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A RELIABILITY ANALYSIS OF OCCUPATIONAL EXPOSURE DATA USING A FAMILY OF PROPORTIONAL AND ADDITIVE HAZARD FUNCTIONS

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ABSTRACT

The growing debate on regulation in the work place calls for an improvement in the analytic techniques used to assess occupational risks and mortality. The intent of this article is to investigate a family of hazard models that may be employed in this effort. The underlying problem is first placed in context with a discussion of risk attitudes and the dangers of exposure. Next, proportional and additive hazard models are introduced which combine a standard (but unknown) hazard function with a set of explanatory variables. The family is developed with the aid of a parameterized link function, while maximum likelihood is used to obtain coefficient estimates. After describing the methodology an application is given for the case where only two samples of grouped data are available. A related example is then worked out using survival data collected on persons employed in the asbestos processing industry. The results confirm both the flexibility and sensitivity of the approach while leaving open the possibility of further refinements.

When an activity or substance is deemed risky, implicit in the judgement is the likelihood that exposure will produce some amount of harm or damage. Accordingly, risk can be defined as the probability of incurring a unit loss per unit time [1], where loss may be measured in terms of injuries, monetary penalties, or total social costs. This definition then involves the integration of probability of occurrences, the severity or magnitude of the exposure or event, and the range of resultant personal injuries and related costs. The determination of insurance premiums for workers engaged in dangerous occupations such as coal mining illustrates these components. Rate structures are based on the frequency of accidents, the distribution and size of their occurrence, and the

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extent of personal loss [2]. Discussions of safety and exposure potential often fail to make these distinctions although they are crucial in any decision analysis.

From a public point of view, many of the programs undertaken by government have the dual objective of increased protection and risk reduction for the individual. The benefits of such programs are often difficult to determine, though, because of a lack of information on the valuation people place on a safer environment. In addition, an incomplete understanding of the relationships between exposure and damage tends to undermine policy formulation and strain consensus. For a more thorough discussion of risk assessment and the specifics of data requirements see Bard [3], Fischoff [4], and Sage and White [5].

Because of the basic data limitations, it is important that the most sensitive analytical techniques be used in gauging risk. A procedure frequently employed in the analysis of occupational mortality data compares the number of deaths observed in the study population with the number expected, based on some control group. More detailed approaches designed to take into account intervening variables have also been investigated using markov chains (e.g., see Shachtman, *et al.* [6]).

A third procedure extending Cox's notion of proportional hazard models [7] has recently proven effective in fitting nonlinear forms (e.g., see Hennessey [8]). The purpose of this article is to examine a family of these models in an attempt to quantify the risks associated with certain hazardous occupations. In so doing, we will present a methodology based on grouped data and the corresponding interval approximations. To begin the underlying problem is placed in context by discussing the nature of risk. Some of the issues surrounding the biological effects of exposure and the need for a better understanding of dose-response relationships will be highlighted. Finally, an application based on the two-sample case and an example drawn from asbestos workers' survival data will be discussed. The model is shown to provide a good fit for a range of parameter values.

NATURE OF RISK

The first component of risk, probability of occurrence, has three major facets: space, population, and time dependency. The spatial characteristics of exposure can range from local damage to world-wide catastrophe. For example, the persistent and highly mobil nature of some waste streams pose proximate risks to soil fertility and water supplies, while deposits from acid rain have a global aspect. A second facet of exposure probability is determined by population traits. In many situations, selected group characteristics may be identified as the determinants of the risk bearing population. Among these are heredity, age, occupation, and sex. Time dependence may be classified as either continuous or periodic (as well as cumulative or noncumulative) to distinguish whether a hazard exists over a substantial time horizon and whether effects of separate exposure to the hazard for various doses are cumulative.

Generally, risks are evaluated intuitively rather than subjected to an explicit analysis. For the case of the individual who is contemplating a potentially dangerous activity where the outcome will affect him alone, he is wont to use a personal value system to evaluate the possible loses. When the risks have wider ranging consequences, the criteria are likely to be different. Lowrance points out some of the issues that surface in public policy making where a different value system is applied. These fall into the following nonmutually exclusive categories [9]:

- technically complex risks comprehensible only to highly trained people;
- risks that can be significantly reduced by applying technology;
- risks that constitute public problems and whose technical components need to be distinguished explicitly from their social and political components so that their responsibilities are assigned properly;
- risks whose possible consequences appear to be so grave or irreversible that prudence dictates the urging of extreme caution, even before the risks are known precisely; and
- risks that result from technological intrusions on personal freedom that are made in pursuit of safety.

