspond to what other authors (e.g., Rose and Charlesworth, 1981*a*) have called, more precisely, the phenotypic and genetic correlations, but because the genetic difference, if any, between the clones used here is unknown I have avoided so strong a statement.

By comparing individual values within clones, and mean values between clones, we can thus identify the variable survival cost, the acquired survival cost, the variable fecundity cost and the acquired fecundity cost. Pooling individuals from all clones gives a general cost which combines variable and acquired effects.

The Costs of Reproduction

General Life History.—The overall pattern of age-specific survival and fecundity is shown in Figure 1. Survival rates were very high for the first 15 days of life, but thereafter declined more and more steeply, with very few individuals surviving for more than 35 days. Reproduction typically began four or five days after birth, reached a peak at seven to nine days of age, and thereafter declined, with old animals reproducing only very slowly.

If we attempt to detect acquired costs by comparing mean reproduction at some given age with mean expected reproduction at some other age, it is difficult to know which age-classes to choose, out of the very large number of pairwise combinations available. A more general approach is to describe the age-specific survival and fecundity of each clone in terms of functions with a small number of parameters, and then to define the relationships between these functions predicted by the cost hypothesis. Ideally, one would choose to fit functions whose parameters could receive some natural biological interpretation—as representing, for instance, the age at first reproduction. Unfortunately, such functions, though easy

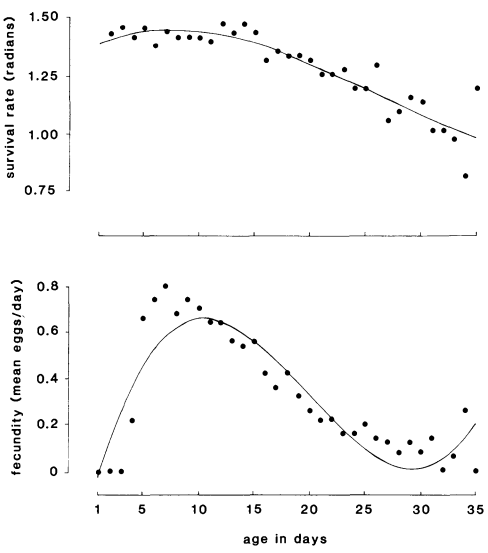

FIG. 1. Diagrammatic summary of the life history of *Platyias patulus* in laboratory culture. Figure 1A is the age-specific daily survival rate. The cubic equation fitted by least-squares has the parameters $s_0 = +1.369 \pm .068$, $s_1 = +.0216 \pm .015$, $s_2 = -.00164 \pm .009$, $s_3 = +.0000205 \pm .000017$; coefficient of determination $r^2 = 0.835$. Figure 1B is the age-specific daily fecundity rate. The cubic equation fitted by least-squares has the parameters $b_0 = -.1949 \pm .088$, $b_1 = +.1825 \pm .021$, $b_2 = -.01165 \pm .0013$, $b_3 = +.000194 \pm .00002$; $r^2 = 0.819$. These parameter values (and the plotted points) are based on mean values at each age for all individuals.

to define, cannot be linearized and are difficult and expensive to fit to data. Instead, I have chosen to use cubic equations, which are quickly, cheaply and reliably fitted using standard statistical routines.

The Acquired Survival Cost.—Cubic equations were fitted to the survival and fecundity schedules of each clone. Age-specific survival $s(x)$ is then described in terms of four parameters, say s_i, where $i = 0$ through 3. An increase in the value of any of these parameters implies an increase in the survival rate at any given age, provided that the other three parameters are held constant, i.e., $\partial s(x)/\partial s_i > 0$ for all i and x. Similarly, $\partial b(x)/\partial b_i > 0$, where $b(x)$ is age-specific fecundity and the b_i are the parameters of the fecundity schedule. According to the cost hypothesis, those clones which have greater mean fecundity will have lesser mean survival, or $ds(x)/db(x) < 0$. The hypothesis can be tested by examining the sign of the 16 partial correlation coefficients which relate the s_i to the b_i; all should be negative. The purpose of this analysis is to avoid the necessity of choosing age-classes for comparison according to a possibly erroneous preconception of where cost effects are to be expected, or alternatively of performing a very large number of pairwise comparisons. Comparing regression coefficients of different degrees should reveal any systematic effect of present on expected future reproduction: for instance, if increased reproduction early in life is associated with a decrease in survival much later in life, then b_1 and s_3 will be negatively correlated. The matrix of partial correlation coefficients is given as Table 3. All of the partial correlation coefficients are positive, and the hypothesis is therefore rejected.

