A hazards approach to the biometric analysis of infant mortality

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Abstract

A variation of the Bourgeois-Pichat biometric analysis of infant mortality is suggested. In the original model, cumulative mortality in the last eleven months of infancy is assumed to follow a uniform distribution given a log-cube transformation of age. Instead, we assume an exponential distribution. The difference is that while the denominator is constant in the Bourgeois-Pichat model, equal to the number of births, in our model, the denominator is the current population at risk. We argue that our assumption is more satisfactory from a theoretical point of view, since it focus on the conditional probability of dying. Our model gives different estimates of endogenous and exogenous mortality and, in addition, the model fit is slightly better, especially in cases with higher levels of infant mortality.

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1 Introduction

In this version the cumulative hazard functions are depicted as-is, but on the log-cube time scale. See the new \mathbf{R} function drawBB2.

[Figure 1 about here.]

Lois Bourgeois-Pichat saw the first year of life not only as the period when mortality is highest, but also the period when improvements should be possible (Bourgeois-Pichat, 1951a,b; Bourgeouis-Pichat, 1952). He argued that infant deaths should be divided into two categories, endogenous and exogenous deaths. Endogenous deaths are, by definition, due to inherited factors, or acquired during gestation or delivery. They typically occur at the initial period of life, though sometimes much later and include congenital debility, prematurity, malformations, and disease in early life. Exogenous infant mortality, which Bourgeois-Pichat regards as accidental, are deaths for which the society must hold itself responsible and to reduce endogenous mortality, medical intervention is essential (Bourgeouis-Pichat, 1952). Since it is, based on causes of deaths, difficult to distinguish between endogenous and exogenous factors, either because they are inaccurate or difficult to make due to the diseases themself, or the information do not exist, Bourgeois-Pichat offered a solution to the problem.

Bourgeois-Pichat's biometric model to differentiate between endogenous and exogenous mortality is based on an idea of a universal law governing the distribution of deaths in the first year of life (Bourgeouis-Pichat, 1952). He assumes that all deaths taking place in the last eleven months of the first year are exogenous. He argues that, although there are also some endogenous mortalities during this period, they do not affect the development of mortality much. Supported by examples from mid-twentieth century Western and Southern Europe, the US and Canada, New Zeeland, and other countries, Bourgeois-Pichat argued that the cumulative infant deaths after the first month follows a linear development given a log-cube transformation of age (Bourgeouis-Pichat, 1952). Mortality in the first month of life is then divided into an endogenous and an exogenous assuming that the same linearity also exists in this period. It leads to the conclusion that exogenous deaths in the first month of life represent 25 percent of the deaths from the second to twelfth months (Bourgeouis-Pichat, 1952).

Applying this method, Bourgeois-Pichat also finds deviations from the linear death pattern after the first month. To take one example, the curve for Sardinia in 1948 bend strongly upward after four months (Bourgeouis-Pichat, 1952, Figure 12). To take another example, in the case of Quebec 1944–1947, the curve bends down quite strongly after the sixth month (Bourgeouis-Pichat, 1952, Figure 8). A downward bend is also what has been found using historical Swedish parish data (Bengtsson, 1999, Figure 6b; Bengtsson and Lundh, 1999, Figures 6 and 7; Lynch et al., 1998, Figures 1 and 2; Sundin and Tedebrand, 1981, Figure 6d), though less pronounced than in Quebec. The curve for Sweden 1910–1946 show, however, no such downward bend (Bourgeois-Pichat, 1951b, Figure 9; Bourgeouis-Pichat, 1952).

Bourgeois-Pichat's biometric method has also been used to identify problems with data recording (Wrigley, 1977; Wrigley and Schofield, 1981). While a high level of endogenous mortality cannot be used as a criterion for high data quality, the opposite holds true. A very low level of endogenous mortality indeed indicates data problems, just like large deviations from the normal sex ratio at birth do. These two criteria are, in fact, often used in historical studies to evaluate data recording (see Bengtsson et al 2004). They become even more precise if they are applied to different social strata, since under-recording of early deaths often have a social gradient, possibly due to the costs involved in a burial (Bengtsson, 1999; Bengtsson and Lundh, 1999).