Many of these issues are marked by an aggregation of preferences, limited knowledge, uncertainty, irreversibility, and the distribution of benefits versus costs. In trying to formulate acceptable social policy, it is often difficult to separate these factors from individual interpretations and biases.

BIOLOGICAL EFFECTS OF EXPOSURE

An individual's perception and appreciation of risks and benefits vary widely (e.g., see [10-12]). When one has "lived with" the risk (as does a toxic chemical worker, for instance) and when the activity is an accepted practice of society, awareness of it may be less than for risks of unfamiliar activities. The time lag between exposure to the hazard and the occurrence of injury also affects the individual's perception.

This is a vital point in light of the persistent and bioaccumulative nature of many hazardous materials. The risks of drinking water contaminated with trace metals would seem far less acceptable if the health consequences were experienced immediately. It is a common tendency to heavily discount future risks both to ourselves and succeeding generations.

Toxicity

Any substance if taken in large enough concentrations will produce adverse health effects. Although the relationships between individual exposures and the degrees of injury for many substances are not known precisely, and are difficult to quantify, they do possess certain general characteristics. For many types of

hazards two thresholds may be defined. The first places a lower bound on exposures below which no damage will be experienced; the receiving medium responds elastically. The second provides an upper bound above which injury or death will occur. For those situations where the two thresholds are relatively close in value (such as for brain damage resulting from the inhalation of carbon monoxide) the body is said to be quite resilient [9]. This implies that recovery from a nonlethal dose is usually rapid.

In actuality, more than two thresholds may exist, each being a function of the particular receiving medium and dosage. As an example, consider the biological effects of cadmium on humans [13]. Basically, cadmium is a highly toxic element which reaches the body through the food chain and accumulates in various organs such as the liver. It is identified as a stock pollutant because it doesn't degrade in the environment; its toxicity is a direct function of body weight. At fairly low densities it has been implicated in renal dysfunction and cardiovascular disease; in massive doses its effects are dramatic, producing a softening and eventual collapse of the skeletal structure.

Dose-Response Relationships

The amount of a substance required to cause harm varies with its chemical and physical properties and the sensitivity of the affected organism. Thus, the dose of one chemical required to produce death in a particular species, say a guinea pig, will be different from that of another chemical for the same species; the lethal dose of one chemical also will vary among species (e.g., monkey, dog, and human). In addition, not all animals or humans respond to the same concentration or exposure route in a similar fashion. Nitrates in water, for example, can be ingested by an adult human with no adverse effect, but for an infant the same concentrations can be fatal. Finally, some chemicals are toxic if inhaled, but present no risk if taken internally or applied to the skin.

The common measure of acute toxicity is LD_{50}, the amount (mg/kg body weight) that is lethal for 50 percent of the test population. Differences in harmful concentrations are particularly significant when considering constituents of hazardous materials, as illustrated in Table 1. All of the compounds presented in this table have been declared hazardous by the Environmental Protection Agency (EPA) based on results from controlled studies with rats centering on toxicity, carcinogenicity, mutagenicity, and teratogenicity [14]. The amount of each substance that is seen to cause death in 50 percent of the test population varies greatly and ranges from 3 mg/kg for cyanide to 5000 mg/kg for toluene. It is interesting to note that the lethal dose for a commonly used product – table salt – is equal to 3000 mg/kg, an amount less than that for toluene. Because of the lack of evidence that salt is carcinogenic, mutagenic or teratogenic, however, it has not been designated a hazardous substance.

The quality of an adverse effect (i.e., immediate death, reversible or irreversible illness) is also influenced by the exposure period. For some types of

Table 1. Toxic Doses for Selected Hazardous Materials

Compound	LD_{50}[a]
Cyanide	3
Phenylmercuric Acetate	30
Dieldrin	46
Pentachlorophenol	50
DDT	113
Naphthalene	1780
Saccharin	1820
Toluene	5000

[a] Amount (mg/kg body weight) that is lethal for 50 percent of the test population of rats following oral administration.

substances unfavorable consequences may not be observed or may be much less serious when the same dose is encountered over a long time period. Although table salt can produce severe effects at extremely high doses, small amounts ingested over a lifetime do not present health problems, except for extremely sensitive individuals. The opposite problem of persistence is taken up in the next section.

