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"Analysis of Life Table Response Experiments 1. Decomposition of Effects on Population Growth Rate"

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ANALYSIS OF LIFE TABLE RESPONSE EXPERIMENTS I. DECOMPOSITION OF EFFECTS ON POPULATION GROWTH RATE

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ABSTRACT

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Life table response experiments use the vital rates of an organism as the response variable in studies of the population-level response to environmental or biological factors. Demographic indices, particularly the asymptotic population growth rate λ (or $r = \ln \lambda$), are commonly used as summary statistics to integrate the multifarious effects of the environmental factors on the life table. This raises the question of how to decompose the overall effect of a treatment on λ into contributions due to its effects on the individual survival and fertility rates. These contributions can be calculated from matrix projection models. Examples are presented, including a two-way factorial experiment in which both main effects and interactions are decomposed into contributions. In general, it cannot be assumed that large effects on the vital rates translate into large contributions to the effects on λ.

INTRODUCTION

A life table response experiment (LTRE) is an experiment in which the life table or, more generally, the collective vital rates of an organism, appears as the response variable in a more or less complex, but standard experimental design. LTREs evaluate population-level responses to environmental or biological factors which have individual-level effects on the vital rates. Since the population-level response to an environmental factor depends on that factor's effects on the life table, these experiments have provided powerful tools for the investigation of the impact of a variety of biotic and abiotic factors.

The response of the complete life table can provide an experimenter with more information than can be comfortably digested, for two reasons. First, different ages or stages in the life cycle have vastly different sensitivities to a

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given factor. This is well documented in acute toxicological bioassays [e.g., 96-h LC_{50} values for the copepod *Tigriopus californicus* exposed to copper vary 40-fold between instars (O'Brien et al., 1988); LC_{50} values for *Chironomus riparius* larvae exposed to cadmium vary by as much as 1000-fold between instars (Williams et al., 1986)] and is likely to be the rule rather than the exception. Second, a single environmental factor often produces very heterogeneous effects on the vital rates, e.g., chronic exposure to cadmium reduces survival but increases brood size in *Daphnia galeata mendotae* (Marshall, 1978). The complete life table reflects all this diversity.

This surfeit of information can be reduced to manageable proportions by calculating integrative demographic statistics, particularly the intrinsic rate of increase (r or its discrete version $\lambda = e^r$). One of the earliest LTREs to be analyzed in this way was a study by Birch (1953), who analyzed the effects of temperature, moisture, and food on three species of flour beetles. He presented the results as contour plots of λ as a function of temperature and humidity for two species, and plots of λ versus temperature for different humidity levels.

LTREs are frequently used to study the effects of chronic exposure to toxicants – Marshall, 1962 (gamma radiation); Hummon and Hummon, 1975 (DDT); Winner and Farrell, 1976 (copper); Daniels and Allan, 1981 (Dieldrin); Allan and Daniels, 1982 (Kepone); Fitzmayer et al., 1982 (Simazine); Gentile et al., 1982 (heavy metals); Walton et al., 1982 (pH).

LTREs may be extended to arbitrarily complex factorial designs, which permit the examination of interactions between factors. Examples include Birch's (1953) study cited above, Stiven's (1962) study of temperature and food effects in three species of *Hydra*, the study of Birch et al. (1963) on temperature and genetic strain in *Drosophila serrata*, King's (1967) examination of the effects of food type, food level, and clone age on the rotifer *Euchlanis dilatata*, George's (1985) study of temperature and salinity effects in the copepod *Eurytemora herdmani*, and the study by Rao and Sarma (1986) on the effects of DDT and food level on the rotifer *Brachionus patulus*. Most of these studies report significant interactions between the factors, emphasizing the importance of examining combinations of treatment factors.

Integrative demographic statistics like λ reduce treatment effects on the life table to effects on a scalar index, but in so doing they obscure the source of those effects. Obviously, not all changes in the vital rates will have the same effect on λ; as an extreme example, λ is completely independent of the mortality of individuals in post-reproductive age classes. Knowing that an environmental factor has a large effect on a particular vital rate does not guarantee that its impact on λ is due to that effect.

What is needed is a way to *decompose* the effect of a factor on λ into

contributions due to the effects of that factor on age-specific survival and reproductive rates. This paper provides such a decomposition: it allows the results of a LTRE to be interpreted in terms of the effects of a factor on λ and of the particular vital rates which account for those effects. Keyfitz (1968, pp. 189–190) considered a similar decomposition problem in the context of human populations, but limited his analysis to determining the extent to which observed differences in r reflected differences in survival or fertility, without examining age-specific effects.

A subsequent paper will consider LTREs based on size- or stage-specific demographic models, which are now known to be more appropriate than classical age-specific models for many species (e.g. Caswell, 1986, 1988). These more general analyses can also be applied to the analysis of LTREs based on only partial life table information. This is particularly useful when experiments must be completed in a shorter time than required to estimate a complete life table.

