

## **Part 8. Extensions and Other Comments on Methodology**

### **8.1. Introduction**

Many extensions and alternative approaches are possible for the class of experiments discussed in this monograph. Studies involving several groups allow further analysis and modeling to be conducted. In some studies, the treatments are ordered, again allowing extended analysis. We here discuss some general ideas for such extensions.

The statistical theory presented here has many similarities with the Jolly-Seber model for one group. Therefore, some existing methods have potential application to the extended treatment-control models we have examined. In Chapter 8.3, we consider designs for heterogeneous populations and age-specific treatment effects. Regression approaches have been used frequently in fisheries work for many decades and this approach is covered briefly under the first and unknown capture history protocols in Section 8.4.1. Finally, some experiments on small hydrodams have involved the release of dead fish as an integral part of the study. The theory for this unique case is covered in Section 8.4.2.

### **8.2. Studies with More Than Two Groups**

Most of this monograph involves analysis of recapture data from two groups of marked animals corresponding to treatment and control groups. An exception is Part 3, which deals with the situation where there are more than two groups and which presents statistical theory and an example (Chapter 3.10). However, the methods in Part 3 do not consider specific relationships between groups corresponding, for example, to treatment with increasing levels of a toxin or to animals in different size-classes. In the present chapter, we concentrate on methods of analysis that are appropriate when more than two groups have a specific "treatment structure."

It is not possible to give a detailed account of all the variations likely to arise or of the most appropriate analyses (which require PROC SURVIV or program RELEASE). We instead discuss two general situations and indicate how to use some basic results from program RELEASE for further analysis. We consider first the case where groups correspond to ordered levels of a single factor and then groups corresponding to two factors, each at two or more levels.

### 8.2.1. Ordered Treatments

8.2.1.1. *Direct modeling of treatment effect.* – We assume here that the initial release consists of  $V$  groups of marked animals, where the labels  $v = 1, 2, \dots, V$  correspond to ordered treatment levels. For example, we may have  $V = 3$  groups, where group 1 animals receive no treatment, and animals of groups 2 and 3 are dosed with lead at different levels.

Several difficulties are associated with determining an appropriate way to assess treatment effect. One concerns whether the effect is direct (acute) or indirect (see Chapter 1.5). If the effect is direct, it is reflected by a ratio of first-period survival rates; if it is chronic, the separation of treatment mortality from natural mortality is difficult (see Section 1.5.4), though the ratio (or difference) between treatment and control survival rates in some sense measures treatment-related mortality.

One way to study a dose-related treatment response is to build the relationship into the parameters of the model. Thus, assume that  $D_1, \dots, D_V$  represent increasing dose levels applied to groups  $v = 1, \dots, V$ , respectively. A model often used in dose-response studies to relate survival and dose is the logistic model (Cox 1970). The simplest way to incorporate this model in the context of a release-recapture study is to relate total survival to dose, making no attempt to partition treatment-related and natural mortality (except for direct effect).

The survival rate parameters of a general model, allowing for a chronic dose-related treatment effect persisting throughout the study, are

$$\phi_{vi} = \frac{1}{1 + \exp(-a_i + b_i D_v)},$$

$v = 1, \dots, V$  and  $i = 1, \dots, k - 2$ . Thus, the  $V(k - 2)$  survival rate parameters,  $\phi$ , are effectively replaced by the  $2(k - 2)$  parameters  $a_1, b_1, \dots, a_{k-2}, b_{k-2}$ . The remaining parameters are

$$p_{vi}, i = 2, \dots, k - 1, v = 1, \dots, V, \text{ and} \\ \phi_{v,k-1} p_{v,k}, v = 1, \dots, V.$$

This model uses a separate logistic structure for the survival of each group. Note that with this parameterization, if  $b_i = 0$  for some  $i$ , then survival  $\phi_{vi}$  is the same for all groups in period  $i$ . Thus, to test for a treatment effect on survival in period  $i$ , we test  $b_i = 0$  against  $b_i \neq 0$  or a one-sided alternative. The sequence of models  $H_0, H_{1\phi}, H_{2\phi}, \dots, H_{k-1,\phi}$  now has the following representation.

