

On Certain Problems Involving Non-identifiability
of Distributions Arising in Stochastic Modeling^{*,**}

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ABSTRACT. Some examples from recent literature involving nonidentifiability of distributions arising in stochastic modeling for live situations, are briefly surveyed. In particular, examples are drawn from the areas of competing risks, Markov illness-death models, reliability theory and accident proneness. For the reliability models, the discussion here is restricted only to shock-type models, although similar nonidentifiability problems arise in more general models involving damage, wear or fatigue besides shocks. Finally the author emphasizes that in stochastic modeling, the nonidentifiability problem is typically much more acute than is usually thought of or looked into or even reported; that it should be investigated first, before the model is put to any practical use, and that in the presence of nonidentifiability, one needs to search for additional conveniently observable variables, which could be used to reduce or if possible to eliminate the existing nonidentifiability.

Key Words: Stochastic Models, Non-identifiability of Distributions, Competing Risks, Markov Illness-Death Models, Reliability Theory, Accident Proneness, Threshold Versus Nonthreshold Hypothesis.

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1 INTRODUCTION. In order to study a natural random phenomenon arising in some live situation, often an attempt is made to idealise the underlying stochastic mechanism, while still trying to keep it close enough to reality and then construct a stochastic model based on this mechanism. As a result, one usually ends up with a family of distributions of the observable random variable (r.v.) X , generated through a set of unknown parameters. Let this family of probability measures corresponding to X , as given by the stochastic model, be represented by

$$\mathfrak{F} = \{P_{\theta}, \theta \in \Theta\}, \quad (1)$$

where θ is a labeling parameter taking values in an appropriate set Θ . Here X could be a random vector or the realization of a stochastic process over a fixed time-interval, etc., as the case may be. Similarly the possible values of the parameter θ could be over a class Θ of vector-valued functions (see sections 3 and 5), or over a subset of k -dimensional Euclidean space, etc.

A natural question, although not that often raised in practice, is whether the members of \mathfrak{F} are distinct for distinct θ 's. If they are, then based on the observable r.v. X , we say that the family \mathfrak{F} of distributions is Identifiable. Otherwise we say that it is Nonidentifiable (see sections 2, 3, 4.1 and 4.2, for examples). The question of nonidentifiability must in general be settled before one attempts to estimate the parameter θ by suitable statistical methods and uses the model and the estimate of θ to draw practical inferences.

Again, it is not uncommon to try to explain the same observable r.v. X , through more than one stochastic model, each based on its own set of underlying assumptions and on possibly different mechanisms. Each then will generate its own family of probability distributions for the observable r.v. X in question. Let

$$\mathfrak{F}_i = \{P_{\theta_i}, \theta_i \in \Theta_i\}, \quad i=1,2 \quad (2)$$

be two such families corresponding to two different stochastic models.

DEFINITIONS. The family \mathfrak{F}_1 is said to be nonidentifiable with respect to the family \mathfrak{F}_2 , if for each $\theta_1 \in \Theta_1$ there exists $\theta_2 \in \Theta_2$ such that $P_{\theta_1} = P_{\theta_2}$, so that $\mathfrak{F}_1 \subset \mathfrak{F}_2$. If however $\mathfrak{F}_1 = \mathfrak{F}_2$, we say that \mathfrak{F}_1 and \mathfrak{F}_2 are completely mutually nonidentifiable. Again, \mathfrak{F}_1 and \mathfrak{F}_2 are said to be partially mutually nonidentifiable, if $\mathfrak{F}_1 \cap \mathfrak{F}_2$ is nonempty and is a proper subset of each \mathfrak{F}_i , $i=1,2$. Finally, if $\mathfrak{F}_1 \cap \mathfrak{F}_2$ is empty, the families \mathfrak{F}_1 and \mathfrak{F}_2 are said to be mutually identifiable. (For examples, see sections 4.3 and 5).