MODEL DEVELOPMENT

A standard approach to assessing mortality in an occupational setting is to compare the number of deaths observed, O, with the number expected, E, as inferred from a control population. The quantity $100 \times O/E$ is known as the standardized mortality ratio (SMR). Under the null hypothesis, the test statistic $(O - E)^2/E$ is asymptotically chi-square distributed with one degree of freedom (df). This method of analysis can be applied to the entire population, but may be more informative if restricted to a specific segment such as those persons who have experienced a certain level of exposure.

As an alternative to the SMR approach, we will investigate a procedure based on an extension of the proportional hazard models first proposed by Cox [7]. The procedure involves comparing exposures received by workers who either die or suffer a particular disease with those who remain healthy but continue to be at risk. Here, "comparable" is used in a general sense to mean similar with respect to age and other variables, which would, of course, need to be specified precisely in any given analysis.

To begin, some basic notation along with the general form of the model will be presented.

Notation

Let (t_j, δ_j) , $j = 1, \ldots, n$, be an observed sample of failure times with $\delta_j = 0$ indicating a right-censored observation and $\delta_j = 1$ indication a failure. It will be assumed that each individual has an associated (known) vector of covariates or explanatory variables $z_j = (z_{1j}, \ldots, z_{pj})$ which are time-independent, and that the underlying failure time density $f(t:z)$ is continuous. The survivor or reliability function will be denoted by $S(t:z)$ and the corresponding hazard function by $h(t:z) = f(t:z)/S(t:z)$.

Grouping of Continuous Data

In many instances the only survival data available are tabulated by group for a given epoch, rather than by individual at a discrete point in time. In order to accommodate this situation it will be necessary to partition the time axis accordingly. Let I_i , $i = 1, \ldots, m$, be the resulting intervals, where $I_i = [t_{i-1}, t_i)$, $t_0 = 0$ and t_m is defined to be greater then the last failure. The conditional probability, call it θ_i, that an individual fails during the i^{th} study interval, I_i, given that he was alive at time t_{i-1} may be expressed as

$$\theta_i(z) = P(t_{i-1} < T \leqslant t_i \mid T > t_{i-1}) , \tag{1}$$

where T is the time to failure random variable. Note the dependence of θ_i on z. It will be assumed that for a given z, $\theta_i(z)$ does not vary with t over the partition I_i so it is easy to see that

$$\theta_i(z) = 1 - S(t_i:z)/S(t_{i-1}:z) . \tag{2}$$

This expression takes different forms depending on the representation given for the hazard function. Note that

$$S(t:z) = \exp \left\{ - \int_0^t h(u:z)du \right\} . \tag{3}$$

The Models

We will now develop a collection of models based on the two specialized versions of the hazard function below:

$$h(t:z) = h_o(t)\exp(\beta' z) \tag{4}$$

and

$$h(t:z) = h_o(t) + \beta' z \tag{5}$$

where β is a $p \times 1$ vector of unknown parameters and $h_o(t)$ is an unknown hazard function for the standard set of conditions $z = 0$. Making use of (1) - (3), these terms can be rewritten in the form of a linear regression model. For (4) we get

$$\ln[-\ln\{1-\theta_i(z)\}] = \ln[-\ln\{1-\theta_i(0)\}] + \beta' z \tag{6}$$

and for (5)

$$-\ln\{1-\theta_i(z)\} = -\ln\{1-\theta_i(0)\} + \beta' z(t_i - t_{i-1}). \tag{7}$$

If the interval partitions are taken to be of unit length; that is, $t_i - t_{i-1} = 1$ ($i = 1, \ldots, m$), then the second term on the right hand side of (7) reduces to that of (6).