For model  $H_0$ ,  $b_1 = b_2 = \dots = b_{k-2} = 0$ ;  $p_{vi} = p_i, v = 1, \dots, V, i = 2, \dots, k - 1$ ;

$$\phi_{v,k-1} p_{vk} = \phi_{k-1} p_k, v = 1, \dots, V.$$

For model  $H_{1\phi}$ ,  $b_1 \neq 0, b_2 = \dots = b_{k-2} = 0; p_{vi} = p_i, v = 1, \dots, V, i = 2, \dots, k - 1;$

$$\phi_{v,k-1} p_{vk} = \phi_{k-1} p_k, v = 1, \dots, V.$$

For model  $H_{2p}$ ,  $b_1 \neq 0; p_{v2} \neq p_{v'2},$  all  $v, v'$  in  $1, \dots, V;$

$$b_2 = \dots = b_{k-2} = 0; p_{vi} = p_i, v = 1, \dots, V, i = 3, \dots, k - 1;$$

$$\phi_{v,k-1} p_{vk} = \phi_{k-1} p_k, v = 1, \dots, V.$$

⋮

For model  $H_{k-1,\phi}$ ,  $b_i \neq 0, i = 1, \dots, k - 2; p_{vi} \neq p_{v'i},$  all  $v, v', i = 2, \dots, k - 1;$

$$\phi_{v,k-1} p_{vk} \neq \phi_{v',k-1} p_{v'k}, \text{ all } v, v'.$$

Unfortunately, estimation under the models  $H_{1\phi}, \dots, H_{k-1,\phi}$  requires a computer routine more general than program RELEASE. Testing between any two models in the sequence is carried out by means of likelihood ratio tests, which are also beyond the capabilities of program RELEASE. PROC SURVIV of program RELEASE is used to provide input to program SURVIV (White 1983) for the appropriate analyses. In some applications, it may be appropriate to model the capture probabilities ( $p_{vi}$ ) as a logistic model based on effort or other variables. The above discussion applies largely to complete capture history (or scheme A) data because estimation with first capture histories is not possible under models more general than  $H_{1\phi}$  (see Chapter 2.2).

If the treatment effect is entirely direct, and if group 1 is a control or zero-dose group (i.e.,  $D_1 = 0$ ), a parameterization that separates treatment-related and natural mortality is

$$\phi_{v1} = S_v \phi_{11},$$

where

$$S_v = \frac{1}{1 + \exp(-a + b D_v)}, v = 2, \dots, V,$$

and

$$\phi_{vi} = \phi_i, i = 2, \dots, k - 2; p_{vi} = p_i, i = 2, \dots, k - 1; \phi_{v,k-1} p_{vk} = \phi_{k-1} p_k, v = 1, \dots, V.$$

Again, estimation is possible with first capture or complete capture history data but cannot be carried out for the above model by using program RELEASE. The test of no treatment effect, i.e., of  $b = 0$ , also needs a more general computer routine. Here again, the output of PROC SURVIV from program RELEASE can be modified for input to program SURVIV. Other programs for this type of modeling were discussed by North and Morgan (1979), White (1983), Conroy and Williams (1984), and Clobert et al. (1985).

*8.2.1.2. Indirect modeling of treatment effect.* – We now discuss alternative (and less satisfactory) procedures that can be applied to the output from program RELEASE. Tests carried out in RELEASE to compare models (see TEST 1, Chapter 2.1) provide some indication of how prolonged the treatment effect is. However, each component of TEST 1 tests the null hypothesis of homogeneity of some parameters across all groups against the general alternative that not all groups are the same. The tests are not designed to be sensitive to the types of alternatives expected with ordered groups, such as an ordering of group-specific survival rates. To achieve greater power or sensitivity, a test designed specifically for ordered alternatives (see Cochran 1954; Armitage 1955) can be applied to data in each of the  $V \times 2$  contingency tables, which are printed out by program RELEASE as components of TEST 1.

Application of tests for ordered alternatives is illustrated here for TEST 1.R1, which is based on the  $V \times 2$  contingency table with rows  $(r_{v1}, R_{v1} - r_{v1})$ ,  $v = 1, \dots, V$  (see Section 2.1.2). When the row labels of this table correspond to increasing dose levels  $D_1, \dots, D_V$ , an alternative to the null hypothesis of no treatment effect states that survival  $\phi_{v1}$  is a decreasing function of dose  $D_v$ . For a direct treatment effect, this corresponds to stating that the probability of recapture (estimated by  $r_{v1}/R_{v1}$ ) decreases as  $D_v$  increases. If the relation of  $\phi_{v1}$  to  $D_v$  (or to scores  $Z_v$  related to  $D_v$ , e.g.,  $Z_v = \ln D_v$ ) is thought to be approximately linear, the Cochran-Armitage test for a linear trend in proportions is appropriate. In TEST 1.R1, the proportions of interest are the recapture probabilities  $E(r_{v1})/R_{v1}$ ,  $v = 1, \dots, V$ . Computational details were given by Cochran (1954), Armitage (1955), and Snedecor and Cochran (1980, Section 11.9). The test statistic is chi-square with 1 df, which is more sensitive to a trend in the proportions  $E(r_{v1})/R_{v1}$  than is the nonspecific contingency chi-square with  $V - 1$  degrees of freedom computed by TEST 1.R1.