If a family \mathfrak{F} itself is nonidentifiable then typically in practice one attempts to find some additional suitable observable r.v. Y to go with X and enhances the original family \mathfrak{F} to cover the joint probability distributions of (X,Y) , so that the enhanced family with the added information may become identifiable. If it is possible to find such a Y , we shall call the original nonidentifiable family \mathfrak{F} of distributions of X as 'rectifiable' (see for example, section 4.3). Again similar added information may make the previously mutually nonidentifiable families \mathfrak{F}_1 and \mathfrak{F}_2 , now mutually identifiable. One early example of this type occurs in the well known work of Bates and Neyman [3] on the theory of accident proneness. There exist however situations, where no such Y exists and as such it is impossible to make any progress in reducing the nonidentifiability (see section 5, for such an example).

The purpose of this presentation is to briefly survey some examples recently arisen in literature, which involve nonidentifiability problems in stochastic modeling for live situations. In these examples, our primary concern will be of nonidentifiability and not about the reasonableness of a model for the intended live situation.

2. PROBLEM OF COMPETING RISKS. Our first example arises in the area popularly known as 'Problem of Competing Risks', associated with simple survival experiments, as opposed to the ones considered in the next section. Here in a biological context, a sample of N newborn animals, belonging to a 'conceptual' population of animals, are observed for the duration of their lives. For each animal, the age L at death and the cause C of death are recorded. With these data at hand, what one can estimate at best are the probabilities $P(L < t, C = d)$, with $d \in D$, where D is the preconceived finite (exhaustive) list of distinct, well defined and mutually exclusive causes of death. For this, one repeatedly studied model in literature is based on the so called Potential Survival Time (see Berman [5], David [10], David and Moeschberger [11], Elandt-Johnson [12], Gail [15], Hoel [16] and Hoel and Walburg [17]). Here it is postulated that for each cause of death $d \in D$, there exists an associated nonnegative r.v. Z_d , called the potential survival time. The distribution of the age L at death of a typical animal is then assumed to be the same as that of $\min_{d \in D} Z_d$ with cause of death given by $C = \{k: Z_k = \min_{d \in D} Z_d\}$. Also it is typically assumed that the joint distribution of Z 's is continuous, so that with probability one there are no ties among Z 's and the cause C is uniquely defined. If F_Z denotes such a joint distribution function of the potential lifetimes $\{Z_d, d \in D\}$, then for each such F , one can easily obtain the distribution P of the observable r.v. $X = (L, C)$. Let us denote by \mathfrak{F} , the family of distributions of X , as generated by varying F , which correspond to our parameter θ in (1).

Until recently, it was commonly assumed that the potential survival times Z_d 's are independently distributed with continuous distribution functions F_d , $d \in D$. (Even now one continues encountering papers in literature based on such an assumption). Let \mathfrak{F}_I denote the subfamily of distributions of X , which are computed subject to this independence assumption, so that $\mathfrak{F}_I \subset \mathfrak{F}$. Based on the r.v. X , it is then possible to show that this smaller family \mathfrak{F}_I of distributions is identifiable. However, as was brought out in a recent workshop on

Evaluation of Environmental Biological Hazards and Competing Risks, organized at Oak Ridge, Tennessee, since around 1972, about half a dozen authors, some independently, some not, have discovered that corresponding to each element in $\mathfrak{F}-\mathfrak{F}_I$, there always exists one in \mathfrak{F}_I , such that the corresponding distributions of $X=(L,C)$ are identical (see David [10], Gail [15], Miller [20], Peterson [23], Rose [31], Tsiatis [32]). The original family \mathfrak{F} is therefore nonidentifiable. Thus, generally speaking, any prediction based on these models, to author's mind, are purely of academic interest, unless of course in a highly rare and exceptional situation, one has some definite prior knowledge about the form of $F_{\mathcal{K}}$, which forces his model to correspond to an identifiable subset of \mathfrak{F} . A similar caution is required when it comes to using these models in reliability theory contexts (see Proschan and Serfling [24]).

