The breast-feeding hypothesis and regional differences in marital fertility and infant mortality in the Low Countries during the 19th Century. - Comments on a debate.

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Note: These comments are based on the articles by E.W. Hofstee and by C. Vandenbroecke, F. Van Poppel and A. van der Woude as they appeared in Bevolking en Gezin, 1983. Subsequent English versions prepared for the current volume may contain changes of which I was unaware of at the time of writing (February 7th, 1987). Finally, I would like to acknowledge Professor Hofstee's forwarding of various Dutch registration and census data.

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1. Introduction

The issue addressed in this article is whether regional differences in the duration of breast-feeding can simultaneously account for regional differences in marital fertility and infant mortality levels in the Low Countries around the middle of the previous century. According to the "breast-feeding hypothesis", shorter lactation durations would be a decisive common cause of both increased fertility and infant mortality, and any positive association between the latter two variables would thus be explained. Recently, Vandenbroeke, Van Poppel and van der Woude (1981) have been defending this thesis, whereas Van Hofstee (1983) claims that lactation differentials were far too weak to figure among the decisive factors. Instead, he points at cultural features (such as sexual abstinence) which may have affected fertility and at cultural and ecological variables (such as use of tranquilizers, types of supplementary feeding, incidence of malaria, quality of drinking water, etc.) which affected infant mortality.

Vandenbroeke et al. were led by both micro-level data and materials for geographical aggregates in advancing the breast-feeding hypothesis. The geographical data are presented in Figure 1 in the form of provincial

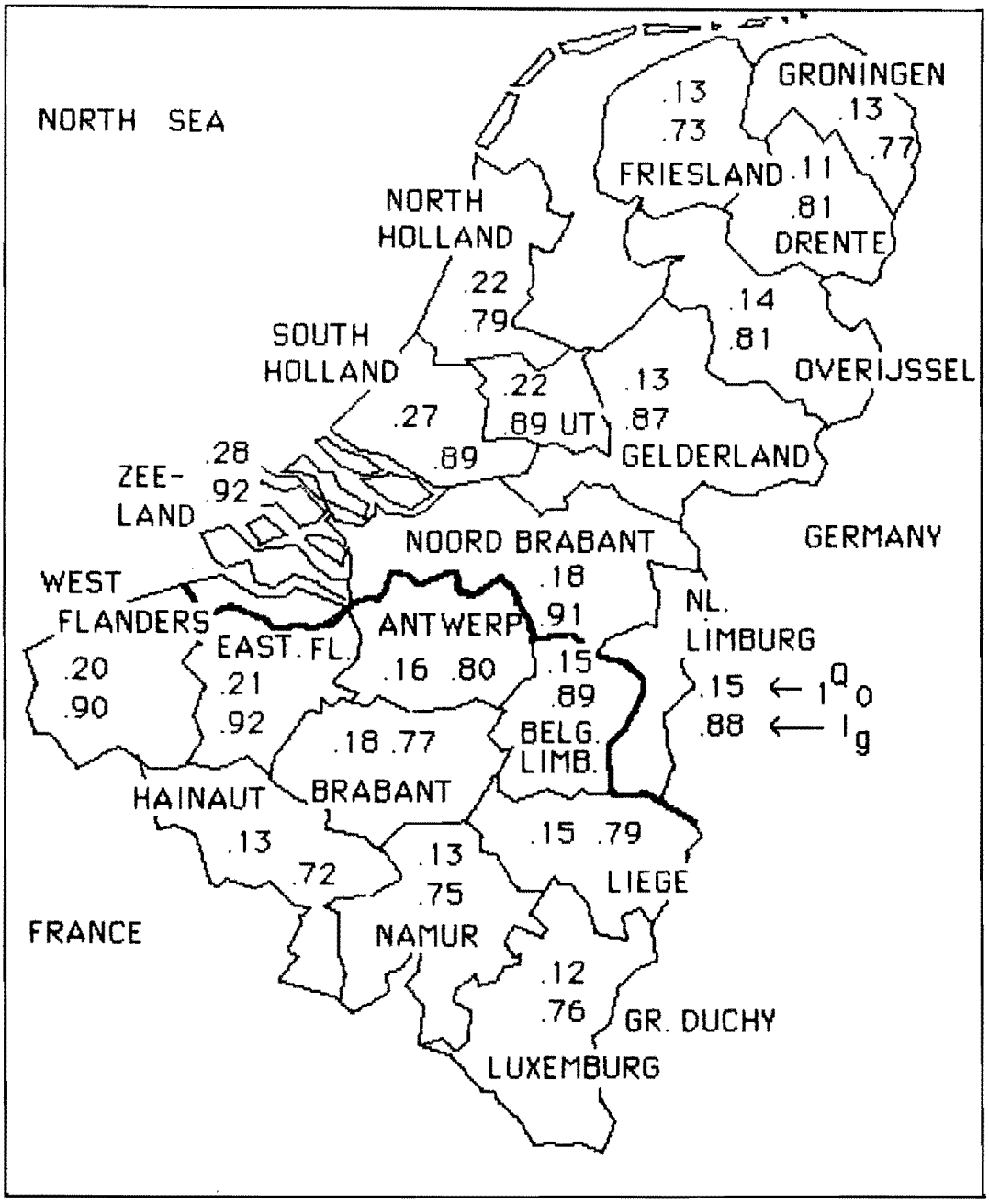

FIGURE 1 : INFANT MORTALITY RATES (IN %) AND INDICES OF MARITAL FERTILITY IN BELGIUM AND THE NETHERLANDS, ± 1860

SOURCES : LESTHAEGHE (1978), DUCHENE AND LESTHAEGHE (1975), HOFSTEE (1982), ENGELEN AND HILLEBRAND (1986)

values of infant mortality rates ($_1q_0$ in per cent) and of Coale's Hutterite-based index of marital fertility (I_g). All values are measured around 1860, i.e. before significant declines occurred. The expected positive relationship between $_1q_0$ and I_g emerges. The 4 north-eastern provinces of the Netherlands (Groningen, Friesland, Drente, Overijssel) and the 4 Walloon provinces of Belgium (Liège, Namur, Hainaut and Luxemburg) have low marital fertility levels (72-81 per cent of Hutterite fertility) combined with low infant mortality (rates below 150 per cent). In the West, infant mortality rates easily reach the level of 200 per cent and Ig-values are in the vicinity of 90 per cent of Hutterite fertility (cf. East an West Flanders, Zeeland, South Holland, Utrecht). These 13 provinces produce the positive relationship. The pattern in the remaining 7 of the 20 provices is less clear, and the most striking outliers are North Holland, with high infant mortality paired to low marital fertility, and Gelderland with the reverse combination. Judging from the overall spatial pattern, the breast-feeding hypothesis would indeed provide a good point of departure.