We suggest a variation of the Bourgeois-Pichat biometric analysis of infant mortality. Instead of assuming that the cumulative mortality in the last eleven months of infancy follows a uniform distribution, given a log-cube transformation of age, we assume an exponential distribution. The difference is that while the denominator is constant in the Bourgeois-Pichat model, equal to the number of births, in our model, the denominator is the current population at risk. This assumption is more satisfactory from a theoretical point of view, since it models the conditional probability of dying. The growth in birth weight also follows this distribution (Bourgeois-Pichat, 1951a,b). In addition, the model fit is slightly better for our model, especially in cases with high level of infant mortality, common in pre-modern societies. The advantage is that not only is our assumption more attractive from a theoretical point of view, and therefore easier to interpret, but also that it is makes it easier to estimate exogenous and endogenous infant mortality with standard survival analysis programs.1 In addition, we give examples from eighteenth Sweden, where the curve do not follow the uniform distribution during the last eleven month of infancy, despite high levels of mortality in the first month of life.

2 The Bourgeois-Pichat procedure

Central in the procedure suggested by Bourgeois-Pichat (1951a) is the log-cube transform, see Equation (1).

$$g(t) = C \log^3(t+1), \ 0 \le t \le 365,$$
 (1)

where t is age measured in days and C is a normalizing constant,

$$C = \frac{365}{\log^3(366)}.$$

The constant C is chosen so that g(365) = 365, see Figure 1. Note that C is not part of the original definition of the log-cube transform, but provided here only to make graphical comparisons easier to interpret. It makes no difference otherwise.

Assume that a cohort of infants is followed over time from birth to age one. There are no drop-outs (no right censoring except at age 365 days). The exact age in days at each observed death is noted and transformed by g, and due to imperfect time measuring there may be tied death ages. As an illustrative example, we use a data set from northern Sweden, covering the years 1861-1950.

For the first and last 30 years in this data set, we have the results shown in Figure 2.

Table 1: Life table, 1861-1890.

Day	g(Day)	Population	Deaths
0.25	0.0	50034	564
1.00	0.6	49470	138
2.00	2.4	49332	81
3.00	4.7	49251	83
4.00	7.4	49168	64
5.00	10.2	49102	73
6.00	13.1	49028	57

[Figure 2 about here.]

The cumulative numbers of death are plotted against the corresponding death ages on the g time scale, and as is seen, the fit to linearity after 28 days (68 on the g scale) is excellent for both time periods.

3 The hazard-based procedure

Instead of using the total number of births throughout in the denominator as in the Bourgeois-Pichat procedure, we suggest using the present risk-set size, that is, the the total number of infants still alive immediately prior to the death age under investigation. Given proper data, it is easily achieved. For instance, by the use of the function risksets in the R package eha we can get Table 1.

It starts with 50034 live births, of which 564 dies on the day of birth, on average at the age of six hours (a quarter of a day), and so on.

The plot corresponding to Figure 2 is shown in Figure 3.

[Figure 3 about here.]

Both methods show an excellent fit to linearity for this data set.

4 Theoretical considerations

We note that the Bourgeois-Pichat method requires that no right censorings (infants lost to follow-up before one year of age) are present, and no left truncations (infants under observation only from an age later than birth). The hazards approach, on the other hand, allows left truncation and right censoring as long as they are *non-informative* in the usual sense. Often, though, this not very important, since new-born babies tend to be geographically stationary for their first year of life.

Throughout the rest of the paper, everything happens on the g time scale.

Table 2: Distribution over age periods of exogenous mortality (per 1000).

Neonatal	Total	Fraction
0.930	5	18.60
1.864	10	18.64
9.478	50	18.96
19.371	100	19.37
40.581	200	20.29

4.1 Post-neonatal mortality

We concentrate on the *postneonatal* period, since that is the period where Bourgeois-Pichat claims that the *cumulative distribution function* (CDF) is *Uniform*, and we suggest that the CDF is *exponential*, that is, the *cumulative hazard function* is "uniform" (linear).