3. TIME NONHOMOGENEOUS MARKOV ILLNESS-DEATH MODELS.

In connection with what was discussed in the last section, it may be mentioned that the cause of death strictly speaking is only a fiction. What is more appropriate instead is to talk of combinations of various possible illnesses or 'morbid conditions' present at the time of death. The particular combination of conditions (illnesses) present at death can be established through proper autopsies. One of the early models for this situation advanced by Neyman [22] and then applied by Fix and Neyman [14], is based on a time homogeneous Markov illness-death model. The reader may find a generalization of these models to the case of time-nonhomogeneous Markov processes in Chiang [7]. The parameter θ of (1) for these models is typically represented by a vector of appropriate nonnegative functions $v_{ij}(\cdot), i \neq j$, and $\mu_i(\cdot), i, j=1, 2, \dots, s$, where s is the number of possible states (conditions' combinations) allowed; $v_{ij}(\cdot)$ is the usual nonnegative intensity function for a transition from state i to j for the assumed Markov process and $\mu_i(\cdot)$ is the nonnegative intensity function for occurrence of death from state i . The usual data based on a follow-up of

several experimental animals correspond to the observable random variable $X=(L,S)$. Here L is the time of death of an animal and S represents the state it was in at death. Unfortunately the corresponding family \mathfrak{F} of distributions of X with θ as specified earlier by $(\nu(\cdot), \mu(\cdot))$, is highly nonidentifiable. From a paper written by a group of biologists and published in 1969 (see Upton, et al [33]), it appears that, at least in principle, they were aware of the difficulties connected with the nonidentifiability of the family \mathfrak{F} associated with the present model. In order to cope with these, they introduced a novel experiment, where apart from naturally dieing animals, they also serially sacrificed a number of animals over a certain period of time to find out the states (conditions) they were in, at the time of sacrifice. This leads to an additional observable r.v. $Y=(S(t_\ell), \ell=1,2,\dots)$, where $S(t_\ell)$ is the state a live animal is in, at the time t_ℓ of its sacrifice. In the language of section 1, the question arises whether or not the enhanced family $\tilde{\mathfrak{F}}$ of distributions of (X,Y) is now identifiable. As has been recently shown by Clifford [9] (see also Berlin, Brodsky and Clifford [4]), the answer to this question unfortunately is still negative. In [9], Clifford considers a time-nonhomogeneous Markov illness-death model of the progressive type, where the transition intensity functions $\nu_{ij}(\cdot), i \neq j$, are possibly positive only for those pairs (i,j) , for which the state j contains exactly one more 'morbid condition' (or disease) than the state i did; all other $\nu_{ij}(\cdot)$'s are identically zero. For such a model, based on the random vector (X,Y) defined above, Clifford proves in his theorem 1 that while it is possible to identify the time-dependent death intensities $\mu_i(\cdot), i=1,2,\dots,s$, the various time-dependent intensities $\nu_{ij}(\cdot)$ of transitions between states are not identifiable. A close scrutiny of his proof reveals that he assumes the possibility of estimating the probabilities $P_i(t) = P$ (an animal is alive and is in state i at time t),

for all t-values over a certain time-interval, using the data on Y-values.

This is unfortunately impossible in practice, since the animals can be sacrificed only at a finite number of time-points. Thus to the author it appears that, based on the usual data on (X,Y) , even the death intensity functions $\mu_i(\cdot)$, $i=1,2,\dots,s$, are not quite identifiable, so that the problem of nonidentifiability appears to be much more severe than projected in [9]. However as indicated in [9], it is encouraging to note that the subfamily $\tilde{\mathcal{F}}_1$, obtained by assuming that the various intensities ν_{ij} 's and μ_i 's are all constant (so that the underlying Markov process is time-homogeneous), is identifiable provided the various sacrifices are made on a number of different days for observing Y.

4. CERTAIN STOCHASTIC MODELS IN RELIABILITY THEORY.

In Section 4.1, we discuss briefly an extensively studied stochastic model arising in reliability theory, based on a (random) threshold hypothesis, while in Section 4.2 we discuss its analog based on a nonthreshold hypothesis, which to us appears more appropriate. In Section 4.3, these two models are compared from the mutual identifiability point of view.