Hofstee's critique essentially stems from the confrontation of documentary evidence on lactation durations provided by 19th century observers and the durations he inferred from the levels of the general marital fertility rates (births per 1000 married women aged 15-44). For instance, the low fertility level of Friesland would imply long lactation durations which seem implausible when checked against the documentary evidence. Conversely, high fertility in Zeeland or South Holland imply very short durations that are not supported by the descriptions of contemporary observers either. As a consequence, Hofstee rejects the decisive role attributed to breast-feeding and opts for a variety of alternative explanations.

Both points of view draw support from the literature. Table 1, for instance, shows that the infant mortality rates by feeding status varied considerably in early 20th Century urban environments: the risks of dying are always at least twice as high for infants who are bottle-fed compared to those who are still breast-fed. However, the data of table 1 are not broken down by smaller intervals and strong selection-effects can operate. Much better evidence would be provided by sorting the infants that were still breast-fed at the beginning of say, monthly or three-monthly intervals, and by calculating relative mortality risks for those already weaned at the start of the interval compared to those being breast-fed. There is even more evidence on the fertility-reducing capacity of prolonged lactation, and formulae have been worked out to relate breast-feeding durations to the postpartum amenorrhoea component of birth intervals (e.g. Bongaarts and Potter, 1983, or Lesthaeghe and Page, 1980). But it is also well known that levels of natural fertility are equally determined by factors other than postpartum amenorrhoea, and that sexual abstinence, or more generally the frequency of intercourse, is an important determinant of fecundity. Finally, it goes without saying that sanitation conditions, quality of drinking water and epidemiological features all affect infant mortality. Hence, at first sight, the arguments of both sides seem plausible. The question is then whether more refined demographic calculations can tip the balance in favour of one of these two hypotheses. The link between fertility an breast-feeding is documented first as we can draw on well known demographic regularities.

2. Marital fertility, post-partum amenorrhoea and lactation durations

The deduction of average birth intervals for non-sterile married women

Table 1. Infant Mortality Rates (pro mille) by Feeding Status in Selected Cities, 1900-42

	Breast	Partial breast-f.	Bottle	Risk bottle/breast
Paris 1900	140	-	310	2.2
Derby 1900-03	70	99	198	2.9
Amsterdam 1904	144	-	304	2.1
Liverpool 1905	84	134	228	2.7
Boston 1911	30	-	212	7.1
8 US-cities 1911-16	76	-	225	3.4
Cologne 1908	73	-	241	3.3
Liverpool 1936-42	10	26	57	5.6

Sources : Knodel (1977); Carballo (1981)

constitutes the cornerstone of Hofstee's paper (1983, pp.24-29). Since no age-specific marital fertility rates are available for provinces during the 19th Century, Hofstee initiates a decomposition of birth intervals starting from the general marital fertility rate and a set of assumption provided by Buissink and Vincent. The latter assumptions are plausible, but the main problem is that Hofstee's inferred birth intervals are obviously averages for the broad age group from 15 to 44 years. There is hence the implicit assumption of homogeneous fecundity in this wide age interval. This assumption is unrealistic and can easily be avoided. A more flexible approach consists of working out plausible results starting from hypothetical combinations of values for the major components of the birth interval and from working up toward an overall fertility level, rather than by proceeding the other way around. Hypothetical values for the components of the birth interval can be converted into age-specific marital fertility schedules using recurrent historical patterns of natural fertility, and these age-specific schedules can be applied to observed age distributions of married women to produce a set of hypothetical general fertility rates. Observed general marital fertility rates can be located in this network and compatible combinations of fecundity and postpartum amenorrhoea can be inferred. In the process, we are not only introducing heterogeneity of fecundity by age, but also any provincial dissimilarities in the age compositions of married women in the reproductive age range can be accomodated. The logic of the procedure is being illustrated for the Netherlands as a whole in 1859.

The marital fertility rate for a specific age group of married women, e.g. 25-29, is the reciprocal of their average birth interval:

$$m(25\text{-}29) = 1/(\bar{X} \text{ interval in years}) = 12/(\bar{X} \text{ interval in months}) \quad [\ 1\ ]$$

This relationship is valid for instances with homogeneous fecundity in the interval. Heterogeneity can be taken care of by multiplying equation [1] by a factor of approximately 0.875 (Bongaarts, 1976, pp.234-235). Equation [1] furthermore assumes the absence of sterility. If we take the proportion sterile (s) into account for that age group, equation [1] becomes:

$$m(25\text{-}29) = (1\text{-}s)12/\bar{X} \text{ interval (25-29) in mths} \qquad [2]$$

The denominator of [2] can be written as a function of the various components

f = feduncity or the monthly prabability of conception;

I_a = the duration of postpartum amenorrhoea following a miscarriage;

a = the probability of miscarriage;

ppa = the duration of lactational amenorrhoea in months.

Equation [2] then becomes (Bongaarts, 1976, p.235):

$$m(25\text{-}29) = \frac{(1-s)\,12}{ppa + \frac{1}{(1-a)\,f} + \frac{a}{1-a} I_a + 9} \times 0.875 \qquad [3]$$

Equation [3] does not take account of infant mortality interrupting lactation and shortening the length of the postpartum period of amenorrhoea. This can be taken care of by splitting the ppa into a duration typical for the proportion of children dying ($_1q_0$) and a duration for those surviving ($1-{}_1q_0$). Here we shall assume that the ppa for women in the former instance is a third of that for women with surviving children. Equation [3] is then:

$$m(25\text{-}29) = \frac{(1 - s)\,12}{{}_1q_0\left(\frac{ppa}{3}\right) + (1 - {}_1q_0)ppa + \frac{1}{(1-a)f} + \frac{a}{1-a} I_a + 9} \times 0.875 \qquad [4]$$

Bongaarts checked equation [3] against measurements for historical population for which the most reliable estimates could be obtained for f, ppa, s and a. His results for Crulai and Tourouvre au Perche, studied by Henry, Gautier and Charbonneau, are presented in Table 2. Bongaarts' estimates of m(25-29) are indeed close to the observed values, and the same holds for Wilson's application to 16 English parishes (Wilson, 1981).