In order to see this, the data set from above is *left truncated* at age 28 days (at 68 on the "g" scale), that is, we are considering the *conditional* survival distribution, given survival to age 28.

The *Exponential* fit is excellent, with a slight edge for the early period data, where postneonatal mortality is high.

The fit of the *Uniform* distribution is already shown in Figure 2. Also a very good fits in the post-neonatal period.

4.2 Exogeneous mortality

We now consider the whole infant age span (on the g scale), and compare the uniform and exponential densities, especially of interest is the fraction of deaths that occur in the neonatal phase. Manfredini (2004) argues that a less satisfactory property of the B-P model is that this fraction is constant, 18.56 percent, independent of the overall level of infant mortality p. This is not the case for the hazards model, see Table 2.

4.3 Endogeneous mortality

In order to calculate the *endogeneous* infant mortality, a simple two-step procedure leads to the goal.

1. Estimate the post-neonatal mortality following the exponential route. It reduces to a simple occurrence/exposure calculation: The total number of post-neonatal deaths D is divided by the total postneonatal exposure E on the g scale. So

$$\hat{\lambda} = \frac{D}{E}$$

Table 3: First five rows of data frame.

birthdate	sex	enter	exit	event	period
1861-01-01	girl	0	163	1	1861-1890
1861-01-01	boy	0	365	0	1861-1890
1861-01-01	boy	0	365	0	1861-1890
1861-01-01	boy	0	365	0	1861-1890
1861-01-01	boy	0	57	1	1861-1890

is the estimated hazard function (constant), and the cumulative hazard function is

$$\hat{H}(t) = \hat{\lambda}t, \ t > 0$$

2. On the full infant interval, estimate the total cumulative hazard rate A(t) with the usual Nelson-Aalen estimator $\hat{A}(t)$ (Nelson, 1972; Aalen, 1978). Then, subtract H(t) from $\hat{A}(t)$ to get $\hat{E}(t)$.

$$\hat{E}(t) = \hat{A}(t) - \hat{\lambda}t, \ 0 < t \le g(28).$$
 (2)

Note that subtraction and addition of competing risks are okay on the hazards scale, but not with probabilities, which is yet another argument in favor of the hazards approach.

Let us do it with the given data, and the first period 1861–1890.

4.3.1 Step 1: Estimate post-neonatal (exogenous) mortality

Post-neonatal mortality is the same as exogenous mortality in the sense that the hazard functions are the same on the post-neonatal age interval. So the first step involves data left truncated at age 28 days. The maximum-likelihood estimator of the rate in the exponential distribution is simply the number of observed post-neonatal deaths 1.0136×10^4 , divided by the total post-neonatal exposure time 6.1492634×10^7 (still on the g scale), giving a rate of 1.6483275×10^{-4} .

The rate is a very small number as a consequence of the very small time unit (implying large total exposure time), originally day.

So we are done with the simple post-neonatal period.

4.3.2 Step 2: Estimate total and endogenous mortality

Here we focus on neonatal period.

The estimation of the cumulative hazard function for the total neonatal mortality is easily achieved by calling the function hazards in the *eha* package, see also Figure 5.

```
neo <- age.window(ginfant, c(0, g(28)))
par(las = 1)
fit <- coxreg(Surv(enter, exit, event) ~ 1, data = neo)
hneo <- hazards(fit, cum = TRUE)
x <- c(0, hneo[[1]][, 1])
y <- c(0, 1000 * cumsum(hneo[[1]][, 2]))
plot(x, y, type = "l", xlab = "Day", ylab = "by 1000")
lines(x, 1000 * rate * x, col = "blue", lty = 2)
abline(h = 0, v = 0)
text(8, 15, "Total", col = "black")
text(8, 5, "Exogenous", col = "blue")</pre>
```

[Figure 5 about here.]