The Cochran-Armitage test is most effective when the proportions  $E(r_{v1})/R_{v1}$  are linearly related to doses  $D_v$  (or some appropriate transformation,  $Z_v$ , of  $D_v$ ). An alternative approach may be needed when the relationship is not linear – e.g., when there is a threshold dose below which treatment has no effect. In this situation, a useful procedure is to partition the  $V \times 2$  tables for TEST 1.R1 into a series of  $2 \times 2$  tables, each yielding a chi-square with 1 df. One possible partitioning gives the series of tables

First table:

$r_{11}$	$R_{11} - r_{11}$
$r_{21}$	$R_{21} - r_{21}$

Second table:

$r_{11} + r_{21}$	$R_{11} + R_{21} - r_{11} - r_{21}$
$r_{31}$	$R_{31} - r_{31}$

V - 1 table:

$r_{11} + \dots + r_{V-1,1}$	$R_{11} + \dots + R_{V-1,1} - r_{11} - \dots - r_{V-1,1}$
$r_{V1}$	$R_{V1} - r_{V1}$

These and other series of  $2 \times 2$  tables can be used to combine the  $V$  groups into a smaller number of "classes" within each of which survival and recapture rates appear to be homogeneous. An example of such a series of tests is given in Chapter 7.6 for recapture data on tortoises. A useful discussion of partitioning chi-squares for a  $V \times 2$  table was given by Cochran (1954, Section 6.1).

Tests that take into account the ordering of the  $V$  groups should be applied in a similar manner to the other components of TEST 1. These test results should then be used to select the most appropriate model in the sequence  $H_0, H_{1\phi}, \dots, H_{k-1,\phi}$ . If group-specific survival estimates can be computed under the chosen model for the type of data available, these estimates would then be examined for dose-related trends.

Thus, with first capture history data, if model  $H_{1\phi}$  fits and if group 1 at dose  $D_1$  is a control, the period 1 survival rate ratios (relative to group 1), i.e., the  $\hat{S}_v, v = 2, \dots, V$ , can be examined for trends. Unfortunately, regression analysis (with a linear or nonlinear model such as the logistic model) to relate  $\hat{\phi}_{v1}$  or  $\hat{S}_v$  to  $D_v$  is not straightforward, because the usual regression assumptions concerning independent observations with common variance do not hold; the  $\hat{S}_v$  are correlated and have different (not equal) sampling variances. We are not aware of any packaged routine that will straightforwardly perform the appropriate weighted regression analysis for the case where the error variance-covariance matrix (or the weighting matrix) is not diagonal. An additional problem is that these variances and covariances are unknown, and their estimates produced by program RELEASE may not be reasonably accurate unless sample sizes are large. Relating  $\hat{\phi}_{v1}$  or  $\hat{S}_v$  to  $D_v$  by regression analysis will be approximate and possibly crude. One such approach to a nonlinear regression of  $\hat{\phi}_{v1}$  or  $\hat{S}_v$  on  $D_v$  (e.g., with the logistic model) is to ignore covariances between estimates and carry out the weighted regression, weighting by the inverse of the estimated variance produced by RELEASE.

With complete capture history data, correlations among the  $\hat{\phi}_{vi}$  can be avoided by using estimates from a sufficiently general model. Thus,  $\hat{\phi}_{v1}, v = 1, \dots, V$  are uncorrelated under  $H_{2\phi}$  and more general models (see Part 3). However, using a general model can result in unnecessary loss of precision; thus, this approach is not satisfactory either.

### 8.2.2. Two Factors Each at Two or More Levels

Consider a release-recapture study where the releases of treatment and control groups consist of individuals in four size-classes, marked accordingly. We can regard this study as two-factor:  $V = 8$  groups in a  $2 \times 4$  classification with treatment at two levels and size-class at four levels. Other examples of studies with two factors each at several levels include treatment and control groups released at each of several locations (see Chapter 7.3), and several treatment levels applied to different sex or age-classes (see Chapter 7.2). In all these examples we can impose a definite structure upon the group labels  $v = 1, \dots, V$ ; this structure should not be ignored.