We can now proceed with the Dutch illustration by fixing the parameters of the birth interval which produce the smallest amount of variation in m(25-29):

s = 7% sterile;

Ia = 1.5 months of amenorrhoea following a spontaneous abortion;

a = 0.20 or one fifth of conceptions ending in a miscarriage;

${}_1q_0$ = 0.200

The actual infant mortality rate for the Netherlands was 0.195 for the period 1850-59 and 0.204 for the period 1860-74 (Hofstee, 1978, table 20). An alternative input of ${}_1q_0$ = .100 will also be used in order to document the effect of a considerable reduction of infant mortality on fertility. The length of the period of postpartum amenorrhoea following a live-birth and for surviving children is allowed to vary from 3 months to 12 months, with jumps of 3 months. The values of fecundity are set 0.20 and 0.28 respectively. These values are close to the extremes witnessed in Western Europe (England, France, Germany, Flanders; see Wilson, 1981, pp.130-131). English and French populations tended toward values of f=0.20, whereas Flemish and German reconstitutions often yielded values above 0.25. The

Table 2. The use of Bongaarts' equation (eqn. 3) for the estimation of marital fertility rates at age 25-29; test involving two French populations

Inputs	Crulai	Tourouvre au Perche
- fecundity (f)	0.18	0.21
- spontaneous abortion risk (a)	0.19	0.19
- length of postpartum amenorrhoea (ppa)	9.1 mths	7.1 mths
- gestation period	9.0 mths	9.0 mths
- postp. amenorrhoea following miscarriage (l_a)	2.5 mths	2.5 mths
- proportion sterile (s)	0.048	0.082
Output		
- Estimated value of m(25-29)	0.400	0.424
- Observed value of m(25-29)	0.420	0.419

Source : Bongaarts, 1976, p.233, table 1.

Table 3. Marital fertility rates for the age group 25-29 obtained via combination of infant mortality ($_1q_0$), fecundity (f) and duration of postpartum amenorrhoea (ppa)

		ppa = 3	6	9	12 mths
i) $_1q_0$ = 0.200	f = 0.20	0.536	0.469	0.417	0.375
ii) $_1q_0$ = 0.100	f = 0.20	0.530	0.460	0.406	0.364
iii) $_1q_0$ = 0.200	f = 0.28	0.594	0.513	0.451	0.403
iv) $_1q_0$ = 0.100	f = 0.28	0.587	0.502	0.439	0.390

levels of f are commonly derived from the first marriage to first birth interval. Since the age group 25-29 was also the modal marriage category in the 19th Century, values of f obtained in this way are quite valid up to age 35, but not thereafter. Equation [4] and ${}_1q_0 = 0.200$, f = 0.20 and ppa = 3 months yield the following value of m(25-29):

$$m(25\text{-}29) = \frac{(1-0.07)\times 12}{(0.8\times 3) + \left(0.2 \times \frac{3}{3}\right) + \frac{1}{0.8\times 2} + \frac{0.2}{0.8} \times 1.5 + 9} \times 0.875 = 0.536$$

The denominator itself equals 18.3 months, which is a rather typical value for birth intervals among non-breast-feeding women. Similar calculations with ppa-values of 6, 9 and 12 months, for ${}_1q_0 = 0.200$ and 0.100 and for f = 0.20 and 0.28 yield the results brought together in Table 3. The following observations can be made:

i) the influence of variation in ppa is strongest as a shift of 3 months produces a change in m(25-29) of 42 to 85 points (pro mille);

ii) the influence of f comes next with a change in the marital fertility rate of 28 to 58 points for a shift in f from 0.20 to 0.28;

iii) the reduction in ${}_1q_0$ from 0.200 to 0.100 is substantial, but its effect on m(25-29) is smallest, i.e. only 6 to 13 points. For infant mortality reductions to have a more sizeable impact on the birth interval via the differential length of amenorrhoea, we would have to assume a fixed lactational amenorrhoea subsequent to an infant death of 1 month only, irrespective of whether the child

dies in the first days or during the 12th month. With the present assumption, the ppa would be 3 to 4 months following a death in the 12^{th}_{+} month, provided that we deal with the long breastfeeding population (ppa = 12). To sum up, there is a causality from infant mortality to marital fertility, but its effect via differential lactation is much smaller than that of fecundity and the overall mean length of lactational amenorrhoea.

The next step consists of inferring a complete age-specific schedule of marital fertility from the rate among women aged 25-29. Profitable use can be made of the general characteristic of natural fertility regimes, i.e. its relatively constant age-profile irrespective of its level. The classic way of showing this consists of setting the rate for women aged 25-29 equal to 100 and to express the other age-specific rates as a percentage of the observed rate for the group 25-29. This operation has been performed on 7 schedules of natural fertility observed in historical populations and on the "standard schedule" proposed by Coale, Hill and Trussell (further abbreviated as the CHT-standard). The results are given in Table 4. The CHT-standard neatly fits the other schedules as its values fall precisely in the middle of the range provided by the values in the historical populations. This range indicates the existence of some variation. Some of it is due to small population sizes, but there are also other systematic effects producing variations in age schedules of natural fertility. One of these effects, studied by Page (1977) in the large Swedish national data set, is that fertility not only tapers off in function of age, but also in function of marriage duration. Women aged 30-35 for instance with a few years of marriage tend to have higher fertility than women of the same age but married for more than a decade. Hence, heterogeneity with respect to the duration of marriage in a specific age group matters, and different

Table 4. Schedules of natural marital fertility standardized on the level at age 25-29 (=100)

	20-24	25-29	30-34	35-39	40-44	45-49
16 English villages	109	100	87	69	38	8
3 Bavarian villages	103	100	92	76	35	6
4 Waldeck villages (Germany)	109	100	90	72	42	6
French Canadians	103	100	97	83	47	7
N.W. French villages	105	100	89	69	35	2
Blankenberge (Flanders)	108	100	90	73	38	4
Hutterites	110	100	89	81	44	12
Range:	103-110	-	87-97	69-83	35-47	2-12
Coale, Hill, Trussell standard	107	100	92	75	39	6

Note : marital fertility rates in the CHT-standard are, starting from age group 20-24: .460; .431; .395; .322; .167; .024.

Table 5. Three alternative age profiles of natural marital fertility depending on the pace of its decline beyond age 30.

	20-24	25-29	30-34	35-39	40-44	45-49
i) pattern of faster decline (NW France)	107	100	89	69	35	2
ii) CHT-standard	107	100	92	75	39	6
iii) pattern of slower decline (Fr. Canadians)	107	100	97	83	47	7

Note : m(25-29) still set at 100.

"marriage duration mixes" between populations produce different age profiles of natural marital fertility. The age patterns of sterility produce additional variation, especially beyond age 35. Finally, certain populations have earlier cessation of sexual relations, which may not be explicitly practised to curtail the size of offspring, but because older women are no longer assumed to become pregnant (e.g. when achieving grand-maternal status). The outcome is that we shall make use of <u>three</u> possible links between m(25-29) and the rest of the age-specific marital fertility schedule, rather than solely mainaining the link of the Coale, Hill, Trussell-standard (see Table 5).