DEMOGRAPHIC BACKGROUND

Consider a population classified into s stages (age classes, size classes, instars, developmental stages, or any other biologically relevant classification). The vital rates are incorporated into an $s \times s$ population projection matrix **A**, the (i, j) entry of which (a_{ij}) gives the number of individuals in stage i at time $t + 1$ per individual in stage j at time t. In the age-classified models considered here, **A** contains positive entries only on the subdiagonal (P_i is the survival probability of age-class i) and in the first row (F_i is the effective fertility of age-class i).

The demographic statistics implied by the schedule of vital rates are given by the eigenvalues and eigenvectors of **A**. The dominant eigenvalue λ of **A** gives the asymptotic rate of population growth. The intrinsic rate of increase $r = \ln \lambda$. The corresponding right and left eigenvectors w and v give the stable stage distribution and stage-specific reproductive value distribution, respectively. The sensitivity of λ to changes in the a_{ij} plays a pivotal role in the analysis; it is given by:

$$\frac{\partial \lambda}{\partial a_{ij}} = \frac{v_i w_j}{\langle w, v \rangle} \tag{1}$$

where $\langle \cdot, \cdot \rangle$ denotes the scalar product (Caswell, 1978).

Population growth rate is not the only demographic statistic that could be used in the analysis of LTREs (Caswell, 1986, 1989), but it is the most important and frequently used. The value $\lambda = 1$ marks a critical borderline between persistence ($\lambda \geq 1$) and certain extinction ($\lambda < 1$). It also figures prominently as a measure of fitness in age-structured population genetic models (Lande, 1982).

DECOMPOSITION OF TREATMENT EFFECTS

I turn now to the problem of the decomposition of treatment effects on λ. (The analysis can be recast in terms of r by noting that $\partial r/\partial a_{ij} = \lambda^{-1} \partial\lambda/\partial a_{ij}$.) The treatments may be applied in an arbitrarily complex factorial design. The goal is to characterize the contributions of treatment effects on the a_{ij} to effects on λ; we do so by using a linear approximation for λ as a function of the a_{ij}, using the sensitivity formula (1).

I will use superscripts in parentheses to denote treatments, and subscripts to denote matrix elements. Thus $\mathbf{A}^{(i)}$ denotes the matrix of vital rates in treatment i, $\lambda^{(i)}$ the dominant eigenvalue of $\mathbf{A}^{(i)}$, and $a_{kl}^{(i)}$ the (k, l) entry of $\mathbf{A}^{(i)}$. Means are denoted by replacing a superscript by a dot; e.g.

$$\mathbf{A}^{(\cdot)} = \frac{1}{m} \sum_{j}^{m} \mathbf{A}^{(j)} \tag{2}$$

where m is the number of levels of the second treatment.

One-way designs

Consider n treatments (e.g. levels of exposure to a toxicant), yielding projection matrices and growth rates $\mathbf{A}^{(i)}$ and $\lambda^{(i)}$, $i = 1, \ldots, n$. By analogy to the analysis of variance, we write a linear model:

$$\lambda^{(i)} = \lambda^{(\cdot)} + \alpha^{(i)} \tag{3}$$

where $\alpha^{(i)}$ is the effect of the ith level of the treatment, measured as a deviation from the growth rate generated by the average projection matrix. Standard least-squares estimation theory yields the estimates:

$$\hat{\alpha}^{(i)} = \lambda^{(i)} - \lambda^{(\cdot)} \tag{4}$$

The effect $\alpha^{(i)}$ reflects all the differences in survival and fertility between the treatment matrix $\mathbf{A}^{(i)}$ and the mean matrix $\mathbf{A}^{(\cdot)}$. We wish to decompose $\alpha^{(i)}$ into the contributions due to the differences in each matrix element. This decomposition is provided by a first order approximation of $\hat{\alpha}^{(i)}$:

$$\hat{\alpha}^{(i)} \approx \tilde{\alpha}^{(i)} = \sum_{k,l} \left(a_{kl}^{(i)} - a_{kl}^{(\cdot)}\right) \left.\frac{\partial\lambda}{\partial a_{kl}}\right|_{(\mathbf{A}^{(i)} + \mathbf{A}^{(\cdot)})/2} \tag{5}$$

Each term in the summation (5) gives the contribution of the differences in one matrix element to the effect of the treatment on λ. This contribution consists of the difference in the matrix element weighted by the sensitivity of λ to that element. The mean value theorem guarantees that the approximation (5) is an identity when the sensitivity is evaluated at some point between $\mathbf{A}^{(i)}$ and $\mathbf{A}^{(\cdot)}$; for the sake of this approximation I have evaluated it at a matrix 'midway' between the two treatments.