The above formulations can be generalized to incorporate time trends as follows:

$$\ln[-\ln\{1-\theta_i(z)\}] = \alpha_i + \sum_{k=0}^{r} (t_i^k)\beta' z \tag{8}$$

and

$$-\ln\{1-\theta_i(z)\} = \alpha_i + \sum_{k=0}^{r} (t_i^k)\beta' z. \tag{9}$$

Expressions (8) and (9) embody an asymmetric transformation of the conditional probability or interval hazard function $\theta_i(z)$. In order to construct a family of such transformations on which an additive representation of the hazard function can be assessed for consistency with the data, a link function will be introduced:

$$V_\lambda(\theta) = \frac{\{-\ln(1-\theta)\}^\lambda - 1}{\lambda}, \quad 0 \leqslant \lambda \leqslant 1. \tag{10}$$

When $\lambda = 0$, this expression is equivalent to the log-log transformation given by (8); when $\lambda = 1$, it reduces to the negative complementary log transformation given by (9). Thus, the link function may be used to define a comprehensive family of additive models.

We now propose to fit the model

$$V_\lambda\{\theta_i(z)\} = \alpha_i + \sum_{k=0}^{r} (t_i^k)\beta' z, \tag{11}$$

which may be easily inverted to give

$$\theta_i(z) = \begin{cases} 1 - \exp\{-(1+\lambda U_i)^{1/\lambda}\}, & \lambda U_i > -1 \text{ and } \lambda \neq 0 \\ 0, & \text{otherwise.} \end{cases} \tag{12}$$

where U_i is equal to the right-hand side of (11).

Analytic Procedure

Assuming that the failure pattern of the subjects has a binomial distribution, the theory of generalized linear models can be invoked to estimate the coefficients of (11) as λ is parametrically varied between zero and one (see Nedler and Wedderburn [15]). Note that a least squares fit will yield maximum

likelihood estimates for α and β; if an estimate of λ is desired, a sequential procedure would have to be used due to the nonlinearity of the link function. Once a fit is obtained, the expected number of individuals failing during I_i can be found as follows:

$$\hat{\mu}_i = n_i\theta_i(z), \quad i = 1, \ldots, m$$

where n_i is the total number at risk during I_i.

For either grouped or individual data, the proposed methodology is summarized below:

1. partition the time axis into intervals $[t_{i-1}, t_i)$;
2. form a frequency table of observed failures and numbers at risk;
3. form the appropriate partial likelihood (see (13) below); and
4. fit the generalized linear model defined by the partial likelihood and the link function (10) for various values of λ.

It should be pointed out that the above steps can be further generalized in a straightforward manner for time dependent explanatory variables if it is assumed that they are constant over each interval. The question still remains, though, as to the choice of interval widths, should it be a prerogative of the analyst. In such cases, an "optimal" spacing may be obtained for each set of data by making a tradeoff between the loss of information and the over-parameterization of the model.

AN APPLICATION BASED ON THE TWO SAMPLE-CASE

For simplicity the two-sample case will be used to illustrate the methodology. Suppose that there is just one z variable, $p = 1$, which takes the values 0 or 1 for either of two groups. Further, suppose that initially there are $n = n_1 + n_2$ individuals at risk, n_j in Group j ($j = 1,2$). During the i^{th} period an individual may fail, be censored or survive into the next period. For the j^{th} group, denote the observed number at risk in the i^{th} interval by n_{ij}, the observed number of individuals failing by f_{ij}, and the corresponding number of censored individuals by c_{ij}. This gives $n_{(i+1)j} = n_{ij} - f_{ij} - c_{ij}$ $(1 < i + 1 \leqslant m; j = 1, 2)$ and $m_{ij} = n_j$. It will be assumed that all censoring takes place at the end of the interval, although other possibilities can be taken into account with little alteration of the basic approach. A final assumption will be that censoring and failure are generated by independent mechanisms.

Let the random variable C_{ij} represent the number of individuals censored in the j^{th} group just prior to the $(i - 1)^{th}$ interval ($C_{1j} = 0$), and let D_{ij} represent the number of failures in the i^{th} interval for Group j. Denote by θ_{ij} the probability that an individual at risk in the j^{th} group fails during the i^{th} interval, having survived until the beginning of that interval. Then if the failures are independent, Cox has defined the partial likelihood for the sequences $\{C_{i1}, D_{i1}\}$ and $\{C_{i2}, D_{i2}\}$ to be [16]:

$$L = \prod_{i=1}^{m} \prod_{j=1}^{2} \binom{n_{ij}}{f_{ij}} (\theta_{ij})^{f_{ij}} (1 - \theta)^{n_{ij} - f_{ij}} \tag{13}$$