Analysis procedures are generalizations of those described for ordered treatments (Section 8.2.1), and again involve two different approaches. The first approach is to build the relationship between survival and treatment structure into the model parameters; the second approach is to obtain survival estimates, ignoring treatment structure and using program RELEASE, and then determine if in some way these estimates reflect the treatment structure. As noted in Section 8.2.1, the first method is beyond the capabilities of RELEASE, but is statistically sound; the second method is, at best, approximate.

Comments in Section 8.2.1 concerning direct and indirect effects and related models apply here also. Thus, a model that allows for a prolonged or chronic effect, for the study with treatment and control and four size-classes, has

$$\phi_{vi} = \frac{1}{1 + \exp(-a_i + b_i D_v + c_i X_v + d_i D_v X_v)}, \quad (8.2.1)$$

$$v = 1, \dots, V,$$

$$i = 1, \dots, k - 2,$$

where  $D_v = 0$  or 1 for control and treatment, respectively, and  $X_v$  has four values corresponding to the midpoints of the four size-classes. More generally,  $D_v$  and  $X_v$  represent levels of two factors, one of which may be the dose of a toxin and the other a covariate such as size. A test of  $b_i = c_i = d_i = 0$  is a test of the null hypothesis that survival in period  $i$  is not affected by either of the two factors. A test of  $b_i = d_i = 0$  is a test of no effect on survival by factor 1 in period  $i$ .

The first type of analysis would estimate  $a_i, b_i, c_i, d_i$  using a model with  $\phi_{vi}$  defined as in 8.2.1. Tests concerning  $b_i, c_i,$  and  $d_i$  would be likelihood ratio tests. The second type of analysis would use estimates  $\hat{\phi}_{vi}$  and variances and covariances from RELEASE in a nonlinear weighted regression with  $\hat{\phi}_i = E(\hat{\phi}_{vi})$ , as given by the equation above to obtain  $\hat{a}_i, \hat{b}_i, \hat{c}_i,$  and  $\hat{d}_i$ . Note that this second approach involves estimating a larger number of parameters than the first.

Examples of studies with two factors are given in Chapter 7.2. However, in both of these examples, only first capture histories are available; thus, unless the treatment effect is largely

direct, it cannot be estimated satisfactorily. In each example, the data were partitioned into subsets, each containing a treatment and control group so that estimates  $\hat{S}$  produced by RELEASE for different subsets were uncorrelated. The analyses used correspond to the second type of approach described here. However, there will be little difference between the two types of approaches for these examples because estimation with models more general than  $H_{1\phi}$  is not possible and because the  $\hat{S}_i$  are independent.

### 8.3. Testing for Treatment Effects in a General Capture-Recapture Setting

In this monograph we have concentrated on the situation where released, marked animals are recaptured. This allows estimation of survival and capture rates with primary emphasis on comparisons of survival rates between treatments. More generally, however, we may also obtain information on unmarked animals as part of the capture process. In these cases, we may also obtain estimates of population size and recruitment number for each period and test for effects on these abundance parameters.

A fisheries example might involve fish populations in small experimental ponds subject to different treatments (e.g., a control pond versus a pond subject to mild acidification). In some experiments, one pond may be used for each treatment; however, several ponds probably should be allocated to each treatment so that a valid estimate of experimental error can be obtained (see Part 4). Fish could be captured and marked as the study progressed so that survival rates, capture rates, population sizes, and recruitment numbers could be estimated. This chapter (i.e., estimation of population size) is not applicable when fish are migrating in a large river system.

As an illustration, suppose we have the situation described in Part 2 where there are two treatments with no replication. To begin with, let us assume that all the parameters are distinct so that we have

	<u>Treatment group</u>	<u>Control group</u>
Survival	$\phi_{t1}, \dots, \phi_{t,k-1}$	$\phi_{c1}, \dots, \phi_{c,k-1}$
Capture	$p_{t1}, \dots, p_{tk}$	$p_{c1}, \dots, p_{ck}$
Population size	$N_{t1}, \dots, N_{tk}$	$N_{c1}, \dots, N_{ck}$
Recruit. number	$B_{t1}, \dots, B_{t,k-1}$	$B_{c1}, \dots, B_{c,k-1}$

When treatment is determined by the investigator, the  $N_{v1}$  will be known; and subsequent  $N_{vj}$  are just survivors from earlier releases. Moreover, in this situation, recruitment is known:  $B_{vj}$  are the known releases to treatment groups  $v$ , at time  $j$ , of previously unmarked animals. If unmarked animals are being caught and used in the study, there can be contexts in which  $N_{vj}$  and  $B_{vj}$  are of interest, especially if one defines being unmarked as a treatment category, or if population totals  $N_j$  and  $B_j$  are of interest. The alternative situation is that treatment is some naturally occurring distinction, such as strain (see Manly 1985). Then  $N_{vj}$  and  $B_{vj}$  are meaningful parameters to be estimated.