4.1. A THRESHOLD-TYPE SHOCK MODEL. Consider a system which involves only a single component. It is commonly assumed that the system receives in some random manner over time, the so called 'shocks' or 'blows'. Associated with each such system, the existence of a random threshold K for the total number of shocks is then postulated, so that as soon as the threshold K is reached the system fails. More specifically, following the treatment in a key paper by Esary, Marshall and Proschan [13] (see also Barlow and Proschan [1]), we assume that the arrivals of shocks is governed according to a time-homogeneous Poisson process with parameter λ , so that the distribution of the length of life L of the system ^{is} given by

$$\bar{H}_1(t) = P(L > t) = \sum_{k=0}^{\infty} \frac{(\lambda t)^k}{k!} e^{-\lambda t} \bar{P}_k, \quad t > 0, \quad (3)$$

where

$$\bar{P}_k = P(K > k), \quad k=0,1,2,\dots, \quad \text{with } \bar{P}_0 = 1. \quad (4)$$

Such a model will be called a 'Threshold-type' model. Typically the occurrences of shocks are not observable, so that the only observable r.v. is L . Let \mathfrak{F}_1^* denote the family of distributions of L , generated by varying λ and $\{\bar{P}_k\}$. Unfortunately, as also noted by Clifford [8] and by Esary, Marshall and Proschan [13], this family is nonidentifiable. This follows from the fact that for every $\nu > \lambda$, we can write $\bar{H}_1(t)$ of (3) in the form

$$\bar{H}_1(t) = \sum_{k=0}^{\infty} \frac{(\nu t)^k}{k!} e^{-\nu t} \bar{Q}_k, \quad t > 0, \quad (5)$$

where corresponding to (4) we now have

$$\bar{Q}_k = \nu^{-k} \sum_{j=0}^k \binom{k}{j} (\nu - \lambda)^{k-j} \lambda^j \bar{P}_k, \quad k=0,1,2,\dots, \quad (6)$$

with $\bar{Q}_0 = 1$. Again, if on the other hand λ were known, then the family \mathfrak{F}_1^* generated by only varying $\{\bar{P}_k\}$, can be easily seen to be identifiable.

4.2. A NON-THRESHOLD-TYPE SHOCK MODEL. Under a nonthreshold type shock model, the assumption about the existence of a threshold is abandoned and instead, perhaps more realistically, one assumes the existence of a nonnegative risk function $\beta(N(t), t)$ depending on time t and the number $N(t)$ of shocks received until time t , such that

$$\begin{aligned} P(\text{failure occurs in } (t, t+\tau) \mid \text{No failure occurred until } t, \text{ and } N(t) = n) \\ = \beta(n, t)\tau + o(\tau). \end{aligned} \quad (7)$$

The reader may refer to Puri ([25], [26], [27] and Puri and Senturia [30], for general discussions about threshold versus nonthreshold type models arising in several live situations in biology. In the present context of reliability theory, the reader may refer for nonthreshold type models to Mercer [19] and a recent paper of the author [28].

Considering a simpler case for the present, we assume that $\beta(n,t) = n\alpha(t)$, where $\alpha(\cdot)$ (i) is a nonnegative function, (ii) integrable over time intervals (a,b) , for all $0 < a < b < \infty$, and (iii) is such that it satisfies

$$\int_0^{\infty} (1 - \exp\{-\int_{\tau}^{\infty} \alpha(u) du\}) d\tau = \infty. \quad (8)$$

Thus for a nonthreshold type model, we have the distribution for the length of life L of the system, given by

$$\bar{H}_2(t) = P(L > t) = E\{\exp[-\int_0^t N(\tau)\alpha(\tau)d\tau]\}. \quad (9)$$