In the present application to the Netherlands, we shall only use the link provided by the standard, but in applications to Drente or Friesland versus that for Zeeland we shall move in the direction suggested by Hofstee's "cultural influence" by respectively using the slower or the faster schedule of Table 5. The calculations involved are fully shown in Table 6. The example assumes the following values for the inputs:

$$f = 0.20$$

$$I_a = 1.5 \text{ months}$$

$$a = 0.20$$

$$_1q_0 = 0.200 \text{ (observed level)}$$

The duration of postpartum amenorrhoea is allowed to vary from 3 to 12 months. The age structure of married women is known and so are the observed number of legitimate births and the general marital fertility rate (15-44). The combination of f, I_a and $_1q_0$ presented above and ppa = 3 months yields a value of m(25-29) of 0.536 (see Table 3). The value of m(25-29) in the CHT-standard is only 0.431, so that an inflation factor

Table 6. Illustration of the calculation of the expected general marital fertility rates for the Netherlands (1859) using hypotheses with respect to various components of the birth interval.

inputs : f=0.20; s=0.07; l_a=1.5 mths; a=0.20; $_1q_0$=0.200; CHT-standard link; variable duration of ppa.

Age group	observed number of married women by age	Expected number of legit. births for ppa=...months: 3 (.536/.431)	6 (.469/.431)	9 (.417/.431)	12 (.375/.431)
15-19	984	502	440	391	352
20-24	21 904	12 485	10 963	9 748	8 766
25-29	58 868	31 436	27 605	24 548	22 074
30-34	80 406	39 399	34 555	30 728	27 632
35-39	84 710	33 884	29 677	26 390	23 731
40-44	72 333	15 022	13 143	11 687	10 509
45-49	61 485	1 844	1 605	1 428	1 284
Totals	380 690 (15-49) 319 205 (15-44)	134 572	117 988	104 920	94 348

General marital fertility rates (legit. births/marr. women 15-44)[a]

Observed rate 1859 = .347 .422 .370 X .329 .296

Interpolated value ppa (see X)

6 mths + 3(.023/.041)mths = 7.7 mths

Inferred duration breast-feeding (see Table 7)

12 mths + 3(0.55/2.19)mths = 12.2 mths

Note : (a) the general marital fertility rate for the Netherlands 1859 can be computed for the entire age range 15-49, but those for the provinces are only available as the total number of all legitimate births divided by the married population aged 15-44.

Table 7. Relationship between average durations of breast-feeding and of postpartum amenorrhoea proposed by J. Bongaarts ($\bar{A} = 1.753 \exp.(0.1396\ \bar{B} - 0.001872\ \bar{B}^2)$)

Average duration breast-feeding ($\bar{B}$)	Average duration pp. amenorrhoea ($\bar{A}$)
0	1.75
1	2.00
2	2.30
3	2.62
4	2.97
5	3.36
6	3.79
7	4.25
8	4.75
9	5.28
10	5.87
11	6.49
12	7.15
15	9.34
18	11.80
21	14.40
24	17.00
27	19.41
30	21.42

Source : Bongaarts (1981); Bongaarts' link between means correspons closely to the Lesthaeghe-Page (1980) link for medians, largely because a similar set of populations has been used to arrive at the translation formula.

equal to .536/.431 is applied to the age-specific marital fertility rates of the standard (see note Table 4) in order to adjust the standard for the actual level. These estimated fertility rates are applied to the numbers of married women by age (see column 1 in Table 6) to obtain the expected numbers of births (column 2 in Table 6). The remaining columns are produced in the same way, but obviously for increasing values of ppa. The total numbers of legitimate numbers of births for each of these 4 combinations are readily obtained and the general marital fertility rates can be calculated. The location of the observed value (here = 0.347) among the 4 hypothetical ones provide an estimate of the suitable duration of postpartum amenorrhoea given, of course, the specific inputs for f, I_a, a and ${}_1q_0$. Finally, the interpolated duration of lactational amnorrhoea can be converted into a duration of breast-feeding via the schedule proposed by Bongaarts and reproduced in Table 7. If f=0.20, the best fitting duration of breast-feeding equals 12.2 months, but an increase in f to 0.28 allows for a longer duration of postpartum amenorrhoea of 9.7 months instead of 7.7 months, and hence also for a higher estimate of the duration of breast-feeding of 15.4 months instead of 12.2 months. We have no extra information on the true level of f, but a value of 0.28 for the country as a whole seems to be on the high side. Presumably, the real duration of breast-feeding was of the order o 12 or 13 months and that of lactational amenorrhoea of 7 to 8 months around 1860 in the Netherlands.

Identical calculations were also performed for Friesland, Drente and Zeeland. The results are given in Table 8. Note that the actual levels of infant mortality have been used as inputs and that allowance is made for a difference in the tempo of the natural fertility reduction past age 30. The size of the married population aged 45-49 was also estimated by applying the national ratio of those 45-49 to those 40-44. The error is

Table 8. Comparison of values of the general marital fertility rates produced by combination of postpartum amenorrhoea, fecundity and the pace of natural fertility past age 30 for the Dutch provinces of Friesland, Drente and Zeeland; 1859-60.

	Observed general marit. fert. rate	Interpolated means for: breast-feeding	pp.amenorrhoea
Friesland ($_1q_0$ = 0.125)			
f = 0.20; CHT-standard link	.305	16.9	10.9
f = 0.20; link with faster decline		15.4	9.7
f = 0.28; CHT-standard link		19.2	12.8
f = 0.28; link with faster decline		17.4	11.3
Drente ($_1q_0$ = 0.125)			
f = 0.20; CHT-standard link	.331	14.1	8.7
f = 0.20; link with faster decline		12.8	7.7
f = 0.28; CHT-standard link		16.7	10.7
f = 0.28; link with faster decline		15.4	9.7
Zeeland ($_1q_0$ = 0.220)			
f = 0.20; CHT-standard link	.379	9.9	5.9
f = 0.20; link with slower decline		12.8	7.7
f = 0.28; CHT-standard link		13.3	8.1
f = 0.28; link with slower decline		15.6	9.8

minimal as marital fertility rates are very low for these older women. In fact we are dealing with errors of a dozen births in Friesland and a few units in the other two provinces.

If the three provinces were to differ only with respect to lactation durations, these durations would be as follows:

	Friesland	Drente	Zeeland
f=.20; CHT-standard	16.9	14.1	9.9 mths
f=.28; CHT-standard	19.2	16.7	13.3 mths

The duration of breast-feeding would be 45 to 75 per cent longer in Friesland than in Zeeland, depending on the value of f for the age group 25-29. Hofstee estimates the duration for Zeeland between 10.3 and 13 months (1983, p.30, 41) which fits the figures represented above. His range, however, results from alternative hypotheses concerning foetal mortality and stillbirths, and its width suggests an implausibly strong effect of these two events upon the birth interval. The range produced here is the result of differences in fecundity which are known to be strong. Hofstee's estimate for Drente is 18.5 months (ibid., p.41) or longer (ibid., p.41), and if his method were applied to Friesland with its low marital fertility, he would have arrived in the close vicinity of 24 months. The method presented here produces much smaller contrasts between these 3 provinces and they only reach 10 months if allowance is made for much higher fecundity in Friesland than in Zeeland (which is implausible). The alternative cannot be ruled out: if Friesand has the lower fecundity (f=.20) and the faster pattern of declining fertility with age, whereas Zeeland has the higher fecundity (f=.28) and the slower pattern of decline past age 30, the durations of lacation would have been very similar, i.e.