Recall from Chapter 2.4 that we can estimate  $\phi_{u_i}, \phi_{c_i}$  for  $i = 1, 2, \dots, k - 2$  and  $p_{u_i}, p_{c_i}$  for  $i = 2, \dots, k - 1$ . A simple application of results for the general Jolly-Seber model (Seber 1982:200) provides estimates for population sizes

$$\hat{N}_{u_i} = \frac{n_{u_i}}{\hat{p}_{u_i}} \text{ and}$$

$$\hat{N}_{c_i} = \frac{n_{c_i}}{\hat{p}_{c_i}}, \text{ for } i = 2, 3, \dots, k - 1,$$

and for recruitment numbers

$$\hat{B}_{u_i} = \hat{N}_{u_{i+1}} - \hat{\phi}_{u_i}(\hat{N}_{u_i} - n_{u_i} + R_{u_i})$$

and

$$\hat{B}_{c_i} = \hat{N}_{c_{i+1}} - \hat{\phi}_{c_i}(\hat{N}_{c_i} - n_{c_i} + R_{c_i}) \text{ for } i = 2, 3, \dots, k - 2.$$

Note that  $n_{u_i} = m_{u_i} + u_{u_i}$  is the number of treatment animals captured in sample  $i$ , of which  $u_{u_i}$  are unmarked and  $m_{u_i}$  are marked. Also,  $R_{u_i}$  is the number of the  $n_{u_i}$  released at time  $i$ . There are similar definitions for  $n_{c_i}$  and  $R_{c_i}$ .

It is important to emphasize that the form of the estimators  $\hat{N}_{u_i}, \hat{N}_{c_i}, \hat{B}_{u_i}$ , and  $\hat{B}_{c_i}$  remain unchanged for all the models in the sequence described in Chapter 2.4. All one needs to do is to use the appropriate  $\hat{p}_{u_i}$  and  $\hat{\phi}_{u_i}$  in the estimators (assuming such appropriate  $p_{u_i}$  exist - see below). Testing between models involves only marked animals, so that all the model sequence tests in Chapter 2.4 still apply with few modifications (the  $u_{u_i}$  effect TEST 3). Those modifications have been built into RELEASE.

It is possible to derive approximate variances and covariances for population size and recruitment number estimates for any of the models in Chapter 2.4; however, we do not present them here. On the basis of these variances and covariances, approximate normal test statistics could be used to test appropriate hypotheses.



Additional complications arise in estimating  $N$ . It is often difficult to interpret what population size means and to what area it applies. Given that we know what our reference population is, then a sample of unmarked animals must be taken along with the marked ones if population size is of interest. Moreover, we must know how the capture probability of unmarked animals relates to the  $p_{wi}$  for marked animals. If "treatment" really means strain (or some other naturally occurring distinction: for example, Manly 1985), then we have  $u_{wi}$  well defined at the time of capture and thus  $p_{wi}$  may apply equally to marked and unmarked animals of strain  $v$ . If treatment is imposed by the investigator, then, at the time of capture of  $u_i$  unmarked animals, those animals are not distinguished by treatment. One then (randomly) allocates these animals to treatment groups. At that point, the  $u_{wi}$  are defined but the recapture probabilities at time  $i$  do not depend on the (subsequent) treatment status of these new captures.

If treatment is imposed by the investigator, then we can partition the population size  $N_i$  into  $V + 1$  classes by letting unmarked animals also be a "treatment" class; hence,

$$N_i = N_{wi} + \sum_{v=1}^V N_{vi}.$$

The  $N_{wi}$  are just the number of marked survivors, by treatment group; thus we actually have

$$N_i = N_{wi} + \sum_{v=1}^V M_{vi},$$

and hence

$$\hat{N}_i = \frac{u_i}{\hat{p}_{wi}} + \sum_{v=1}^V \frac{m_{vi}}{\hat{p}_{vi}}.$$