Note that the full likelihood can be obtained from (13) if data are available on the individual subjects. Now, except for a constant, the log likelihood may be written as

$$\ell = \sum_{i=1}^{m} \sum_{j=1}^{2} \left\{ f_{ij} \ln\theta_{ij} + (n_{ij} - f_{ij}) \ln(1 - \theta_{ij}) \right\}, \tag{14}$$

where

$$\theta_{ij} = V_{\lambda}^{-1}(U_{ij}), \qquad U_{ij} = \alpha_i + \sum_{k=0}^{r} (t_i^k)\beta_k z,$$

and

$$z = \begin{cases} 0, & j = 1 \\ 1, & j = 2 \end{cases}.$$

The objective is to represent the probabilities $\theta_{11}, \theta_{12}, \ldots, \theta_{m1}, \theta_{m2}$ in terms of the regression parameters α and β which, in part, take into account the difference between groups. For various values of λ, this will be achieved in the usual manner by maximizing (14) with respect to α and β (see Nedler and Wedderburn [15] for the details). Recall that by choosing equally spaced time points, we can compare the fit of the proportional hazard model to others in the family.

In this formulation, the customary tests for model adequacy and parameter significance are based on the "deviance" (see Elandt-Johnson and Johnson [17]):

$$\text{deviance} = 2\log(L_c/L_f),$$

where L_c is the likelihood of the model to be tested and L_f is the likelihood of the fully parameterized model (i.e., the model that would in theory give perfect predictions). The mean square error (MSE) computed from the difference between the observed and estimated number of failures will also be presented. Note that a more powerful test for $\lambda = 0$ is available based on the efficient score statistic given by $\partial\ell/\partial\lambda$, but will not be taken up here (see [16]).

AN EXAMPLE

Asbestos is a mineral found in many rock formations, which, when separated, becomes a soft, fibrious material of great utility. There are more than 3000 asbestos based products manufactured today, with the construction, automotive, and textile industries being the major users. If air containing asbestos dust is inhaled, the small, sharp fibers may work their way into the lung tissue and remain

Table 2. Grouped Failure Data for Asbestos Exposure

	Group 1		Group 2	
Years of Exposure	f^a	n^b	f	n
11	0	221	1	320
12	1	218	1	314
13	0	213	2	305
14	2	206	4	298
15	2	201	5	288
16	2	194	4	271
17	1	191	6	259
18	3	183	8	242
19	2	174	11	217
20	2	165	13	198

[a] f = observed failures.
[b] n = number at risk.

embedded for life. This can lead to asbestosis, a lung disease characterized by a scarring and thickening of the lung wall which makes breathing difficult and places a severe strain on the heart. More serious, though, asbestos has been implicated in cancer of the respiratory system, and mesothelioma, a rare form of cancer of the chest or abdominal lining which is usually fatal within one year. In all cases, the danger varies with the intensity of the dust, the size of the fiber, and the susceptibility of the individual.

Table 2 presents two sets of aggregate survival data taken from an EPA study on the health problems of asbestos workers [18]. A failure will be defined as being afflicted with one of the above three diseases. Group 1 represents those persons who were employed in plants or facilities where the material was being processed but did not have direct contact with its use. Exposure levels were relatively low. Group 2 consists of those persons who worked directly with the material and thus experienced high exposure.

Because no failures were observed prior to year ten, the data are recorded from year eleven onward. From the table it can be seen that after eleven years, of the 320 persons at risk in Group 2, one failed and five were censored, leaving 314 at risk in the twelfth year. In an approximate sense, it seems that Group 1 failures grow linearly, while those of Group 2 grow quadratically.

Using the above data, a number of different models were fitted for values of λ between zero and one. R^2 values ranged from a low of 0.84 to a high of approximately 1.0, with the corresponding F-ratios being significant at the .01 level throughout. Highlights of the results are displayed in Table 3. It appears

Table 3. Results for Various Models

Model	λ	*Parameters*	*Deviance*	*MSE*	*Degrees of Freedom*
1	0	α_i,	4.48	2.51	10
2	0	α_i, β_0	12.36	5.82	9
3	0	$\alpha_i, \beta_0, \beta_1$	6.75	3.07	8
4	0	$\alpha_i, \beta_0, \beta_1, \beta_2$	10.93	5.42	7
5	0.2	α_i, β_0	1.92	1.44	9
6	0.2	$\alpha_i, \beta_0, \beta_1, \beta_2$	1.57	1.12	7
5	0.5	α_i, β_0	1.88	1.41	9
8	0.5	$\alpha_i, \beta_0, \beta_1, \beta_2$	1.16	0.55	7
9	0.8	α_i, β_0	2.03	1.57	9
10	0.8	$\alpha_i, \beta_0, \beta_1, \beta_2$	1.08	0.48	7
11	1	α_i	4.37	2.37	10
12	1	α_i, β_0	2.61	1.72	9
13	1	$\alpha_i, \beta_0, \beta_1$	1.24	0.91	8
14	1	$\alpha_i, \beta_0, \beta_1, \beta_2$	1.14	0.52	7