If λ were a linear function of the a_{kl}, $\partial\lambda/\partial a_{kl}$ would be a constant for all k, l, and the $\tilde{\alpha}^{i}$ would sum to zero. In practice, this constraint is only approximately satisfied, so that:

$$\sum \tilde{\alpha}^{(i)} \approx 0 \tag{6}$$

The accuracy of the approximation can be checked by calculating a predicted value of $\lambda^{(i)}$:

$$\tilde{\lambda}^{(i)} = \lambda^{(\cdot)} + \tilde{\alpha}^{(i)} \tag{7}$$

and comparing this with the actual value of $\lambda^{(i)}$.

Larval development mode in Streblospio benedicti

As an example of a one-way design, consider the data of Levin et al. (1987) on the effects of larval development mode on the population growth rate of the polychaete *Streblospio benedicti*. Two genetic strains of this species exist, one of which produces non-feeding lecithotrophic larvae and one of which produces feeding planktotrophic larvae. The lecithotrophic strain produces many fewer offspring, but they are larger because of their yolk supply, and survive better.

Levin et al. (1987) measured life tables for *S. benedicti* under laboratory conditions, and calculated the resulting projection matrices $\mathbf{A}^{(L)}$ for lecithotrophs and $\mathbf{A}^{(P)}$ for planktotrophs, with corresponding rates of increase $\lambda^{(L)} = 1.319$ and $\lambda^{(P)} = 1.205$. Since this LTRE examines the effect of an internal genetic factor rather than an external environmental factor, the interpretation of λ as a measure of fitness is particularly relevant. The effect of larval development mode on fitness is the integrated result of differences in age-specific survival and fertility; which of those differences are most important?

The upper panels of Fig. 1 plot the differences between the strains in age-specific fertility and survival probability, measured relative to the mean $\mathbf{A}^{(\cdot)} = (\mathbf{A}^{(L)} + \mathbf{A}^{(P)})/2$. The most dramatic differences between the two strains are a huge lecithotrophic fertility disadvantage between 20 and 30 weeks of age, a lecithotrophic survival advantage between 0 and 10 weeks of age, and a lecithotrophic survival disadvantage between 30 and 40 weeks of age.

The contributions of these differences to $\tilde{\alpha}^{(L)}$ and $\tilde{\alpha}^{(P)}$ are shown in the lower panels. What is most striking is that the fertility differences between 20 and 30 weeks of age make almost no contribution to the difference in λ. Indeed, all but a very small proportion of the effect on λ is contributed by fertility and survival effects occurring before 15 weeks of age.

Note that the curves for $\tilde{\alpha}^{(L)}$ and $\tilde{\alpha}^{(P)}$ are nearly complements of each other, as implied by (6). The predicted values, $\tilde{\lambda}^{(L)} = 1.335$ and $\tilde{\lambda}^{(P)} = 1.203$ are quite accurate.

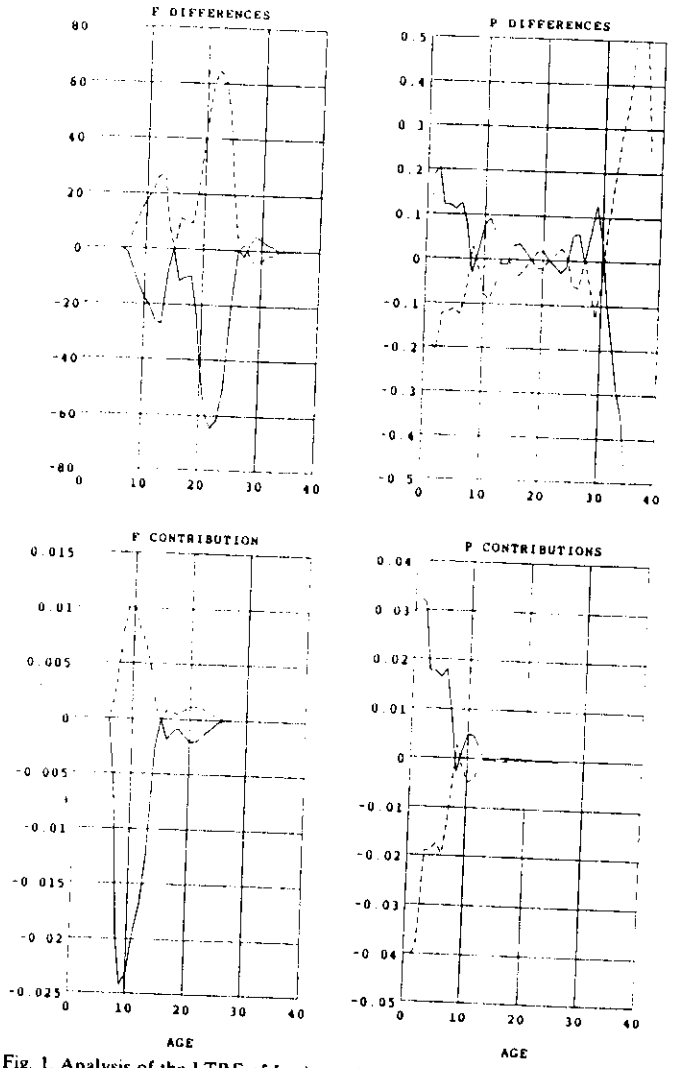

Fig. 1. Analysis of the LTRE of Levin et al. (1987) comparing lecithotrophic (solid lines) and planktotrophic (dashed lines) strains of the polychaete *Streblospio benedicti*. Upper panels: Effects of genetic strain on age specific fertility (left) and survival (right), measured relative to the mean. Lower panels: The contributions of these age-specific effects to $\tilde{\alpha}$, the main effect of genetic strain.