Using the fact that $N(t)$ is a Poisson process with parameter λ , this can be easily shown to be equal to

$$\bar{H}_2(t) = \exp\{-\lambda \int_0^t (1 - \exp[-\int_{\tau}^{\infty} \alpha(u) du]) d\tau\}, \quad (10)$$

so that in view of (8), L is an honest random variable. Again, let \mathfrak{F}_2^* denote the family of distributions of L , generated through (10) by varying λ and the function $\alpha(\cdot)$ subject to the above conditions (i)-(iii). Based on the data only on the length of life L , this family too is unfortunately nonidentifiable, since for a given $\theta_1 = (\lambda_1, \alpha_1(\cdot))$, for every $\lambda_2 > \lambda_1$, there exists a unique function $\alpha_2(\cdot)$ satisfying (i)-(iii), such that the expression (10) corresponding to $\theta_1 = (\lambda_1, \alpha_1(\cdot))$ and $\theta_2 = (\lambda_2, \alpha_2(\cdot))$ becomes identical. The corresponding function $\alpha_2(\cdot)$ is given by

$$\alpha_2(t) = \lambda_1 \alpha_1(t) h_1(t) [(\lambda_2 - \lambda_1)t + \lambda_1 h_1(t)]^{-1} \quad (11)$$

where

$$h_1(t) = \int_0^t \exp[-\int_{\tau}^t \alpha_1(u) du] d\tau. \quad (12)$$

Finally, as before if on the other hand λ were known, the family \mathfrak{F}_2^* , as generated by only varying $\alpha(\cdot)$, becomes identifiable.

4.3. ON MUTUAL NONIDENTIFIABILITY BETWEEN \mathfrak{F}_1^* AND \mathfrak{F}_2^* . In this section, we study briefly the two families \mathfrak{F}_1^* and \mathfrak{F}_2^* , as introduced in Sections 4.1 and 4.2, from the point of view of their mutual nonidentifiability. The reader may find a similar comparison in Puri and Senturia [30] between a threshold versus a nonthreshold type model arising in quantal response assays in biology. In the present case, we shall compare \mathfrak{F}_1^* and \mathfrak{F}_2^* under the assumption that the Poisson parameter λ corresponding to the occurrences of shocks is same in the two cases. Thus the only difference is that whereas \mathfrak{F}_1^* corresponds to a threshold type model characterized by the distribution \bar{P}_k , the family \mathfrak{F}_2^* bears on a nonthreshold approach characterized by the risk function $\alpha(\cdot)$ satisfying conditions (i)-(iii). While the details as well as other general results can be found in Puri [28], we briefly mention here only a few results:

Based only on the observable r.v. L , for each member of \mathfrak{F}_1^* corresponding to a given $\{\bar{P}_k\}$, there always exists one in \mathfrak{F}_2^* with a risk function $\alpha(\cdot)$, such that the corresponding distributions of L match, so that $\mathfrak{F}_1^* \subset \mathfrak{F}_2^*$. Also \mathfrak{F}_1^* is a proper subset of \mathfrak{F}_2^* , since there do exist some respectable members of \mathfrak{F}_2^* , for which there do not exist the corresponding matching members of \mathfrak{F}_1^* . Thus in the language of Section 1, \mathfrak{F}_1^* is nonidentifiable with respect to \mathfrak{F}_2^* . Also the family \mathfrak{F}_2^* (nonthreshold type model) is much more richer than the family \mathfrak{F}_1^* (threshold type model). Again, if at all it is possible to identify the so called 'shocks' in practice, one can reduce the level of nonidentifiability by observing the total number N_L of shocks received by the time L of failure, and by enhancing the families \mathfrak{F}_1^* and \mathfrak{F}_2^* to cover the joint probability distributions of (L, N_L)

under the two models. As it turns out, the joint distributions of (L, N_L) coincide for the two models if and only if the distribution of the threshold K under \mathfrak{F}_1^* is geometric, given by

$$P(K=k) = c(1+c)^{-k}, \quad k=1,2,\dots, \quad (13)$$

for some $c>0$ and the corresponding risk function $\alpha(\cdot)$ under \mathfrak{F}_2^* is given by $\alpha(t) = c/t$, for $t>0$, and with the same constant c as taken in (13). Thus the observability of the additional random quantity N_L does succeed in reducing the nonidentifiability to a considerable extent. On the other hand, in order to completely remove the nonidentifiability between the two families (or models) one needs some further observable information such as the number of shocks $N(t)$ by some time t , prior to the failure of the system. This is analogous to the serial sacrifice experiment mentioned in Section 3, where a live animal is sacrificed to observe the state it is in at a time prior to its death.