Table 4. Comparison of Observed Failures with Model Estimates

	Group 1					*Group 2*				
Years of		*Fitted*					*Fitted*			
Exposure	*Observed*	*M4*	*M8*	*M11*	*M13*	*Observed*	*M4*	*M8*	*M11*	*M13*
11	0	0.0	0.0	0.4	1.2	1	0.4	0.9	0.5	0.0
12	1	0.1	0.4	0.9	1.1	1	24.1	1.9	1.2	0.8
13	0	0.0	0.2	0.7	0.4	2	0.1	0.9	1.0	1.4
14	2	0.7	1.9	2.4	1.6	4	12.2	4.3	3.5	4.6
15	2	1.3	2.0	2.7	1.5	5	7.9	4.9	3.9	5.8
16	2	1.6	1.6	2.4	0.7	4	5.0	4.7	3.4	5.8
17	1	1.6	1.3	2.7	0.5	6	3.7	5.2	3.7	6.7
18	3	3.1	2.4	4.5	2.0	8	7.8	9.2	3.4	9.3
19	2	2.3	2.2	5.4	2.6	11	9.4	10.6	6.8	10.3
20	2	1.6	2.1	6.5	3.4	13	15.9	12.8	7.9	11.4

that the proportional hazard model, originally stated in (4) and realized when $\lambda = 0$ for the link function, is inferior to almost every formulation without regard to the number of β parameters the latter contains. This is supported by an examination of both the deviance and mean square errors, where values less than 1.5 and 1.0, respectively, are considered good. In addition, all models that included only the α_i terms proved to be relatively inadequate. To illustrate the point, consider the additive model $\lambda = 1$ with a linear time trend [Model 13]. The accompanying estimates of β_0 and β_1 are -0.0131 (SE = 0.0071) and 0.0052 (SE = 0.0011) which are highly significant. The point is corroborated by examining the performance of Model 11 which only contains the α_i parameters. (In all cases the α_i's turned out to be significant.) By contrast, the estimates for β_0, β_1, and β_2 of Model 4 are 11.027 (SE = 4.471), -2.925 (SE = 1.867), and 0.204 (SE = 0.165), only the first of which is significant.

Table 4 compares observed failures for the two groups with the estimates obtained from Models 4, 8, 11, and 13. The inclusion of a time trend makes a substantial difference in the performance of each. Model 4 performs adequately for Group 1, but is highly erratic for Group 2, showing oscillatory behavior in the early stages and then switching to geometric growth. Model 11, containing no β parameters, attempts a compromise between the two groups and consequently overestimates the first and underestimates the second. The remaining two models, 8 and 13, do a superior job in fitting the data yielding mean square errors of 0.48 and 0.91, respectively. In fact, all models with at least a linear time trend and a value of λ greater than or equal to 0.5 do quite well at prediction as measured by both the deviance and MSE.

CONCLUSIONS

The need for a better understanding of the relationships between exposure and mortality is essential to the continuing public debate on regulation of hazardous substances. This article has attempted to develop a collection of models that can be used in this effort, which are at once flexible and effective, and yet completely empirical. Using maximum likelihood estimation, we demonstrated that highly accurate fits can be obtained for certain parametric forms. Although this was done for the two-sample case only, had the data been further disaggregated by, say age, smoking habits, or job classification, it would have been possible to include these additional explanatory variables in the analysis, perhaps giving a more powerful result.

One criticism that can be leveled at the approach stems from an interpretation of the models when $0 < \lambda < 1$. In this interval, the hazard does not have a clear meaning. Nevertheless, this point may be outweighed by performance. Finally, the method may prove effective in situations where accelerated life testing is important and only a limited amount of grouped data is available. The ability to

include polynomial time trends and to easily estimate the underlying reliability function underscores this point.

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