Because survival and fertility are measured in different units, the treatment *differences* reveal nothing about the relative importance of survival and fertility. The *contributions*, on the other hand, are directly comparable, and show in this case that survival differences are consistently more important than fertility differences.

The differences in this study between the effects of a factor on the vital rates and the contributions of those effects to λ are typical of LTREs. Clarifying these differences is one of the most important benefits of this form of LTRE analysis.

Factorial designs

Consider an experiment with two cross-classified treatments (the extension to higher-order factorial designs is straightforward). Let $\mathbf{A}^{(ij)}$ denote the projection matrix resulting from the ith level of the first treatment and the jth level of the second treatment, and $\lambda^{(ij)}$ its eigenvalue. The model for such an experiment is:

$$\lambda^{(ij)} = \lambda^{(\cdot\cdot)} + \alpha^{(i)} + \beta^{(j)} + (\alpha\beta)^{(ij)} \tag{8}$$

where $\alpha^{(i)}$ and $\beta^{(j)}$ are the main effects and $(\alpha\beta)^{(ij)}$ is the interaction effect. Estimates of the treatment effects are given by:

$$\hat{\alpha}^{(i)} = \lambda^{(i\cdot)} - \lambda^{(\cdot\cdot)}$$

$$\hat{\beta}^{(j)} = \lambda^{(\cdot j)} - \lambda^{(\cdot\cdot)} \tag{9}$$

$$\widehat{(\alpha\beta)}^{(ij)} = \lambda^{(ij)} - \hat{\alpha}^{(i)} - \hat{\beta}^{(j)} - \lambda^{(\cdot\cdot)} \tag{10}$$

$$\tag{11}$$

These effects can be decomposed, following the approach of the previous section:

$$\tilde{\alpha}^{(i)} = \sum_{k,l} \left(a_{kl}^{(i\cdot)} - a_{kl}^{(\cdot\cdot)}\right) \left.\frac{\partial \lambda}{\partial a_{kl}}\right|_{(\mathbf{A}^{(i\cdot)} + \mathbf{A}^{(\cdot\cdot)})/2} \tag{12}$$

$$\tilde{\beta}^{(j)} = \sum_{k,l} \left(a_{kl}^{(\cdot j)} - a_{kl}^{(\cdot\cdot)}\right) \left.\frac{\partial \lambda}{\partial a_{kl}}\right|_{(\mathbf{A}^{(\cdot j)} + \mathbf{A}^{(\cdot\cdot)})/2} \tag{13}$$

$$\widetilde{(\alpha\beta)}^{(ij)} = \sum_{k,l} \left(a_{kl}^{(ij)} - a_{kl}^{(\cdot\cdot)}\right) \left.\frac{\partial \lambda}{\partial a_{kl}}\right|_{(\mathbf{A}^{(ij)} + \mathbf{A}^{(\cdot\cdot)})/2} - \tilde{\alpha}^{(i)} - \tilde{\beta}^{(j)} \tag{14}$$

Each of these equations approximates an observed change in λ as a linear function of the changes in the entries of the matrix; the slope of the linear approximation is evaluated at the midpoint of the two matrices being compared.

The interaction effect $(\widetilde{\alpha\beta})^{(ij)}$ is the difference between the actual eigenvalue $\lambda^{(ij)}$ and the value predicted on the basis of an additive model. The contributions $(\widetilde{\alpha\beta})^{(ij)}$ are obtained from the corresponding linear approximations. A positive component of $(\widetilde{\alpha\beta})^{(ij)}$ thus indicates that the interaction of treatments i and j increases $\lambda^{(ij)}$ above the value predicted by the additive model; negative components indicate that the interaction decreases $\lambda^{(ij)}$.

Food and DDT toxicity in a rotifer

Rao and Sarma (1986) exposed the rotifer *Brachionus patulus* to five levels (0, 15, 30, 45, and 60 ppm) of DDT and two levels (1×10^6 and 3×10^6 *Chlorella* cells ml^{-1}) of food. The effects of the treatments on λ are shown in Fig. 2. There was a clear positive effect of high food, and a negative effect of DDT concentration. In Rao and Sarma's (1986) analysis of variance, there was also a significant Food × DDT interaction, with the growth rate reduction due to DDT being more severe at low than at high food levels. Let $\alpha^{(i)}$ denote the effect of the ith DDT treatment and $\beta^{(j)}$ denote the effect of the jth food treatment. The effects of DDT on age-specific fertility, and the contributions of those effects to $\hat{\alpha}^{(i)}$, $i = 1, \ldots, 5$, are shown in Fig. 3. The upper panels show the effects on age-specific fertility, measured relative to the overall mean projection matrix. As DDT concentration increases, fertility declines first at older ages, then at progressively younger ages.