15.4 and 15.6 months respectivly. If we settle for the middle road rather than for the calculations that either minimize or maximize the differences, we would come up with a difference of roughly 4 to 6 months between Zeeland and the two north-eastern provinces. This difference is furthermore smaller than the one estimated by Hofstee. Hence, relatively small differences in lactation durations are quite capable of producing noticeable effects on marital fertility levels, and differential breast-feeding cannot be rejected as being one of the main factors underlying the provincial fertility map of the middle of the 19th Century. On the other hand, Hofstee may be quite right in supposing that the very high infant mortality of Zeeland could not solely be due to a lack or very short durations of breast-feeding. His estimates of the duration of lactation in Zeeland are not implausible (despite his methodology) and the mean was indeed still of the order of 12 months around 1860. Other factors must have operated in conjunction with breast-feeding in accounting for the high western infant mortality belt.

3. Breast-feeding and infant mortality

The study of this relationship is far more difficult than that of the link between lactation and fertility. We have no handy decomposition and linkage formulae of the type used in the previous section. The main reason for this is that very strong synergism exist between breast-feeding and other factors in determining infant mortality. Consequently, the notion of statistical interaction is central to this section, and we shall offer several examples to clarify the issue.

By statistical interaction one implies that the effect of a particular variable (in casu breast-feeding) can be enhanced or dampened depending on

the combination with other factors. Reduced breast-feeding in an area with poor drinking water is likely to result in much higher infant mortality than in an area with good quality drinking water. Similarly, interactions are likely to exist with sanitation conditions, presence or absence of malaria etc. Reduced breast-feeding can even result in a near zero effect on infant mortality if strong countervailing or corrective forces exist in the environment.

The problem with the discussion between Hofstee and Vandenbroeke et al. is that it has the tone of a "X versus Y" debate. Such a debate can be settled if the underlying model were indeed of a linear nature and of the form

$$Z = A + B_1 X + B_2 Y + \varepsilon_z$$

In such instances it suffices to estimate the two coefficients B_1 and B_2, and to inspect which one of the two predictors X or Y has the strongest effect and predictive power. The matter is, however, quite different if the effect of X (say breast-feeding) depends entirely on the specific value of Y (say quality of drinking water) and vice versa. We therefore suspect that both Hofstee and Vandenbroeke et al. contribute to the identification of crucial variables involved, but that the discussion evolves along a misspecified statistical model.

A few examples can document this point. Vandenbroeke et al. (1983, p.102, table 4) present data on infant mortality by feeding history and socio-economic category in The Hague (1908) and Tilburg (1912). This information is used here for 4 of the 5 socio-economic groups: the highest group has several missing cells and we suspect that the sample sizes --

which are unfortunately not given -- are smallest for this category. The middle categories of feeding history in the original table are also taken together as the values are virtually identical (exclusive breast-feeding shorter than 8 weeks + breast-feeding shorter than 8 weeks but some supplementation from birth onwards). A Tukey median polish (Tukey, 1977) can be applied assuming an additive model at first. The observed values (O_{ij}) are decomposed into an overall level (A), plus a row effect (R_i -- here the feeding history), plus a column effect (C_j -- here the socio-economic class effect), plus residuals (ε_{ij}) not fitted by the additive model:

$$O_{ij} = A + R_i + C_j + \varepsilon_{ij}$$

with i denoting the rows and j denoting the columns. The procedure and the results are shown in Table 9 for the data from The Hague (1908). Step 1 of the median polish consists of extracting the overall median from all the cell values in the original table (ME = 67). The first set of residuals is calculated in the body of the table, and in step 2 the column medians are extracted from them. A second set of residuals results from which the row medians are extracted in step 3. No further extractions are needed as the estimates have stabilized. Row and column effects are now known and the final set of residuals has emerged. The results can also be represented graphically by means of a rectangular grid with parallel lines for rows at a 45 degree angle with parallel lines for columns (see Figure 2). The intersections identify the mortality level of the cells. The residuals are furthermore located with respect to this grid.

The actual results for The Hague show that exclusive artificial feeding since birth has a major mortality increasing effect relative to exclusive

Table 9. Infant mortality, feeding history and socio-economic group in The Hague 1908; illustration of the Tukey median-polish

	socio-economic group I lowest	II	III	IV highest
8+ weeks excl. breast :	64	49	23	17
shorter breast + suppl. birth:	110	94	53	32
excl. artificial feeding :	311	229	158	70

step 1: extraction overall median

-3	-18	-44	-50	
+43	+27	-14	-35	
+244	+162	+91	+3	
				67

step 2 : extraction remaining column medians

-46	-45	-30	-15	
0	0	0	0	
+201	+135	+105	+38	
+43	+27	-14	-35	67

step 3 : extraction remaining row medians

-9	-8	+7	+22	-37	feeding effect
0	0	0	0	0	
+81	+15	-15	-82	+120	
+43	+27	-14	-35	67	