Next, take the difference between "Total" and "Exogenous" to get "Endogenous", Figure 6.

[Figure 6 about here.]

5 Biometric analysis in practice

The original procedure of Bourgeos-Pichat is compared to the hazard based procedure for some typical cases from the real world.

5.1 Västerbotten 1921–1950

The crude IMR in Västerbotten 1921–1950 was around 56 per thousand, a rather low figure in context. Let us perform the biometric analysis with these data, see Figure 7.

```
[Figure 7 about here.]
```

There is a good hazards model fit, and we see that the endogenous mortality clearly dominates the early days of neonatal mortality, and almost vanishes towards the start of the postneonatal period.

The B-P model fit is slightly worse, but not much to bother about.

5.2 Västerbotten 1890–1920

The crude IMR in Umeå 1891–1920 was around 95 per thousand, clearly higher than the later time period. Let us perform the biometric analysis with these data, see Figure 8.

```
[Figure 8 about here.]
```

The dominance of endogenous mortality in early life is still clear. The hazards fit is still slightly better.

5.3 Västerbotten 1861–1890

The crude IMR in Umeå 1861–1890 was around 105 per thousand, highest of the three time periods. The biometric analysis is shown in Figure 9.

[Figure 9 about here.]

The conclusion here is almost the same as for the later time periods, good model fit and endogenous dominance in the very early days of life. However, the B-P method seems to have a slight upper hand.

5.4 Scania 1710-1800.

[Figure 10 about here.]

Very bad fits in both cases.

5.5 Skellefteå 1820–1835

An example of severe under-registration during the neonatal period, see Figure 11. There is no room for endogeneous deaths at all, but otherwise a reasonably good fit with both methods.

[Figure 11 about here.]

6 Conclusion

If anything, the hazards method never performs worse than the B-P method. But the real strength of the hazards method is that it fits naturally into general modern survival analysis with censored and truncated data, and also proportional hazards models with covariates.

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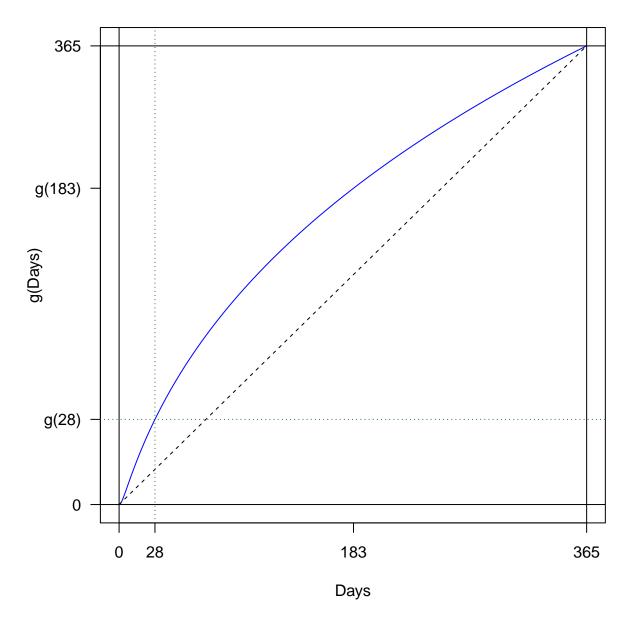


Figure 1: The log-cube transform of time in days versus the identity transform (dashed). Note that g(365)=365.

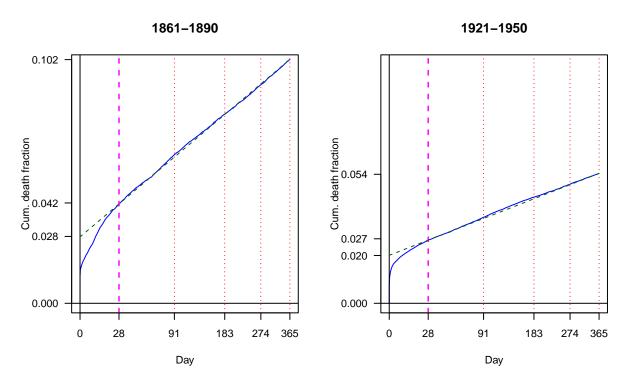


Figure 2: The periods 1861–1890 and 1921–1950 in Västerbotten, Bourgeois-Pichat method.