TABLE 3. Test for an acquired survival cost of reproduction. The s_i and b_i are the parameters of the cubic equation relating survival rate and fecundity to age in each clone. Entries in the body of the table are partial correlation coefficients: * indicates $P < .05$. Cost hypothesis predicts that entries should be negative.

	b_0	b_1	b_2	b_3
s_0	+.067	+.168	+.285	+.350
s_1	+.221	+.292	+.369	+.409
s_2	+.511	+.484	+.472	+.461
s_3	+.697*	+.447	+.247	+.133

The Variable Survival Cost.—Let us choose some age x, and for a given clone compute the mean fecundity at age x of individuals surviving to that age, $b(x,x)$. According to the cost hypothesis, individuals with greater fecundity have a lower probability of survival. Therefore the mean fecundity at age x of individuals which survive to age $(x + 1)$ should be lower than the fecundity of those which do not survive. The mean fecundity at age x of those individuals surviving to age $x + 1$ should therefore be less than the mean fecundity of all individuals alive

TABLE 4. Test for a variable survival cost of reproduction. The $b(x,x+z)$ is the mean fecundity at age x of individuals surviving to age $x + z$. Cost hypothesis predicts the slope of $b(x,x+z)$ on z is negative. Autocorrelation prevents test of significance for each clone, but preponderance of positive values is sufficient to falsify hypothesis.

Clone	Slope of $b(x,x+z)$ on z
1	+.024
2	+.028
3	+.022
4	+.015
5	−.025
6	+.007
7	+.277
8	−.022
9	+.002
10	−.008
11	+.004
12	−.014
13	+.011
14	+.002
15	+.017
Pooled	+.0076

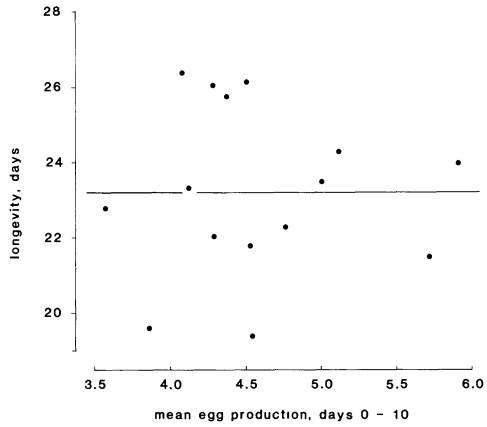

FIG. 2. Simple test for an acquired survival cost of reproduction. Mean total egg production during the first ten days of life is regressed on longevity. The two variables are uncorrelated, the slope of the graph being .000225 and the correlation coefficient 6×10^{-5}. The cost hypothesis predicts a negative correlation, and is therefore rejected.

at age x, i.e., $b(x,x+1) < b(x,x)$. More generally, we expect that $b(x,z) < b(x,x)$ for all $z > x$. The cost hypothesis therefore predicts that the graph of $b(x,z)$ on z should have negative slope. The probability that the observed slope differs from zero only by chance cannot be computed straightforwardly, since successive points are autocorrelated: if by chance $b(x,x+1) < b(x,x)$ then it is likely that $b(x,x+2) < b(x,x)$ also, and so forth. Nevertheless, the cost hypothesis unequivocally predicts the sign of the regression coefficient. Since 11 of the 15 clones give a positive regression coefficient (Table 4), the cost hypothesis is rejected.

Overall Survival Cost.—To reduce the risk of failing to observe some real effect, we can restrict the analysis to a particular age-class, and examine the effect of fecundity on subsequent survival. I have calculated the effect of early fecundity (total egg production up to and including day ten) on longevity (last day of life for individuals surviving at least to day ten). This is predicted by the cost hypothesis to be negative. When the regression is calculated separately for each clone, there are six positive and nine negative results, giving a mean value of $+.107 \pm 1.318$ for the regression coefficient. The regression of clonal means has a value very close to zero (Fig. 2). By pooling the data for all individuals we can measure an overall survival cost which conflates the variable and acquired effects. This has a non-significant positive slope. There is therefore no evidence either for a variable or for an acquired survival cost, supporting the conclusions reached previously.