5. TWO CONCEPTUALLY DIFFERENT MODELS ON ACCIDENT PRONE-NESS, THEIR COMPLETE NON-IDENTIFIABILITY AND NON-RECTIFIABILITY.

Our final example in modeling which goes back to the work of Bates and Neyman in 1952, on their theory of accident proneness, is given below. The reader may find in Bates and Neyman [2], [3], details for the practical appropriateness of these models.

Nonfatal accidents are assumed to occur to an individual over time according to a Poisson process with constant parameter λ . However, it is postulated that λ varies randomly over the population of individuals according to some distribution function $F(\cdot)$ concentrated over $(0, \infty)$, so that the distribution of the number of accidents $N(t)$ occurring during time interval $(0, t)$ is given by

$$P(N(t) = k) = \int_0^{\infty} \frac{(\lambda t)^k}{k!} e^{-\lambda t} dF(\lambda); \quad k=0,1,2,\dots \quad (14)$$

Let $\hat{\mathfrak{F}}_1$ denote the family of processes $\{N(t), t \geq 0\}$, as defined above, and as generated by varying the distribution function F under the above simple model. Here $N(t)$ is essentially a mixture of Poisson processes with mixing distribution given by F (see also Puri and Goldie [29]). Also, in [3], Bates and Neyman consider only a special case, where the mixing distribution F is taken to be a Gamma distribution given by

$$\frac{dF(x)}{dx} = \frac{\beta^\alpha}{\Gamma(\alpha)} x^{\alpha-1} \exp(-\beta x), \quad x > 0, \quad (15)$$

for $\alpha, \beta > 0$. Notice the distinct conceptual difference of this model with an alternative one, where $N(t)$ is instead assumed to be a time-nonhomogeneous Markov process with some nonnegative intensity functions $\rho_n(\cdot)$, $n=0,1,2,\dots$, for occurrences of accidents, such that

$$P(\text{an accident occurs in } (t, t+\tau) | N(t)=n) = \rho_n(t)\tau + o(\tau), \quad (16)$$

and

$$P(\text{more than one accident occurs in } (t, t+\tau) | N(t)=n) = o(\tau). \quad (17)$$

It is assumed that the functions $\rho_n(\cdot)$ satisfy only the conditions (i) and (ii) of Section 4.2. Let $\hat{\mathfrak{F}}_2$ denote the family of time-nonhomogeneous Markov processes $N(t)$ so defined and as generated by varying the sequence of intensity functions $\{\rho_n(\cdot), n=0,1,2,\dots\}$, which correspond to θ of (1), in the present case. In [3], Bates and Neyman consider another model labeled as 'Polya Model', which is a special case of $\hat{\mathfrak{F}}_2$, with the intensity functions given by

$$\rho_n(t) = \delta(\alpha+n)(\beta+t)^{-1}, \quad n=0,1,2,\dots; \quad (18)$$

where δ, α and β are all arbitrary nonnegative constants. Our aim here is to compare the families $\hat{\mathfrak{F}}_1$ and $\hat{\mathfrak{F}}_2$ with respect to their mutual identifiability properties.

It is known (see McFadden [18]) that each element of $\hat{\mathfrak{F}}_1$ corresponding to an F , is itself a Markov process. However, recently Cane [6] has proved a rather interesting result; namely that for each model in $\hat{\mathfrak{F}}_1$, there exists a corresponding element of $\hat{\mathfrak{F}}_2$ with such a sequence $\{\rho_n(\cdot)\}$ of intensity functions that not only the distributions of $N(t)$ coincide for every t in the two cases, but also so do their conditional joint distributions of times $0 < \tau_1 < \tau_2 < \dots < \tau_n < t$ of occurrences of the accidents, given that $N(t)=n$, for all $n \geq 1$ and $t > 0$. Thus here the corresponding two processes are mutually indistinguishable. More specifically, for a given element of $\hat{\mathfrak{F}}_1$ with a mixing distribution $F(\cdot)$, the corresponding element of $\hat{\mathfrak{F}}_2$ with the above property has the sequence $\{\rho_n(\cdot)\}$ given by