socio-economic effect

step 4 : reconstruction of table assuming additive effects

73	57	16	-5
110	94	53	32
230	214	173	152

step 5 : residuals

-9	-8	+7	+22
0	0	0	0
+81	+15	-15	-82

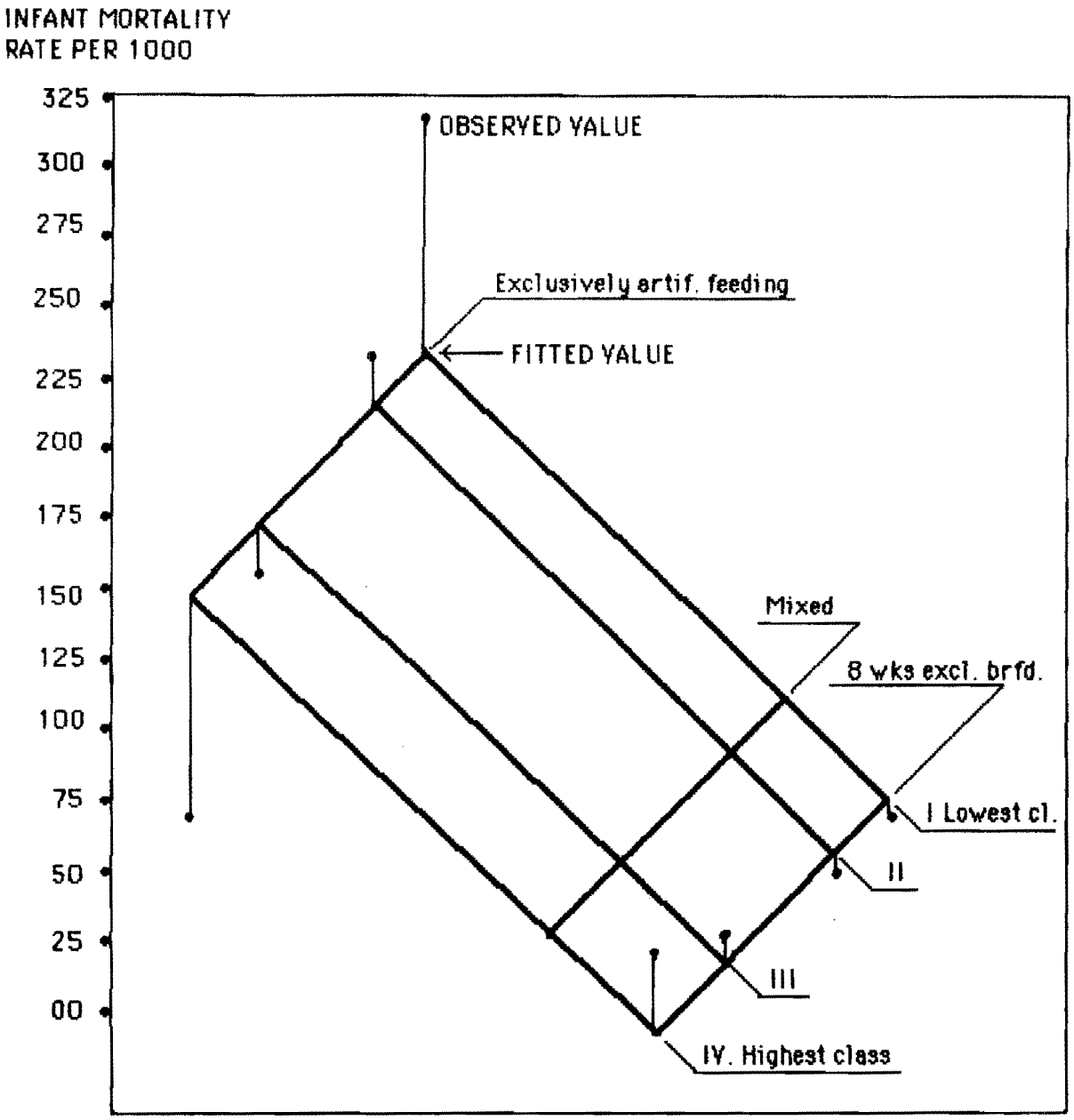

FIGURE 2 : INFANT MORTALITY RATES BY FEEDING HISTORY AND SOCIO-ECONOMIC CLASS IN THE HAGUE, 1908 ; RESULTS OF A TUKEY MEDIAN POLISH ASSUMING ADDITIVE EFFECTS.

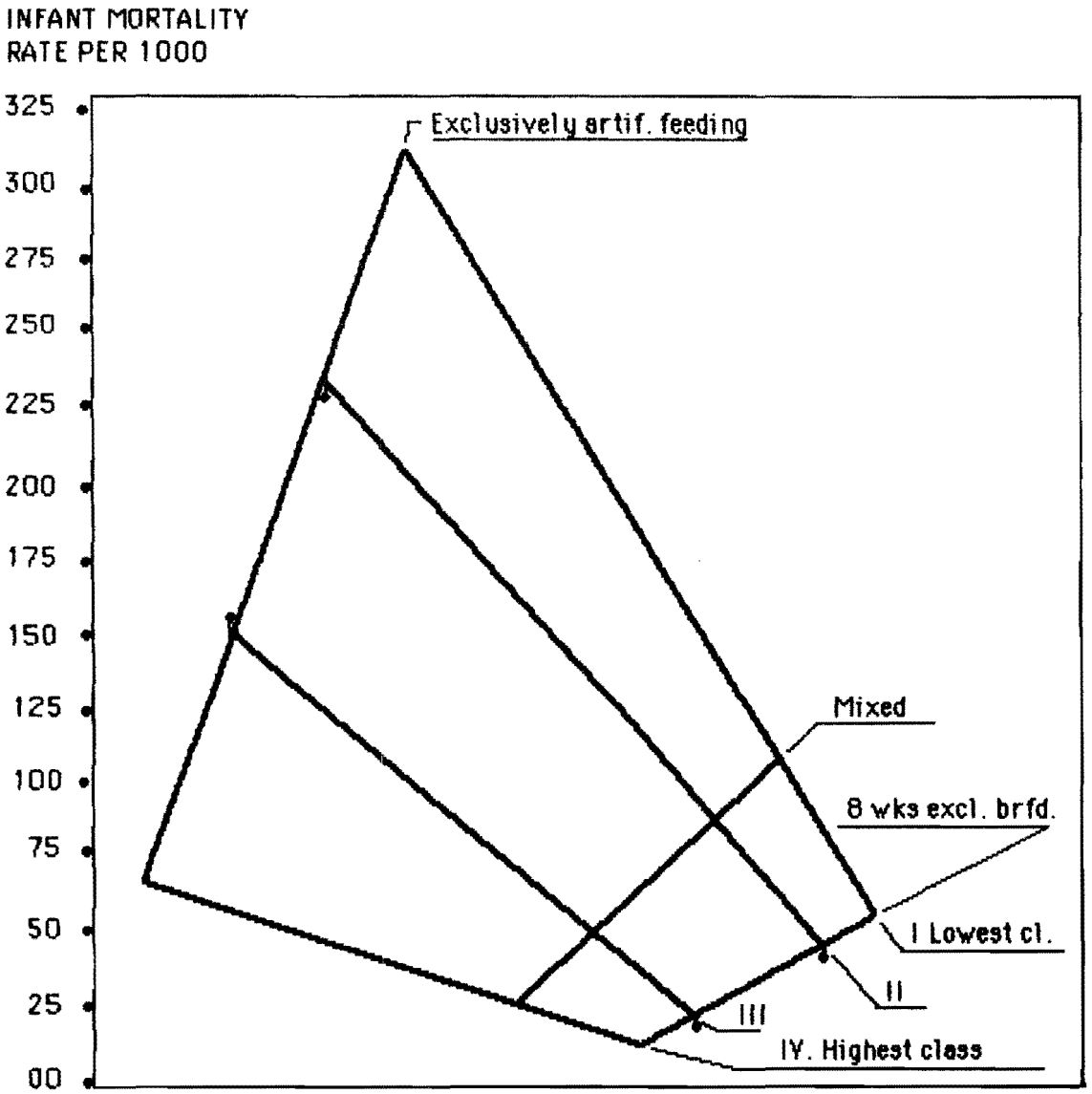

FIGURE 3 : INFANT MORTALITY RATES BY FEEDING-HISTORY AND SOCIO-ECONOMIC CLASS IN THE HAGUE, 1908 ; RESULTS FROM A FIT ASSUMING INTERACTION.