The Acquired Fecundity Cost.—Since the cost hypothesis predicts a negative relationship between present and future fecundity, we expect that $\partial b_i/\partial b_j < 0$ when $i \neq j$, which can be tested by measuring the correlation between b_i and b_j. The procedure is not straightforward, however, because of the substantial autocorrelation between regression coefficients; in the simplest case of linear regression, for example, there will be a negative correlation between the slopes and the intercepts of functions fitted to different random samples from the same population. I have attempted to remove this

TABLE 5. Test for an acquired fecundity cost of reproduction. The b_i are the parameters of the cubic equation relating fecundity to age in each clone. Observed correlation coefficients are compared with values expected from the null hypothesis described in the text. The cost hypothesis predicts that (observed − expected) should be negative. Four of the six results are positive, and no observed result falls more than one standard deviation away from expectation.

Variables	Correlation coefficients		Difference (obs-exp) in units of SD exp.
	Observed	Expected	
$b_0 * b_1$	−.9376	−.9495	+.3895
$b_0 * b_2$	+.8651	+.9080	−.7784
$b_0 * b_3$	−.8151	−.8608	+.5665
$b_1 * b_2$	−.9778	−.9821	+.3361
$b_1 * b_3$	+.9472	+.9440	+.0808
$b_2 * b_3$	−.9929	−.9883	−.5498

TABLE 6. Test for a variable fecundity cost of reproduction. Mean total fecundity during days 6–10 is plotted against mean total fecundity during days 16–20 for all individuals surviving to day 20 within each clone. Entries are regression coefficients (* $P < .05$, ** $P < .01$, *** $P < .001$) and sample sizes. Cost hypothesis predicts that regression coefficients should be negative.

Clone	Regression coefficient, early fecundity on late fecundity	Sample size
1	−.085	15
2	+.725**	13
3	+.660**	14
4	.000	16
5	+.556*	12
6	+.131	13
7	+.207*	7
8	+.375	6
9	+.174	14
10	−.141	16
11	+.443	14
12	+.033	9
13	+.197	10
14	+.155	10
15	+.058	5
Pooled	+.219***	184 individuals
Means	+.441***	15 clones

autocorrelation by constructing numerically a null hypothesis with which the data can be compared. For each age-class compute the mean and the standard deviation of fecundity, using the observed means for each clone. Then for a given age-class choose a value of fecundity at random from a normal distribution with the observed mean and variance. Repeat this procedure for each age-class, up to some limit (in this case, day 25) beyond which the data become too variable for the procedure to be applicable, and fit a cubic equation to the result. When this has been done 15 times the original experiment has been replicated, under the assumption that clonal means represent random samples from normal distributions. The correlation coefficient r_{ij} between each pairwise combination of b_i and b_j can then be computed. This procedure is repeated 100 times to get an estimate of the sampling distribution of the r_{ij} under the null hypothesis. Knowing the standard deviation of the r_{ij}, we can then calculate the difference between the observed and expected values of each correlation coefficient in units of the standard deviation of the expected value. The cost hypothesis predicts that this difference should be negative, the parameters of age-specific fecundity being more negatively related to one another than would be expected by chance. Table 5 shows that the difference is positive in four of the six cases, and it is probably not different from zero in any case. The cost hypothesis is therefore rejected. A simpler alternative test is described below.

The Variable Fecundity Cost.—The cost hypothesis predicts that individuals which have greater fecundity early in life will have lesser fecundity late in life. The graph of the sum of daily fecundities between days 6 and 10 and the sum of daily fecundities between days 16 and 20, for individuals within a clone, should therefore have negative slope. Table 6 shows that the slope of this graph is negative in two cases, zero in one case, and positive for the remaining 12 cases. The slope is significantly different from zero ($P < .05$) in only four cases, all of which are positive. The cost hypothesis is therefore rejected.