$$\rho_n(t) = \left(\int_0^{\infty} \lambda^{n+1} \exp(-\lambda t) dF(\lambda) \right) \left(\int_0^{\infty} \lambda^n \exp(-\lambda t) dF(\lambda) \right)^{-1}, \quad (19)$$

$n=0,1,2,\dots$. In particular, for the special cases with (15) and (18) considered by Bates and Neyman, such an indistinguishability of the two processes arises if in (18) one takes $\delta=1$ and identifies α and β of (18) with those of (15). Thus if we let $\hat{\mathfrak{F}}_2^*$ be the subfamily of $\hat{\mathfrak{F}}_2$, where for each element, the sequence $\{\rho_n(\cdot)\}$ is given by (19) for some $F(\cdot)$, then in view of the above result of Cane, the two families $\hat{\mathfrak{F}}_1$ and $\hat{\mathfrak{F}}_2^*$ are completely mutually nonidentifiable. Furthermore, since for each process in one family, there is a corresponding one in the other, which is indistinguishable from it and can be regarded as describing the experience of the other, the nonidentifiability between $\hat{\mathfrak{F}}_1$ and $\hat{\mathfrak{F}}_2^*$ is not at all rectifiable, in the sense of section 1. From the practical viewpoint, as far as predictions are concerned, this nonidentifiability does not matter, since the two models would anyway predict the same thing. Nevertheless, the two models are conceptually miles apart. Under the first one, λ , which represents the accident proneness (or risk of accident) of an individual, although picked up randomly according to the distribution function F , yet it remains constant throughout the experience of the individual. Contrary to this, under the second

model, the accident proneness of an individual varies both with time as well as with the number of accidents he had in the past. Unfortunately in the present case, it does not appear possible to compare the validity of one model against the other.

6. CONCLUDING REMARKS. From the history of above examples, it appears that in stochastic modeling, the problem of nonidentifiability is typically much more acute than is usually thought of or looked into, or even reported. On the other hand, if such a problem does exist, it should be investigated first, before the model is put to any practical use for the purposes of predictions. Otherwise, as indicated by Clifford [9] through numerical examples in his case, one may arrive at quite conflicting predictions by using them. Also, in the presence of nonidentifiability, one must look effectively for additional conveniently observable quantities, which could be used to enhance the underlying family of distributions in order to reduce or if possible, eliminate the existing nonidentifiability.

REFERENCES

- [1] Barlow, R. E. and Proschan, F. (1975) Statistical Theory of Reliability and Life Testing, Holt, Rinehart and Winston, Inc. N.Y.
- [2] Bates, G. E. and Neyman, J. (1952a) Contribution to the theory of accident proneness. I. An optimistic model of the correlation between light and severe accidents, Univ. Calif. Pub. Statist. 1, 215-254.
- [3] Bates, G. E. and Neyman, J. (1952b) Contribution to the theory of accident proneness. II. True or false contagion, Univ. Calif. Pub. Statist. 1, 255-276.
- [4] Berlin, B., Brodsky, J. and Clifford, P. (1977) Testing disease dependence in survival experiments with serial sacrifice, (unpublished).
- [5] Berman, S. M. (1963) Notes on extreme values, competing risks and semi-Markov processes, Ann. Math. Statist. 34, 1104-1106.
- [6] Cane, V. R. (1977) A class of non-identifiable stochastic models, J. Appl. Prob. 14, 475-482.
- [7] Chiang, C. L. (1968) Introduction to Stochastic Processes in Biostatistics, J. Wiley, N.Y.
- [8] Clifford, P. (1972) Nonthreshold models of the survival of bacteria after irradiation, Proc. 6th Berkeley Symp. on Math. Statist. and Prob., University of California Press, 4, 265-286.
- [9] Clifford, P. (1977) Nonidentifiability in stochastic models of illness and death, Proc. Natl. Acad. Sci. USA., 74, 1338-1340.
- [10] David, H. A. (1974) Parametric approach to the theory of competing risks, in Reliability and Biometry, Statistical Analysis of Life length, eds. Proschan, F. and Serfling, R. J., 275-290, SIAM, Philadelphia.
- [11] David, H. A. and Moeschberger, M. L. (1971) Life tests under competing causes of failure and the theory of competing risks, Biometrics, 27, 909-934.
- [12] Elandt-Johnson, R. (1976) Conditional failure time distributions under competing risk theory with dependent failure times and proportional hazard rates, Scand. Actuar. J. 1, 37-51.
- [13] Esary, J. D., Marshall, A. W. and Proschan, F. (1973) Shock models and wear processes, Ann. of Prob., 1, 627-649.
- [14] Fix, E. and Neyman, J. (1951) A simple stochastic model of recovery, relapse, death and loss of patients, Human Biology, 23, 204-241.