breast-feeding in the first 8 weeks. The effect of mixed feeding prior to 8 weeks is much smaller. Socio-economic class has also a clear effect, but the differences between the lowest and highest class is only half as large as the difference between the extreme feeding categories. The separation of the two effects, that of feeding and that of "culture and environment" associated with social class, is obviously quite simple if such an additive model holds. Unfortunately, the residuals are particularly large for infants with exclusively artificial feeding, and the additive model (or the rectangular grid) is inadequate. A model which takes the interaction between socio-economic environment and feeding history into account produces quite a different picture, as shown in Figure 3. The residuals have virtually vanished, but the grid is far from rectangular. The socio-economic class differences in mortality for breast-fed children have not changed all that much when compared to the results of the additive model, but they have more than doubled for exclusively artificially fed children. These features are of course pointed out by Vandenbroeke et al., but the casting of them in a simple statistical model with interaction would have drawn greater attention to this central synergetic phenomenon and may have led the discussion along a more fruitful track. The data for Tilburg (1912) are even more striking in producing such an interactive pattern (although values of infant mortality of 700 pro mille in the lowest class and among artificially fed children seems excessive and probably due to sample size limitations.

A second example of interactions between determinants of infant and child mortality is available from Eelens (1984) for Kenya 1977-78. The two predictors are weaning at the start of an interval and the incidence of malaria in the Kenyan districts. We have chosen this example since Hofstee explicitly refers to malaria in the wet meadow regions of Zeeland, Utrecht,

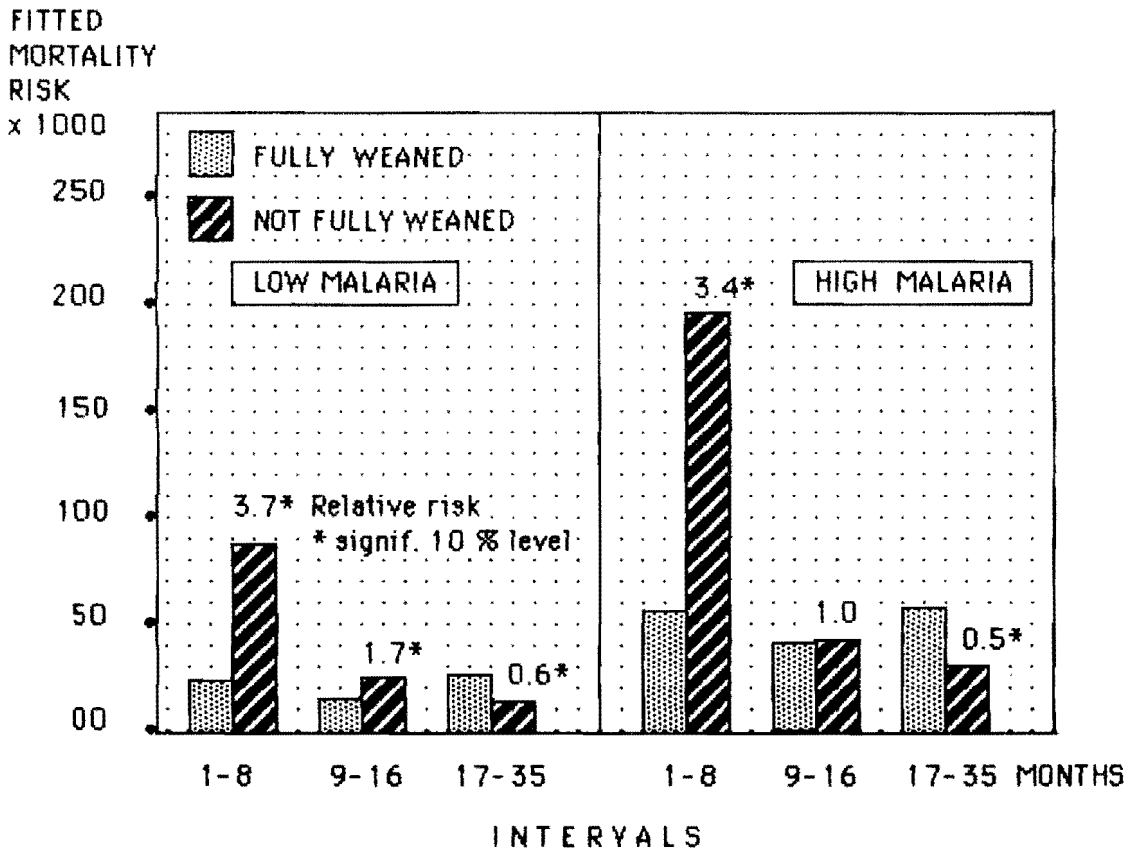

FIGURE 4 : FITTED MORTALITY PROBABILITIES BY WEANING STATUS AT THE BEGINNING OF INTERVAL AND BY PREVALENCE OF MALARIA IN REGION OF RESIDENCE, KENYA 1977-78

SOURCE : EELENS (1984), DATA FROM KENYAN FERTILITY SURVEY.

North and South Holland (a zone which is partially continued in East and West Flanders), and because it is a rare example which jointly utilizes the feeding and malaria contrasts. Eelens furthermore computes mortality probabilities within these intervals for children who are fully weaned at the beginning and for those continue to be breastfed (exclusively or partially). The results from the best fitting logistic regression model are represented in Figure 4. All mortality risks in the districts with a high incidence of malaria are approximately twice those found in the areas without malaria, controlling for interval and feeding category. The effect of weaning is particularly strong in the interval from 1 to 8 months: weaning during the first month increases the chances of dying by a factor of 3.7 in the low malaria zone and by a similar factor of 3.4 in the high malaria districts of Kenya. In the second interval, the effect of weaning before the 9th month is considerably attenuated with relative risk ratios of 1.7 and 1.0 in the respective malaria zones. In the third interval, the relationship is reversed with higher risks for children that are still being breast-fed beyond the 17th month. The relative risks are again very similar: 0.6 and 0.5. The reversal of direction is due to the selection effect whereby only the weaker or sick children are still being breast-fed for extra long durations. To sum up, the feeding effects are very similar on both sides of the malaria divide, but the added malaria risk multiplies the rates by a factor of 2.