By constructing the regression of clonal means, rather than of individual values within clones, we can set up an alternative test of the acquired fecundity cost. This has a higherly significant positive slope (Fig. 3), reinforcing the conclusion reached in the previous subsection.

Pooling data from all individuals shows that no overall fecundity cost exists, the slope of the graph being +.219 ($N = 184$, $P < .001$).

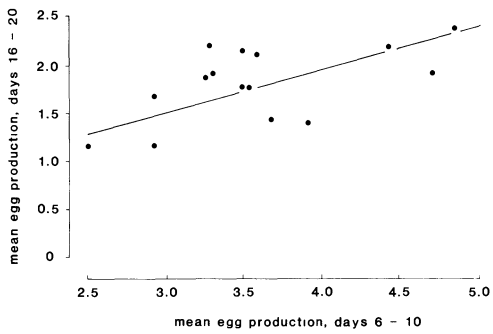

FIG. 3. Simple test for an acquired fecundity cost of reproduction. "Early fecundity" is the sum of eggs produced during days 6 through 10; "Late fecundity" the sum of eggs produced during days 16 to 20. Plotted points are clonal means, calculated for individuals surviving to day 20 at least. The cost hypothesis predicts that the graph should have negative slope: the observed slope is .441 ± .076 and the hypothesis is rejected.

DISCUSSION

The essence of the cost hypothesis is that present and future reproduction should be negatively correlated. A failure to detect any systematic effect would be disappointing; but the results given above show the correlations actually to be consistently positive. How is this discrepancy to be explained?

In the first place, it might be caused by some particular and peculiar property of the organism chosen for study. This possibility can be eliminated only by performing similar experiments with a range of different organisms.

Second, the procedural details of the experiment might be at fault. The detection of a cost might require very careful control of temperature, diet or photoperiod, or a much more detailed study of some small section of the life history, or the use of genetically defined clones.

Third, the general circumstances of the experiment might be inappropriate, suppressing costs of reproduction that would be expressed in a more realistic situation. Thus, the survival cost will not be expressed if excess reproduction does not translate into an additional risk of mortality under the conditions of culture. It is conceivable that a physiologically trivial loss of vigor might enormously increase the probability of being eaten or parasitized or outcompeted, but could not be detected in a situation from which predators, parasites and competitors were excluded. It would be desirable, therefore, to ascertain whether greater reproduction is associated with a greater risk of mortality when predators are present. More generally, minor physiological effects might be magnified by direct or indirect interaction with conspecifics, leading to a frequency-dependent cost of reproduction, but the practical difficulties of running such experiments are formidable. The fecundity cost will be suppressed if excess reproduction does not translate into a loss of potential future fecundity under the conditions of culture. This might be the case if reproduction were unconstrained because a superabundance of food was always available. This does not seem very plausible: if reproduction was unconstrained, why does fecundity decline so dramatically in older animals? One might respond that this is a feature of the life history which, after long-continued selection, has become genetically programmed and therefore inflexible when exposed to new conditions of culture. But why, then, has the negative relationship between present and future reproduction not also been programmed? Nevertheless, the correlation between life-history variables under obviously stressful conditions is clearly a worthwhile measurement to make.

A related point is that by transferring the rotifers to a novel environment the

experiment might reveal the effects of alleles, segregating at low frequency in natural populations, which confer both enhanced fecundity and enhanced survivorship in the new circumstances. This could explain the positive correlations between clonal mean fecundity and survival found in the experiment. It is very difficult to see how this objection could be countered, since even the long-term maintenance of polyclonal cultures, with the eventual re-extraction of clones from individuals surviving competition, is expected to lead to the fixation of whichever clone is optimal under the conditions of culture and the consequent disappearance of genetic variance. However, the objection does not apply to the variable costs expressed within clones.