- [15] Gail, M. (1975) A review and critique of some models used in competing risks analysis, Biometrics, 31, 209-222.
- [16] Hoel, D. G. (1972) A representation of mortality data by competing risks, Biometrics, 28, 475-488.
- [17] Hoel, D. G. and Walburg, H. E. (1972) Statistical analysis of survival experiments, J. Nat. Cancer. Inst., 49, 361-372.
- [18] McFadden, J. A. (1965) The mixed Poisson process, Sankhyā, series A, 27, 83-92.
- [19] Mercer, A. (1961) Some simple wear-dependent renewal processes, J. Roy. Statist. Soc. Ser. B, 23, 368-76.
- [20] Miller, D. R. (1977) A note on independence of multivariate lifetimes in competing risks models, The Ann. of Statist. 5, 576-579.
- [21] Moeschberger, M. L. (1974) Life tests under dependent competing causes of failure, Technometrics, 16, 39-47.
- [22] Neyman, J. (1950) First Course in Probability and Statistics, Holt, N.Y.
- [23] Peterson, A. V. Jr. (1976) Bounds for a joint distribution function with fixed sub-distributions: Application to competing risks, Proc. Natl. Acad. Sci. USA., 73, 11-13.
- [24] Proschan, F. and Serfling, R. J. (1974) Reliability and Biometry, Statistical Analysis of Life length, SIAM, Philadelphia.
- [25] Puri, P. S. (1967) A class of stochastic models of response after infection in the absence of defense mechanism, Proc. 5th Berkeley Symp. on Math. Statist. and Prob., Univ. of California Press, 4, 511-535.
- [26] Puri, P. S. (1969) Some new results in the mathematical theory of phage-reproduction, J. Appl. Prob. 6, 493-504.
- [27] Puri, P. S. (1976) Biomedical applications of stochastic processes, On the History of Statistics and Probability, Ed: D. B. Owen, Marcel Dekker, N.Y.
- [28] Puri, P. S. (1978) Nonidentifiability among some stochastic models in reliability theory, Mimeograph Series , Dept. of Statistics, Purdue University, Indiana.
- [29] Puri, P. S. and Goldie, C. M. (1978) Poisson mixtures and quasi-infinite divisibility of distributions, To appear in J. Appl. Prob.
- [30] Puri, P. S. and Senturia, J. (1972) On the mathematical theory of quantal response assays, Proc. 6th Berkeley Symp. on Math. Statist. and Prob., Univ. of California Press, 4, 231-247.

- [31] Rose, D. M. (1975) Dependent competing risks, Biometrics, 31, Abstract #2311, p.600.
- [32] Tsiatis, A. (1975) A nonidentifiability aspect of the problem of competing risks, Proc. Natl. Acad. Sci. USA., 72, 20-22.
- [33] Upton, A. C. et al. (1969) Quantitative experimental study of low-level radiation carcinogenesis. Radiation Induced Cancer, 425-438, International Atomic Energy Agency, Vienna.