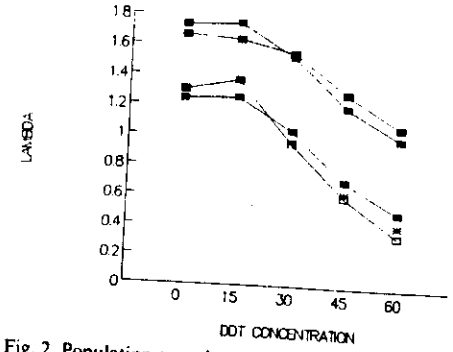

Fig. 2. Population growth rate λ as a function of DDT concentration (abcissa) and food level (lower lines, low food; upper lines, high food) for the rotifer *Brachionus patulus* (Rao and Sarma, 1986). Open squares, observed values; asterisks, values predicted from the linear approximation including the interaction terms; solid squares, values from the additive model, ignoring interaction effects.

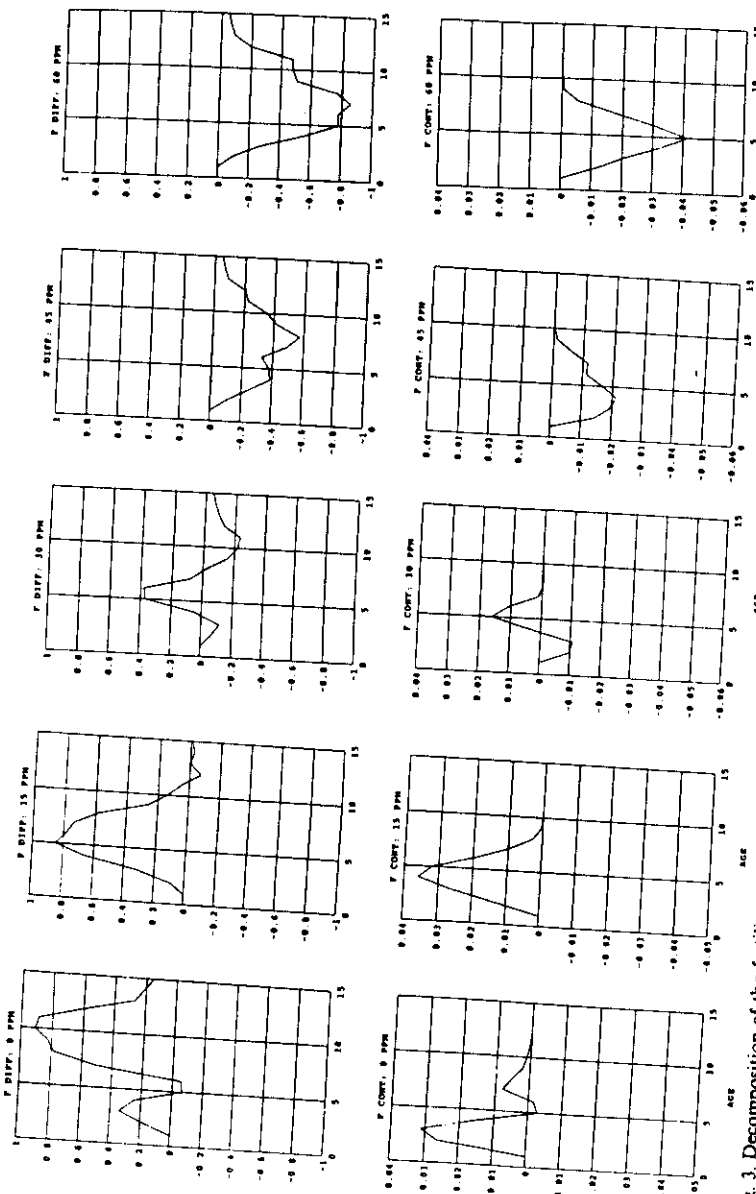

Fig. 3. Decomposition of the fertility contributions to the main effects of DDT concentration. Upper panels: Effects of the treatment levels on age specific fertility, measured relative to the mean. Lower panels: Contributions of those effects to $\hat{\alpha}^{(i)}$. Data of Rao and Sarma (1986).