Fourth, the design of the experiment might be fundamentally unsatisfactory. This would be the case if, when systematic sources of variation due to the genotype and the environment have been eliminated, the remaining variation necessarily creates positive correlations between components of fitness. The following illustration was suggested to me by Dr. D. Kramer. Suppose that an organism can reproduce only if it finds a food source, and that food sources are rare and scattered. Having eliminated any systematic genetic or environmental variation in the availability of food or the ability to find it, the remaining variation is stochastic. Each individual then discovers, by chance, only one or two food sources during its lifetime, and has a substantial probability of discovering none. Clearly, individuals which failed to discover any food would not reproduce either early or late in life, and would probably die young; those which found several food sources would reproduce often both early and late in life, and might survive longer. This particular scenario does not fit the rotifers used in the experiment described here, but others might—the possibility of accidents at birth, for example, or of random differences in the provisioning of eggs. This constitutes a very powerful criticism of passive experiments, including all those reported in the literature, and in principle could explain the positive correlations between individuals within clones that I have described here. It does not seem to provide an adequate explanation for the positive correlations between clonal means (Table 3 and Fig. 3). The use of techniques by which reproduction at a specified age can be manipulated without directly affecting reproduction or survival at later ages is clearly very desirable, and it may be significant that it is experiments of this kind which have provided the most convincing support for a cost hypothesis.

Fifth, we must face the possibility that the cost hypothesis might simply be wrong, or at least that it requires radical restatement before it can be incorporated into realistic optimization models. What is at issue here is the nature of the genotype–environment interaction. Despite controlling both genotype and environment, the experimental design adopted here does not take into account the possibility of an interaction between the two. The point is illustrated by Figure 4. As usually stated, the cost hypothesis posits a negative relationship between present and future reproduction (Fig. 4A) in an essentially uniform environment (Fig. 4B), so that a greater present reproduction causes a comparable decrement in expected future reproduction in all the circumstances the organism might encounter. Since present and future reproduction are negatively correlated within any given niche, while age-specific reproduction is positively correlated between niches, an increase in present reproduction cannot be achieved except at the expense of a lower future reproduction in all niches. But suppose that present and future reproduction really are positively correlated within any given niche (Fig. 4C), whereas age-specific reproduction is negatively correlated between niches (Fig. 4D), so that an individual achieving exceptionally great reproduction in some given niche will tend to perform poorly in another, randomly-chosen niche. If the

organism were to inhabit only a single niche, then selection simply favors the maximization of reproduction; but if its offspring are dispersed to different niches then the negative heritability of reproductive rate between niches will create selection towards some intermediate phenotype. Naturally, when we culture the organism under uniform conditions it is only the positive within-niche correlations we shall observe. This argument may point the way to a qualitatively different optimization model for the life history.

Finally, we must consider the possibility that the axiom lying beneath the cost hypothesis might be invalid. This axiom identifies natural selection as the agent which, acting through reproductive cost, directs the evolution of life histories towards intermediate optima. If all our efforts to demonstrate some form of reproductive cost end in failure, or succeed only in a minority of instances, then we may eventually be forced to seek some other category of explanation. Consider, for example, the relationship between egg size and the age at first reproduction. Larger eggs may have a better start in life, and thereby grow more rapidly and reproduce earlier. Other things being equal, however, an individual reproducing earlier in life, perhaps at a smaller size or with a smaller quantity of stored reserves, may be likely to produce smaller eggs. We than have a causal sequence which reads larger egg → earlier reproduction → smaller egg → later reproduction → larger egg and so forth. This will rapidly lead to the attainment of an intermediate egg size and an intermediate age at first reproduction. Selection is not involved, because the process can work without any genetic variation whatsoever; the physiological response is all that is required.

At this stage, of course, to abandon the cost hypothesis, and still more to abandon the selection axiom, would demonstrate only a lack of caution. What the arguments set out above do provide is a plan for further experimentation. It must

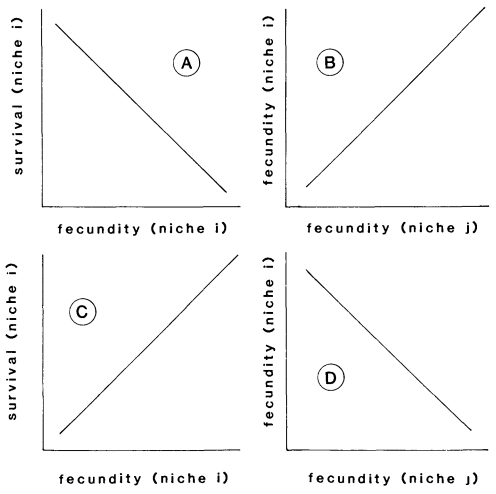

FIG. 4. The cost hypothesis is usually taken to mean that present and future reproduction are negatively correlated within any given niche (Fig. 4A) while life-history variables are in general positively correlated between niches (Fig. 4B). However, it is possible that present and future reproduction are positively correlated within any given niche (Fig. 4C), as the results of the experiment described here suggest, whereas life-history variables are negatively correlated between niches (Fig. 4D). Either scheme (A and B, or C and D) may procure selection towards intermediate values of variables such as survival and fecundity rates.