Capture probabilities of unmarked animals cannot be estimated except by knowing how they relate to capture probabilities for some segment of marked animals; that assumed relationship cannot be tested. The simplest case is that we find  $p_{wi} = p_i$  acceptable and then also assume  $p_{vi} = p_i$ . Then

$$\hat{N}_i = \frac{n_i}{\hat{p}_i},$$

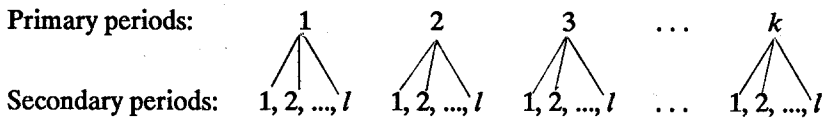
so we get an estimate of total population size. The partition of this  $\hat{N}_i$  into treatment classes is entirely an artifact of allocating captured animals to treatments. As such, estimation (and

testing) of the  $N_{\alpha i}$ ,  $N_{\alpha}$  is of almost no interest, and requires a restrictive model. We conclude that inference on population size and recruitment is only of interest when treatment is some naturally occurring distinction. Otherwise, the total emphasis is, properly, on survival effects of treatment.

8.3.1. Robust Designs that Allow for Heterogeneity and Trap Response

Here we assume that there are  $k$  distinct sampling periods in our study design and that the "treatment" is a naturally occurring distinction. These periods are far enough apart to make it essential that allowances be made for additions (births, immigrants, etc.) and deletions (deaths, emigrants, etc.) between the periods. The models used are based on the Jolly-Seber model for open populations. An alternative approach consists of a design and analysis that incorporates the better features of closed and open models (Lefebvre et al. 1982; Pollock 1982). The unequal catchability could involve inherent heterogeneity of capture probabilities between different animals and trap response, which involves increasing or decreasing probabilities of capture as a result of being marked.

Consider the following representation of a capture-recapture sampling experiment based on Pollock (1982) where there are  $k$  primary sampling periods (e.g., years), and within each of these, there are  $l$  secondary sampling periods that are close to each other in time (e.g.,  $l$  consecutive days).



If there are two treatments as before, the biologist will be interested in the population sizes for each of the primary sampling periods ( $N_{t1}$ , ...,  $N_{tk}$ ,  $N_{c1}$ , ...,  $N_{ck}$ ), assuming that the population is constant over the secondary sampling periods within a primary sampling period. Interest lies in survival probabilities ( $\phi_{\alpha i}$ ,  $\phi_{\alpha}$ ,  $i = 1, \dots, k - 1$ ) and recruitment numbers ( $B_{\alpha i}$ ,  $B_{\alpha}$ ,  $i = 1, \dots, k - 1$ ) between the primary sampling periods.

Assuming that the population is closed over the secondary sampling periods within a primary period, then two estimation procedures are possible. In the first procedure, all the secondary sampling periods within a primary period would be "pooled" (i.e., one would merely count the animals seen at least once in the primary period). These pooled samples would then be analyzed by the model described in the previous section. Heterogeneity and trap response can have a large effect on the estimators of population size (Cormack 1972; Carothers 1973). We suggest a modified procedure as follows.

- (1) Obtain population size estimators for ( $N_{\alpha i}$ ,  $N_{\alpha}$ ,  $i = 1, \dots, k$ ) using closed population models that allow for unequal catchability (Otis et al. 1978; White et al. 1982) and are based only on the captures and recaptures within a primary sampling period.

- (2) Obtain survival rate estimators for  $(\phi_{i1}, \phi_{c1}, i = 1, \dots, k - 2)$  using the "pooled" data and the Jolly-Seber model because survival rates are much less affected by unequal catchability.
- (3) Obtain recruitment number estimators using  $\hat{B}_{i1} = \hat{N}_{t,i+1} - \hat{\phi}_{i1}(\hat{N}_{i1} - n_{i1} + R_{i1})$  as before; however, now the population size estimators are the "robust" versions described previously. Note that  $\hat{B}_{i1}$  can be obtained for  $i = 1, \dots, k - 2$  and that a similar expression is available for  $\hat{B}_{c1}$ .

This strategy is not applicable when fish are moving downstream or when capture of unmarked animals is ignored.

### 8.3.2. Testing for Age-Specific Treatment Effects

Pollock (1981a) developed an extension of the Jolly-Seber model for populations with different identifiable age categories (see also Pollock and Mann 1983 for a fisheries example). He allowed the different identifiable age categories to have different survival and capture probabilities, and found explicit maximum likelihood estimators. The estimators take a form similar to those for the Jolly-Seber model. A computer program, JOLLYAGE, is available for these computations from the U.S. Fish and Wildlife Service, Patuxent Wildlife Research Center, Laurel, Maryland 20708, USA (see Brownie et al. 1986).