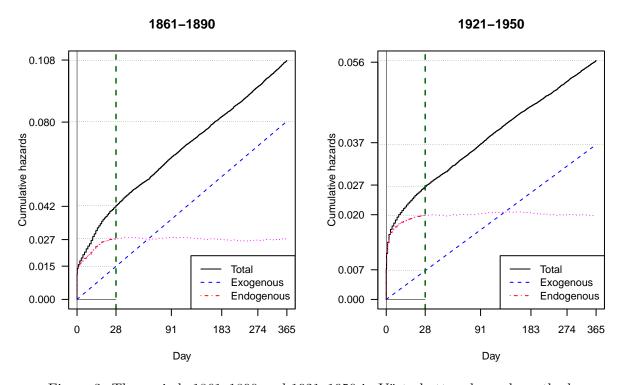


Figure 3: The periods 1861–1890 and 1921–1950 in Västerbotten, hazards method.

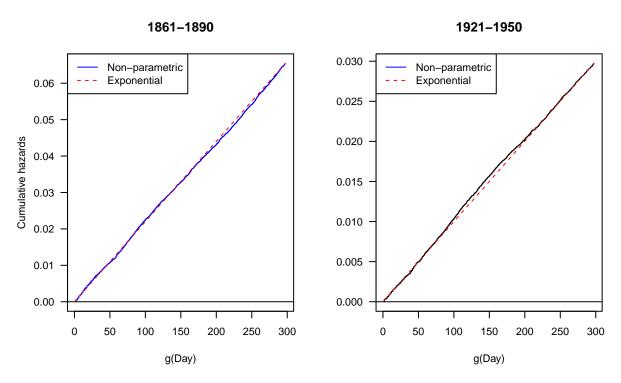


Figure 4: Exponential fits to postneonatal data, Västerbotten.

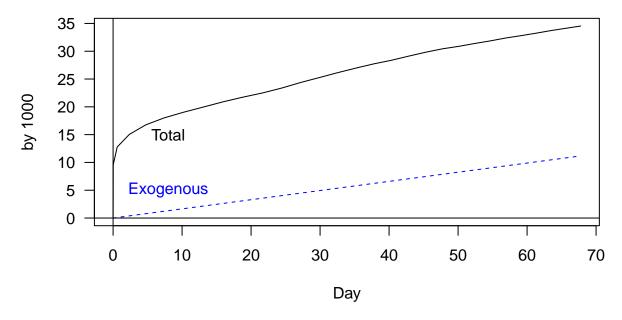


Figure 5: Cumulative hazard functions for neonatal mortality.

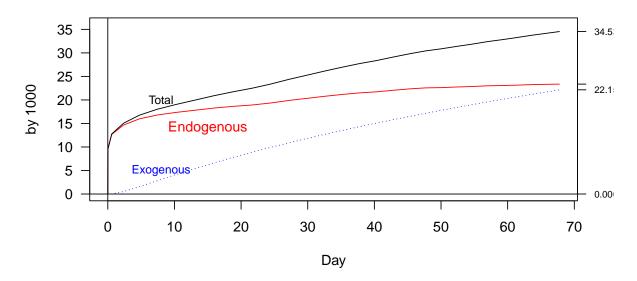


Figure 6: Cumulative hazard function for endogenous neonatal mortality.