first be demonstrated that the result reported here is again obtained when similar experiments are performed with different organisms. The result must be repeated when more detailed and carefully-controlled studies of small sections of life-history of genetically-characterized clones are performed. It must then be shown that stressful conditions, and especially the presence of predators, are not associated with the appearance of negative correlations between present and future fecundity. Finally, it is necessary to develop manipulative techniques to certify the results of passive experiments. All of these experiments have been completed or are currently being run in my laboratory, and will be described in later papers in this series. Only if they fail to impeach the results of the present experiment need any radical restatement of the cost hypothesis be considered, but it re-

mains a matter of some concern that so important, and on the face of things so plausible, an hypothesis should at present lack substantial empirical support.

Summary

The cost hypothesis states that any increment in present reproduction is associated with a decrement in the expectation of future reproduction. It is crucial to the theory of life histories, but has yet to receive clear and general support from empirical studies. This support may be provided by passive experiments, in which the correlation between life-history variables is measured when systematic variation due to genotype or environment has been eliminated or controlled, or by manipulative experiments, in which the fate of individuals whose reproduction has been altered is compared with that of control individuals. Either type of experiment might demonstrate a relationship between present fecundity and survival (the survival cost) or between present and future fecundity (the fecundity cost). These costs might be caused directly (variable costs), in which case they will be detected by comparing the future performance of genetically identical individuals with different present reproduction, or they might have become genetically programmed (acquired costs) as the result of selection acting through the variable costs, and will be detected by comparing the mean performance of different genotypes. The passive experiment described here used the life tables of 15 clones of the rotifer *Platyias patulus* to study the variable and acquired survival and fecundity costs. The negative correlations predicted by the cost hypothesis were not found in any case; instead, correlations tended to be positive. This result might be attributable to the peculiarities of the organism; to the procedural details of the experiment; to the general inappropriateness of the laboratory situation; to fundamental logical flaws in the design of passive experiments; to the fallacy or incompleteness of the cost hypothesis; or to the fallacy of the Darwinian interpretation of life histories. A program of experimentation designed to sift these possibilities is outlined.

Acknowledgments

I am grateful to H. O'Neil and J. Laskey for technical assistance. This work was supported by a grant from the National Science and Engineering Research Council of Canada.

Literature Cited

Askenmo, C. 1979. Reproductive effort and return rate of male pied flycatchers. Amer. Natur. 114:748–752.

Bell, G. 1980. The costs of reproduction and their consequences. Amer. Natur. 116:45–76.

Browne, R. A. 1982. The costs of reproduction in brine shrimp. Ecology 63:43–47.

Bryant, D. M. 1979. Reproductive costs in the house martin (*Delichon urbica*). J. Anim. Ecol. 48:655–675.

Calow, P., and A. S. Woollhead. 1977. The relation between ration, reproductive effort and age-specific mortality in the evolution of life-histories—some observations on freshwater triclads. J. Anim. Ecol. 46:765–782.

Charlesworth, B., and J. A. Leon. 1976. The relation of reproductive effort to age. Amer. Natur. 110:449–459.

Daly, M. The cost of mating. Amer. Natur. 112: 771–774.

Dean, J. M. 1981. The relationship between lifespan and reproduction in the grasshopper *Melanoplus*. Oecologia 48:385–388.

De Steven, D. 1980. Clutch size, breeding success and parental survival in the tree swallow (*Iridoprocne bicolor*). Evolution 34:278–291.

Fairbairn, D. J. 1977. Why breed early? A study of reproductive tactics in *Peromyscus*. Can. J. Zool. 55:862–871.