One will notice that all comparisons in the Kenyan example are based on ratios and not on differences between rates. This facilitates descriptions of situations dominated by interaction effects. The data for The Hague and Tilburg can also be treated this way, as shown in Table 10. Infant mortality rates are now being related to the column and row with the smallest values. No formal model could be fitted as we lack information on

Table 10. Relative risks associated with observed infant mortality rates by feeding-history and socio-economic group in The Hague, 1908

Original data					
socio-econ. gr.:	I	II	III	IV	
8 weeks exclusively breast-feeding	64	49	23	17	
8 weeks mixed	110	94	53	32	
exclusively artificial feeding	311	229	158	70	
Effects of feeding-history					
8 weeks exclusively breast-feeding	1.00	1.00	1.00	1.00	Average
8 weeks mixed	1.72	1.92	2.30	1.88	1.96
exclusively artificial feeding	4.86	4.67	6.87	4.12	5.13
Effects of socio-ecnomic status					
8 weeks exclusively breast-feeding	3.76	2.88	1.35	1.00	
8 weeks mixed	3.44	2.94	1.66	1.00	
exclusively artificial feeding	4.44	3.27	2.26	1.00	
Average	3.88	3.03	1.76		

Note : sample sizes were unknown to the present author so that no attempt has been made to fit a formal model.

cell sample sizes, but these simple illustrative calculations produce a clear message: mixed feeding during the first 8 weeks increases the mortality rates by a factor of 2 and exclusively artificial feeding from birth onwards by a factor of 5, almost irrespective of social class. Compared to the highest class (i.e. IV), class III increases the rates by a factor of 1.75, class II by a factor of 3.00 and class I almost by a factor of 4.00. The estimate for the rate in class I combined with exclusively artificial feeding is then expected to be 4x5=20 times higher than in class IV with 8 weeks exclusive breast-feeding: 20x17=340 as compared to 311. Hence, if Vandenbroeke et al. had analysed their data on Tilburg and The Hague though multiplicative rather than additive effects -- they do notice that an amplification occurs --, they would have seized the underlying regularity much better and accentuated the interactive nature of the process involved. If this point would have been clear, the discusson could have followed a more fruitful direction: feeding and "culture or environment" <u>both</u> matter, and the outcome is a very <u>rapid</u> increase in infant mortality as adverse effect belong to either one of these determining variables are <u>combined</u> through multiplicative rather than through additive effects.

A third example illustrating the interactive effects between feeding practice and sanitation is provided by Da Vanzo on the basis of Malaysian data for 1976-77. She computed the increase in infant mortality to be expected if unsupplemented and supplemented breast-feeding were to drop to zero in situations with and without piped water and toilets. The results are reproduced in Table 11 for 3 intervals. The first set of figures show the expected increase in mortality rates if breast-feeding were absent altogether, and the second set if there were a sudden switch from breast-feeding to other feeding without breast supplementation. In

Table 11. Sanitation environment and the effect of elimination of breast-feeding on infant mortality in Malaysia, 1976-77.

Intervals :		Day 8-28	Months 2-6	Months 7-12
Mortality rates per 1000				
Changes in mortality rates (in points pro mille) if unsupplemented breast-feeding drops to zero				
Toilet	Piped water			
No	No	+44	+90	+43
No	Yes	+37	+76	+29
Yes	No	+16	+22	+25
Yes	Yes	+10	+8	+11
Changes if supplemented breast-feeding drops to zero				
Toilet	Piped water			
No	No	+30	+56	+22
No	Yes	+34	+49	+11
Yes	No	+1	+11	+15

Source : J. Da Vanzo (1984, p.316)

situations with toilets and piped water, the resulting deterioration of mortality rates is nearly unnoticeable. In the absence of these amenities, the increases are 4 to 14 times larger, or expressed differently, the original rates increase by 25 to 75 per cent. Once again: the impact of feeding practices are dramatically amplified or dampened according to the environmental factors that Hofstee identifies. Furthermore, this feature is extremely marked during the first few months of life when infants are particularly vulnerable to all effects.

4. Conclusions

1. Relatively small differences in regional average durations of lactation and amenorrhoea (i.e. of the order of 4 months or higher) can definitely produce noticeable and decisive differences in marital fertility levels. Breast-feeding differences are, however, not the only candidates for providing an explanation: differences in fecundity levels come next, but then, these differences must have been rather large (see contrast between f=.20 and .28 in combination with the tempo of fertility decline past age 30). Hofstee's proposition that regional differences in infant mortality, stillbirths and miscarriages were responsible is not plausible: birth intervals are comparatively speaking more robust to variations in these 3 variables. On the whole, breast-feeding probably provides the main causal variable affecting marital fertility levels in the Low Countries prior to the onset of the demographic transition (i.e. before 1870).

2. The Hofstee-Vandenbroeke et al. debate concerning the factors determining infant mortality is too strongly cast within a dualistic framework. Both sides are insufficiently aware of the multiplicative

nature of the combination effects. Neither breast-feeding nor several of the other cultural and environmental variables identified by Hofstee can be deleted from the list of prime factors influencing infant mortality prior to World War I. In the absence of the tables of the type provided for Tilburg and The Hague, which at least show the more precise nature of the interactive models to be fitted, regional comparisons quickly come to a halt. If such micro-information exists for other cities, or even better, for several rural areas (e.g. clay versus sandy soil regions; agricultural versus cottage-industrial etc.), one could progress significantly by comparing the nature of the best fitting models and the size of the relative risk ratios.

3. One issue has not been discussed so far: data quality. Vandenbroeke et al. produce time series for infant mortality spanning the whole of the 19th Century and interpret short term trends in function of presumed changes in breast-feeding. Aside from the fact that changes in the latter can only be guessed on the basis of fragmentary descriptions by a few 19th Century observers, changes in the former should be treated very cautiously. The Belgian infant mortality rates for the Napoleonic period for instance are based on our fitting of the Princeton North and West-model life tables to mortality schedules for all age groups (Duchène and Lesthaeghe, 1975). These figures are therefore only rough estimates. Their sole value is that they indicate that the Belgian regional pattern at the beginning of the 19th Century was not markedly different from the one observed during the last quarter of that century. Secondly, the data quality varies significantly between the regions at all points in time until about 1870. Data for the two Limburg provinces, for instance, are notoriously bad in the first half of the 19th Century. Thirdly, mortality rates in the first month are affected

by registration and definition problems. Figures for stillbirths and for the special category "presenté sans vie" (i.e. the children presumably born alive but dying before they were registered with the Belgian communal authorities) are volatile and their inclusion or exclusion in the computation has a noticeable effect on the size of the infant mortality rates. This problem does not alter the ranking of Belgian provinces at any point in time, but it definitely creates bumps and troughs in the time series for each province separately. I am less familiar with the quality of the Dutch information on infant mortality, but I suspect that similar problems exist which preclude the analysis of relatively small changes from one decade to the next, especially if they occur outside periods of manifest economic hardship or epidemics. In general, I am only at ease with provincial time series from the last quarter of the 19th Century onwards.

4. Despite the problems discussed in this paper, the contributions by Hofstee and Vandenbroeke et al. have brought together a set of important materials. Clearly, there exists a kind of digging that persistent historians do better than model-fitting demographers. But, both are needed and as interdisciplinary research is synergistic too, the gains from their concerted efforts are likely to be multiplicative.

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