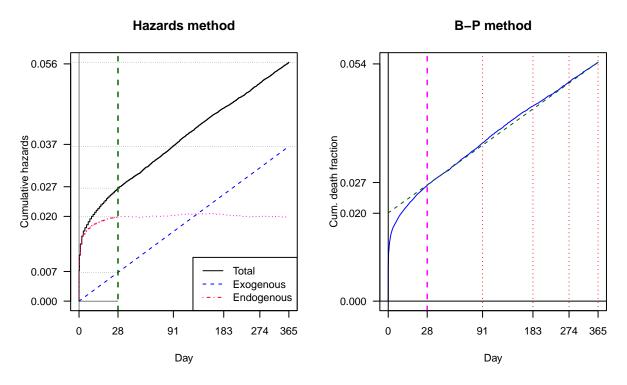


Figure 7: Västerbotten 1921–1950.

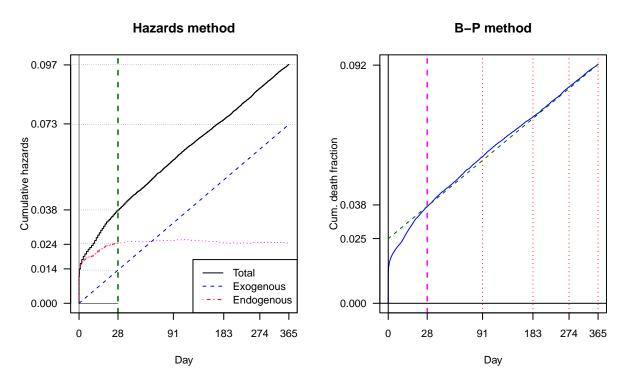


Figure 8: Västerbotten 1891–1920.

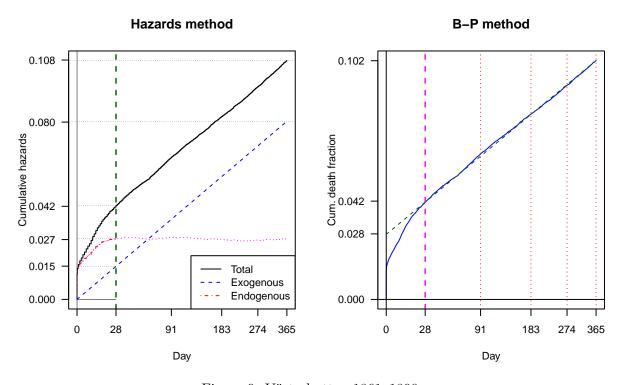


Figure 9: Västerbotten 1861–1890.

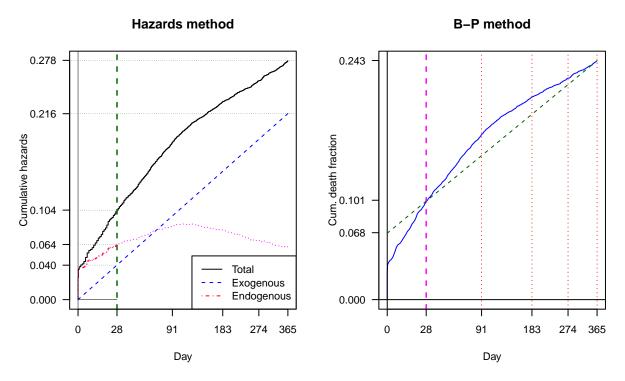


Figure 10: Scania 1710–1800.

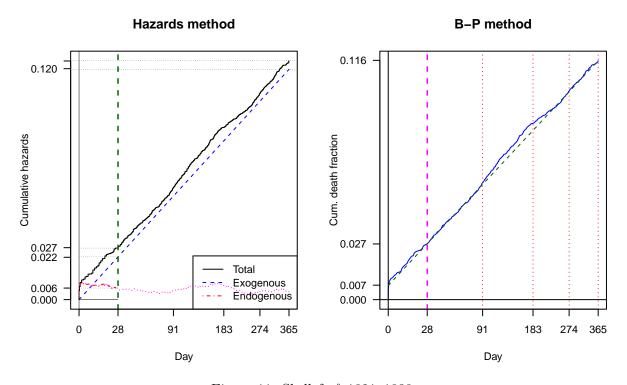


Figure 11: Skellefteå 1821–1838.