Feifarek, B. P., G. A. Wyngaard, and J. D. Allan. 1983. The cost of reproduction in a freshwater copepod. Oecologia. *In press.*

Gadgil, M., and W. H. Bossert. 1970. Life historical consequences of natural selection. Amer. Natur. 106:258–261.

Giesel, J. T. 1979. Genetic co-variation of survivorship and other fitness indices in *Drosophila melanogaster*. Exp. Geront. 14:323–328.

Gowen, J. W., and L. E. Johnson. 1946. On the mechanism of heterosis. I. Metabolic capacity of different races of *Drosophila melanogaster* for egg production. Amer. Natur. 80:149–179.

Hiraizumi, Y. 1961. Negative correlation between rate of development and female fertility in *Drosophila melanogaster*. Genetics A6:615–624.

Hirshfield, M. F. 1980. Experimental analysis

of reproductive effort and cost in the Japanese medaka, *Oryzias latipes*. Ecology 61:282–292.

HOGSTEDT, G. 1981. Should there be a positive or a negative correlation between survival of adults in a bird population and their clutch size? Amer. Natur. 118:568–571.

JENNINGS, H. S., AND R. S. LYNCH. 1928. Age, mortality, fertility and individual diversities in the rotifer *Proales sordida* Gosse. II. Life history in relation to mortality and fecundity. J. Exp. Zool. 51:339–381.

KLUIJVER, H. N. 1971. Regulation of numbers in populations of great tits (*Parus m. major*), p. 507–523. *In* P. J. den Boer and G. R. Gradwell (eds.), Proc. Adv. Study Inst. Dynamics Numbers Popul. Pudoc, Waageningen.

LACK, D. 1966. Population Studies of Birds. Clarendon Press, Oxford.

LANSING, A. I. 1948. The influence of parental age on longevity in rotifers. J. Gerontol. 3:6–17.

LAW, R. 1979. The costs of reproduction in annual meadow grass. Amer. Natur. 113:3–16.

LEGGETT, W. C., AND J. E. CARSCADDEN. 1978. Latitudinal variation in reproductive characteristics of American shad (*Alosa sapidissima*): evidence for population—specific life history strategies in fish. J. Fish Res. Bd. Can. 35:1469–1478.

LEWONTIN, R. C. 1974. The Genetic Basis of Evolutionary Change. Columbia Univ. Press, N.Y.

PARTRIDGE, L., AND M. FARQUHAR. 1981. Sexual activity reduces lifespan of male fruitflies. Nature 294:580–582.

ROSE, M. R., AND B. CHARLESWORTH. 1981*a*. Genetics of life history in *Drosophila melanogaster*. I. Sib analysis of adult females. Genetics 97: 173–186.

———. 1981*b*. Genetics of life history in *Drosophila melanogaster*. II. Exploratory selection experiments. Genetics 97:187–196.

SCHAFFER, W. M. 1974. Selection for optimal life histories: the effects of age structure. Ecology 55: 291–303.

SIMMONS, M. J., C. R. PRESTON, AND W. R. ENGELS. 1980. Pleiotropic effects on fitness of mutations affecting viability in *Drosophila melanogaster*. Genetics 94:467–475.

SMITH, J. N. M. 1981. Does high fecundity reduce survival in song sparrows? Evolution 35:1142–1148.

SNELL, T. W., AND C. E. KING. 1977. Lifespan and fecundity patterns in rotifers: the cost of reproduction. Evolution 31:882–890.

TALLAMY, D. W., AND R. F. DENNO. 1982. Life history trade-offs in *Gargaphia solani* (Hemiptera: Tingidae): the cost of reproduction. Ecology 63: 616–620.

TEMIN, R. G. 1966. Homozygous viability and fertility loads in *Drosophila melanogaster*. Genetics 53:27–46.

TINKLE, D. W. 1969. The concept of reproductive effort and its relation to the evolution of life histories of lizards. Amer. Natur. 103:501–516.

WILLIAMS, G. C. 1966*a*. Adaptation and Natural Selection: A Critique of Some Current Evolutionary Thought. Columbia Univ. Press, N.Y.

———. 1966*b*. Natural selection, the costs of reproduction, and a refinement of Lack's principle. Amer. Natur. 100:687–690.

Corresponding Editor: R. E. Ricklefs