In our framework of comparing treatments, this model could be useful. It would be informative to examine possible interactions between treatment and age in terms of survival rates. In a toxicology study on birds, for example, young birds dosed with lead shot might be influenced either more or less than adult birds dosed with the same amount of lead shot.

## 8.4. Other Procedures

### 8.4.1. Regression Methods

Regression methods have been suggested as an approach to estimating treatment survival under the first and unknown capture history protocols. From the expected values for this protocol (Section 2.2.2), under  $H_{1\phi}$ , we have

$$E(m_{t1i}/R_{t1}) = S E(m_{c1i}/R_{c1}),$$

where  $i = 1, 2, \dots, k$ ,  $S = \phi_{t1}/\phi_{c1}$ . This relationship suggests the regression model

$$\frac{m_{t1i}}{R_{t1}} = b \frac{m_{c1i}}{R_{c1}} + \epsilon_i,$$

leading to the estimation of the slope term  $b (= \hat{S})$  by least-squares methods.

The regression approach is poor relative to the ML approach because the main assumptions of simple linear regression are violated. The general problem falls under the title "errors in variables" in the literature (see Wald 1940 and Bartlett 1949 for early investigations of this problem). In the regression model above, the independent variable is not known without error, rather it is an estimated binomial proportion. This approach causes a bias in the estimator of the regression slope (Kendall and Stuart 1967; Snedecor and Cochran 1967; Johnston 1972). The effect of sampling variation in the independent variable is to diminish the estimated slope of the regression line. Thus, the least-squares estimator of the slope (= treatment survival rate) will be biased negatively.

The second problem is more serious in that a high correlation exists between the residuals and the values of the independent variable. Thus, the variance of the  $\epsilon_i$  is not constant (see Draper and Smith 1981 for references on the analysis of residuals in regression). In addition, the  $\epsilon_i$  are correlated with each other under multinomial sampling. This correlation produces an inconsistent estimator of the slope (Wonnacott and Wonnacott 1970; Neter and Wasserman 1974). Because consistency is perhaps the most fundamental property of an estimator, the lack of consistency in this application of regression makes the procedure undesirable (also see Johnston 1972:278).

The dependent variable is also an estimated binomial proportion and this affects  $\text{var}(\epsilon_i)$ . Thus, the variance in the dependent variable, given the independent variable, will not be constant over dams and violates the final assumption of regression analysis. Therefore, the estimator of the slope lacks the minimum variance property that is usually associated with least squares. A weighted regression might be an improvement if the weights were known, but the issues noted previously are more serious.

In summary, a simple linear regression analysis of the estimated recapture rates produces estimates of the treatment survival rate that are biased, lack the minimum variance property, and are not consistent. Consequently, we do not recommend this procedure; better estimation methods exist.

#### 8.4.2. Release of Dead Fish

In this section we consider the case of a small low-head dam (Olson et al. 1985) where the survival of fish passing a turbine or a spillway is of interest. Conceptually, treatment and control fish are batch-marked and a sample of these fish is captured at a single site, usually close to the dam (e.g., 100 to 300 m downstream), with a single large net or several smaller nets. The intent is to catch only live fish – either control fish or fish that survived passage through the turbine. However, because the nets are so close to the dam, there is concern that some turbine-killed fish might also enter the net. Finally, because the nets are in place for a

period of time, it is possible that some fish (either treatment or control) die in the net, and these fish cannot be distinguished from the treatment fish that entered the net dead.

Some authors have tried to separate these confounded data by releasing a number of dead fish ( $R_d$ ) along with the treatment fish just above the dam (e.g., into the turbine intake or spillway). Of course, the  $R_t$  and  $R_d$  fish must be otherwise as identical as possible. The recaptures of these dead fish ( $r_d$ ) are used in an effort to estimate treatment survival. We attempt to present a rationale for this type of experiment and provide an estimator of the treatment survival and its sampling variance. However, some restrictive assumptions are required and the ability to test the validity of these assumptions is largely lacking.