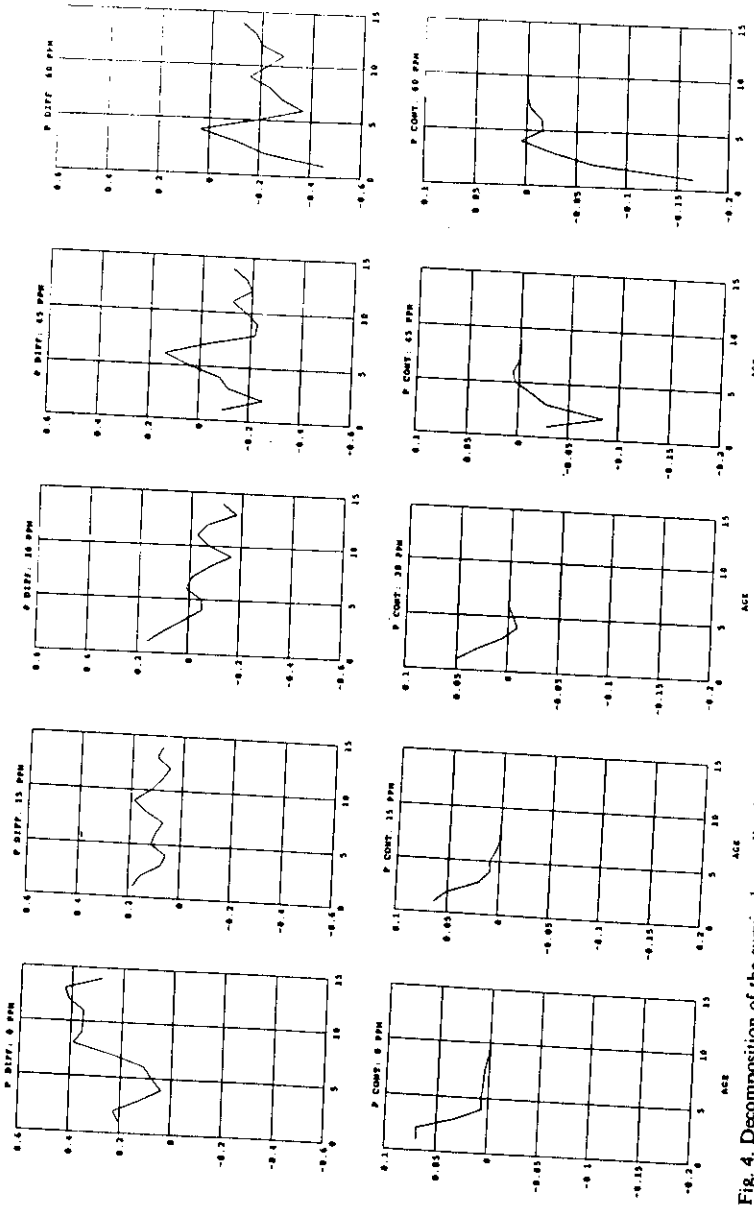

Fig. 4. Decomposition of the survival contributions to the main effects of DDT concentration. Upper panels: Effects of the treatment levels on age specific survival probability, measured relative to the mean. Lower panels: Contributions of these effects to $\tilde{\alpha}^{(j)}$. Data of Rao and Sarma (1986).

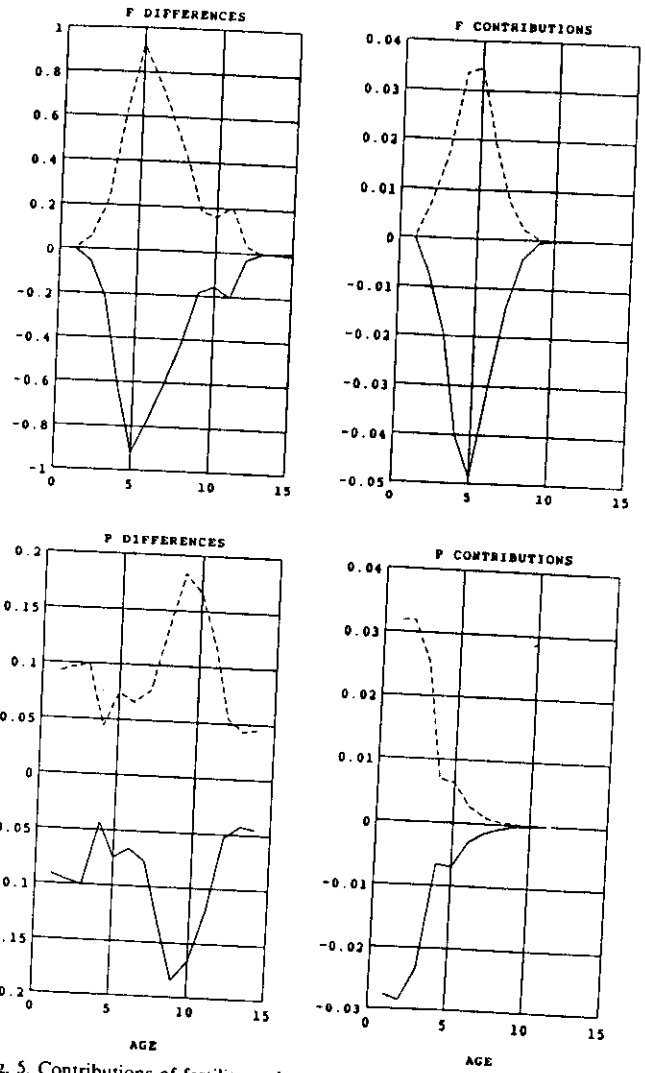

Fig. 5. Contributions of fertility and survival differences to $\tilde{\beta}^{(j)}$, $j = 1, 2$, the main effects of food level. Solid line, low food; dashed line, high food. Upper panels: Effects of food on fertility (left) and contributions of those effects to $\tilde{\beta}^{(j)}$ (right). Lower panels: Effects of food on survival probability (left) and contributions of those effects to $\tilde{\beta}^{(j)}$ (right). Data of Rao and Sarma (1986).

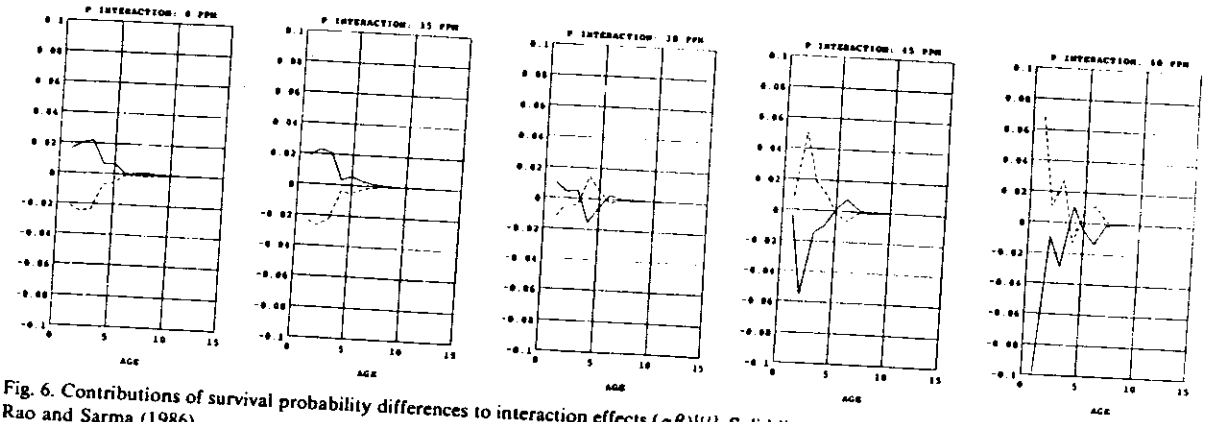

Fig. 6. Contributions of survival probability differences to interaction effects $(\alpha\beta)^{(ij)}$. Solid lines, low food; dashed lines, high food. Data of Rao and Sarma (1986).

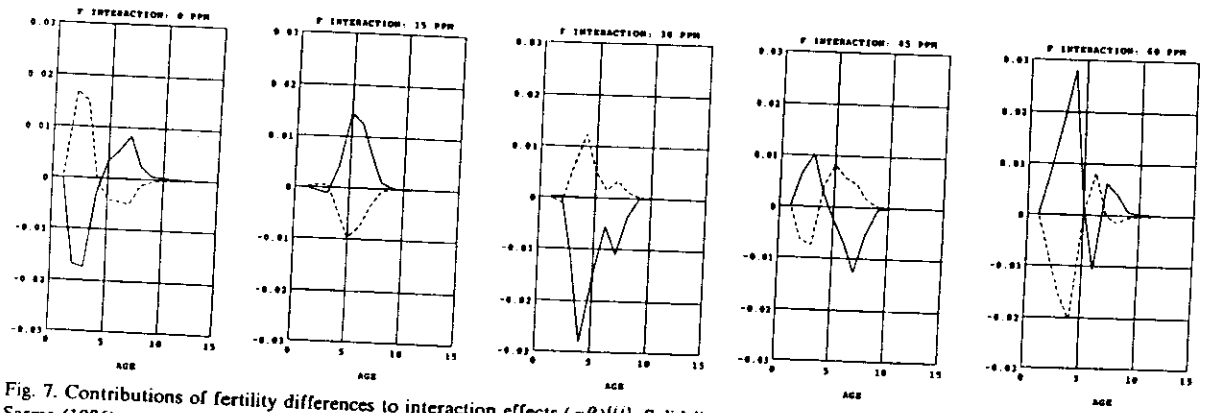

Fig. 7. Contributions of fertility differences to interaction effects $(\alpha\beta)^{(ij)}$. Solid lines, low food; dashed lines, high food. Data of Rao and Sarma (1986).