We denote the number of released treatment, control, and dead fish as  $R_t$ ,  $R_c$ , and  $R_d$ , respectively, and the numbers of these fish recaptured at the single downstream sampling site as  $r_t$ ,  $r_c$ , and  $r_d$ , respectively. However, we use the further subscripts  $l$  (live) and  $d$  (dead) to denote whether a fish, released alive, was alive or dead when it entered the net. For treatment fish, "alive" upon capture means that the fish was not seriously injured by the turbine, and thus, was not fated to die as a direct result of the turbine. The expectations of these five random variables are

$$E(r_{tl}) = R_t S \phi_t p_{tl} ;$$

$$E(r_{td}) = R_t (1 - S) \phi_{td} p_{td} + R_t S (1 - \phi_t) p_{td} ;$$

$$E(r_{cl}) = R_c \phi_c p_{cl} ;$$

$$E(r_{cd}) = R_c (1 - \phi_c) p_{cd} ;$$

$$E(r_d) = R_d \phi_d p_d .$$

The parameters are conditional capture probabilities (given that a fish reaches the downstream site where the net is),  $p_{tl}$ ,  $p_{td}$ ,  $p_{cl}$ ,  $p_{cd}$ ,  $p_d$ ; the treatment survival  $S$ ; and survival probabilities from the tailrace to the site where the net is set,  $\phi_t$ ,  $\phi_c$ ,  $\phi_{td}$ ,  $\phi_d$ . For fish released dead,  $\phi_d$  is the probability that a carcass reaches the net, whereas  $\phi_{td}$  is the probability that a carcass of a fish killed by the turbine reaches the net.

Although the above equations are not as general as they should be to be conceptually valid, they are already too general in the sense that several restrictive assumptions must be imposed to gain identification of the treatment survival rate  $S$ . We must assume that  $\phi_{td} = \phi_d$  and that  $p_{td} = p_d$ ; thus, dead fish have the same parameters, whether they were released dead or killed by the turbine. It is also necessary to assume that capture probabilities of live fish are the same for treatment and control fish:  $p_{tl} = p_{cl} = p_l$ .

Expressions for the expected number of treatment, control, and dead fish netted, given these added assumptions, are

$$E(r_t) = R_t[S\phi_t p_l + (1-S)\phi_d p_d + S(1-\phi_t)p_d];$$

$$E(r_c) = R_c[\phi_c p_l + (1-\phi_c)p_{cd}];$$

$$E(r_d) = R_d\phi_d p_d.$$

Only if  $\phi_t = \phi_c = 1$  will these expressions simplify enough to render  $S$  estimable. Hence, we must further assume  $\phi_t = \phi_c = 1$ , which means no natural mortality between the tailrace and the net. This seems reasonable if the sampling nets are only 100 m downstream. The value of  $p_{cd}$  is irrelevant when  $\phi_c = 1$ . Only the product  $\phi_d p_d$  is estimable, which is all that is needed. Hence, without loss of generality, one can set  $\phi_d = 1$ . The three expectations then become

$$E(r_t) = R_t[S p_l + (1-S)p_d];$$

$$E(r_c) = R_c p_l;$$

$$E(r_d) = R_d p_d.$$

The moment estimator of  $S$  is obtained by solving the above equations, which yield

$$\hat{S} = \frac{(r_t/R_t) - (r_d/R_d)}{(r_c/R_c) - (r_d/R_d)}.$$

For the binomial model, the estimator above is the MLE.

The sampling variance of  $\hat{S}$  is estimated as

$$\hat{\text{var}}(\hat{S}) = (\hat{S})^2 \left[ \frac{\text{var}(A)}{A^2} + \frac{\text{var}(B)}{B^2} - 2 \frac{\text{cov}(A, B)}{AB} \right],$$

where  $A = (r_t/R_t) - (r_d/R_d)$  and  $B = (r_c/R_c) - (r_d/R_d)$ . For the binomial model, the theoretical sampling variance of  $\hat{S}$  is

$$\hat{\text{var}}(\hat{S}) = (\hat{S})^2 \left[ \left( \frac{r_t}{AR_t} \right)^2 \left( \frac{1}{r_t} - \frac{1}{R_t} \right) + \left( \frac{r_c}{BR_c} \right)^2 \left( \frac{1}{r_c} - \frac{1}{R_c} \right) - 2 \frac{r_d(R_d - r_d)}{(R_d)^3} \left( \frac{1}{A} - \frac{1}{B} \right)^2 \right].$$

If  $r_d = 0$  (i.e., no dead fish are recovered), these estimators simplify to those presented in Section 2.2.6.

Few analytical alternatives exist for this particular experiment. Clearly, some strong assumptions must be met for this method to be useful. The use of unique marks adds nothing to the analysis of such experiments. If this procedure is used, replication is needed.