The lower panel in Fig. 3 shows the corresponding contributions to $\tilde{\alpha}^{(i)}$, $i = 1, \ldots, 5$. The pattern is similar to that of the fertility differences, but reflects primarily the change in fertility at young ages. Fertility differences after age 10 have essentially no impact on λ, and most of the fertility-mediated impact of DDT on λ occurs in the first 7–8 days of life.

Figure 4 shows the corresponding analysis for survival effects. Increasing DDT concentrations reduce survival probability, first among older individuals and then, at the two highest concentrations, at younger ages. The contributions of these effects to $\tilde{\beta}^{(j)}$, however, are limited to the first 5–7 days of life. The large effects of DDT on survival of individuals from 7–15 days of age make a negligible contribution to differences in λ.

The main effects of food level are shown in Fig. 5. High food levels increase fertility, especially from 3 to 9 days of age. The contributions of these effects to $\tilde{\beta}^{(j)}$ are similar in form, but essentially limited to the first 7 days of life. The effect of food on survival is greatest at later ages (7–15 days), but these effects make no contribution to population growth rate.

The contributions of survival effects to the interaction terms $(\widetilde{\alpha\beta})^{(ij)}$ are shown in Fig. 6. As with the main effects, contributions beyond age 7 days are negligible. At low DDT concentrations, the interaction terms for low food (solid lines) are positive (the terms for high food are complementary, since the sum of the low and high food interaction effects is approximately zero), indicating that the low food treatment does better than would be expected from an additive model. At higher DDT concentrations, the effects reverse, and the low food treatment does much worse than would be expected on an additive model. Thus, low food levels exacerbate, while high food levels counteract, the survival effects of DDT toxicity.

The contributions of fertility differences to the interaction terms are much smaller than the corresponding survival contributions (compare the scales of Figs. 6 and 7), and are more difficult to interpret. There is some tendency for the interaction effects to be opposite in sign for contributions due to early (0–5 days) and later (5–10 days) fertility. At early ages, low food levels *counteract* the effect of DDT concentration (cf. the interaction plots for 0 and 60 ppm). At later ages, the effect is reversed. The mechanism for this switch is unknown.

The accuracy of the linear approximations in this case is shown in Fig. 2, which shows the observed values of λ, those predicted by the estimated model including the interaction terms, and those predicted by the additive model without the interaction. The observed and estimated values are extremely close. The deviation of the additive model predictions from the observed values shows the nature of the interaction; at low food levels λ declines with DDT concentration faster than predicted by the additive

model, while at high food levels λ declines more slowly than predicted by the additive model.

The results of this analysis can be summarized as follows:

(1) Increasing DDT concentration reduces population growth rate by reducing fertility during the first 10 days of life and survival probability during the first 5–7 days of life. DDT has sizeable effects on later survival and fertility, but these effects have negligible impact on λ.

(2) Low food levels reduce population growth rate by reducing fertility and survival during the first 7 days of life. Large effects of food level on survival between ages 7 and 15 days have negligible impact on λ.

(3) The interaction effect between food level and DDT concentration is mediated mainly through survival effects during the first 7 days of the life cycle. Low food levels exacerbate the survival effects of DDT toxicity; high food levels counteract it.

TESTS OF SIGNIFICANCE

The analysis presented here is concerned with estimation of treatment effects, not with tests of the significance of those effects. One could legitimately question whether the differences between the life tables of, say lecithotrophic and planktotrophic strains of *Streblospio benedicti* are significantly greater than expected by chance. If they are not, their decomposition into age-specific contributions is of little interest.

There are a variety of methods available for testing differences between life tables (Elandt-Johnson and Johnson, 1980, Crowley and Breslow, 1984). Levin et al. (1987) did in fact show that differences between reproductive characters of the two strains were highly significant, and the differences in survivorship were dramatic enough that only a statistical purist could complain about a lack of statistical testing. In many cases, it will be sufficient to conduct such tests, and then, reassured that the life tables do contain significant differences, go on to decompose the contributions of those differences to changes in λ.

Rao and Sarma's (1986) LTRE on *Brachionus patulus* went one step further, by replicating each of their treatments 3 times. They could thus use ANOVA to test directly for the significance of food, DDT, and interaction effects on population growth rate (r in their analysis). All three effects were highly significant. Reassured that not only are the life tables different, but that those differences are reflected in significant differences in λ, one can go on to conduct the contribution analysis.

It is important to note, however, that significant differences in λ are *not* necessary to apply this analysis. Imagine an environmental factor which reduces survival and increases fertility (much as in the *Streblospio* example),

and that those effects exactly cancel each other, leaving λ completely unchanged. There can be no significant differences in λ in this case, but much could be learned from the contribution analysis about how the two effects are balanced against each other.

SOFTWARE

The analysis presented here requires the calculation of eigenvalues and eigenvectors of large numbers of matrices. All the calculations shown here were conducted using PC-MATLAB (MathWorks, 21 Eliot St., Natick, MA 01760), an interactive program particularly suited to this sort of analysis.

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