DEMOGRAPHIC COMPONENTS OF SEASONALITY OF PREGNANCY

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Abstract

Previous studies of the seasonality of birth and/or pregnancy have been based on monthly numbers or proportions of births or pregnancies (i.e., their distribution throughout the year) without any explicit accounting of the size of the population actually at risk of pregnancy by month. The data on which past studies have been based permitted little more than such a numerator analysis. The present paper presents data on monthly populations at risk and monthly probabilities of pregnancy and we subsequently determine the relative contribution of each component to the monthly distribution of pregnancies. The data come from the National Survey of Family Growth, cycle III. We find that while there is some seasonality in the size of the population at risk, it contributes little, in fact, to the overall seasonality of pregnancy. The latter is, instead, determined almost completely by seasonality of pregnancy probability (fecundability).

Every human population evidently has seasonality of birth. The pattern varies considerably world-wide, and has been observed to vary within countries and over time as well.[1] At least for the U.S., most studies have found a consistent seasonality pattern characterized by higher numbers of births in late summer and early fall, and a valley centered in April (Warren, et al. 1986; Lam and Miron, 1985; Seiver, 1985; Rosenberg, 1966). The possible causes of birth seasonality have been discussed by numerous researchers but there are no generally accepted explanations.

The various hypotheses of the causes of birth seasonality can be distilled into three general categories: (1) seasonal changes in the exposure to the risk of pregnancy through socio-cultural mechanisms. Rodgers and Udry (1985), using the NSFG cycle II data, made the first attempts to identify seasonal variations in the size of the population at risk by examining seasonality of contraceptive stopping times. A clear seasonal pattern of stopping times was revealed for those women purposely stopping in order to get pregnant, which is presumptive evidence that there is at least one source of volitional seasonal variation in the size of the population at risk. (2) seasonal differences in fecundability, based on biological factors (changes in hormones, sperm count, photoperiod, etc.) or on behavioral factors such as coital frequency (3) factors occuring after pregnancy, such as induced or spontaneous abortion as well as length of the gestational period.

The first two components of birth seasonality mentioned above, seasonality of the size of the at-risk population and seasonality of fecundability, relate to pre-pregnancy factors and it is these that we will elaborate on, focusing this study on the seasonality of pregnancy rather than birth. To our knowledge, neither of these components have ever been measured nor have their relative importance been assessed. Previous studies have largely been based on monthly numbers or proportions of births (or pregnancies, when available), without any explicit accounting of the size of the population at risk by month. Indeed, the data on which past studies have been based (vital statistics, typically) permit little more than such a numerator analysis, without allowing the identification of the population at risk (denominator) by month or the estimation of fecundability (numerator over denominator) by month. It would be a conceptual advance to move from monthly distributions of pregnancies (numerators) to monthly exposure and fecundability rates.

Our study presents data on the size of the at-risk population by month as well as monthly probabilities of pregnancy. From these data, we can determine the relative contribution of these two components to the monthly distribution of pregnancies. Each component may be considered to be conceptually independent of the other. The varying size of the at-risk population is likely to be determined in part by a volitional element such as contraceptive stopping times (Rodgers and Udry). But if there is seasonal variation in fecundability, then the size of the at-risk population will vary as a function of fecundability as well, being low in some months because of the depletion of the at-risk population in month after month of high fecundability.

THE DATA

The data set we have chosen is the National Survey of Family Growth, Cycle III. It allows us to identify on a monthly basis, for the 36 months between January 1979 and December 1981, whether or not a woman was contracepting, pregnant, sexually active, sterile or sterilized (in short, whether she is at risk), and the timing of pregnancies.

In order to obtain the number of women at risk of pregnancy in any given month, we selected a group of women continuously married between January 1979 and December 1981, and removed not-at-risk women on a month-by-month basis. The criteria for defining women as not-at-risk included: (1) already pregnant during that month; (2) contracepting during that month; (3) had a live birth within the last three months; (4) had a non-live birth within the last month; (5) did not have intercourse in the month of interest; (6) sterile.[2]

The determination of risk status, then, is made separately for each month, and women may either leave or enter the at-risk state in any given month. Those at risk in a given month comprise the denominator which is used to determine the probability of pregnancy (i.e., fecundability) for each of the 36 months. The monthly numerators (numbers of pregnancies) are selected only from those women defined as at-risk by the previous operations.[3]

RESULTS

All of our results are presented by reducing the 36 month. 3-year period into a 12 month. one-year period. That is, we combine the three Januaries (Februaries. March's, etc.) into one January (February, March...). Figure 1 shows the monthly variation in the population at risk (1979-1981 combined). There turns out to be relatively little variation here, with a maximum difference of 6 percent in the size of the population at risk. A peak in June is evident, however, and is consistent with the fact that June is a peak month for stopping contraception in order to get pregnant in this sample of women (analyses not shown). The modest seasonal variation revealed here suggests that seasonal differences in the size of the at-risk population could, but is not likely to, contribute to seasonal variation in pregnancy distributions.

[FIGURE 1 ABOUT HERE]

This monthly enumeration of the population at risk forms the denominator for estimating monthly variations in fecundability (number of pregnancies in month x divided by number at risk in month x). The month in which each pregnancy occured is determined by asking the woman when she became pregnant. This is hardly the best imaginable measure, but it is better than subtracting 40 weeks from the birth date.

Figure 2 shows the monthly probabilities of pregnancy for at-risk women (1979-1981 combined). Fecundability ranges from .055 to .092. These estimates are considerably lower than those obtained by most other studies of human populations, where fecundability estimates typically fall in the vicinity of .20. To boost our estimates into a .20 vicinity, we would need an at-risk group only a third the size that we have, or we would need three times as many pregnancies. First of all, it is likely that pregnancies ending in induced or spontaneous abortion are underreported, which would reduce the numerator and inflate the denominator by placing more women in the at-risk category (for several months). It is also possible that there is a process of selection of subfecund women into the noncontracepting status, and hence the at-risk status by our definition, which could inflate the denominators, leading to underestimation of fecundability. It may also be the case that some women are practicing contraception but tell us they are not, which would cause the at-risk group to be overestimated (but we doubt this is common). All of these artifactual or measurement error processes may work together to produce lower fecundability estimates. In any case, there is little reason to imagine that such measurement error is a significant cause of the pattern of monthly variation we observe in any of the graphs.

[FIGURE 2 ABOUT HERE]

A simple chi-square test was used to test the null hypothesis that the monthly pregnancy rates are equal. The test yielded a chi-square of 22.55 with 11 degrees of freedom ($.025<p<.05$) on the basis of which we reject the null hypothesis. An examination of the contribution of each month to the chi-square statistic and deviations of monthly pregnancies from the expected values show that the month of October gives a large positive deviation of pregnancies and contributes a chi-square value of 8.12 to the total chi-square statistic. A significant negative contribution (4.26) occurs during the month of April.

Figure 3 shows the monthly distribution of the number of new pregnancies by month for the three years combined. We can now address the question, Is the seasonal distribution of pregnancies in this sample determined more by the seasonality of the size of the at-risk population, or by the seasonality of fecundability? By using the eyeball method thus far, the answer is fairly clear. The month with the highest number at risk is 6 percent higher than the month with the lowest number at risk. The month with the highest fecundability is 70 per cent higher than the month with lowest fecundability. The month with the highest number of pregnancies is 73 percent higher than the month with the lowest number of pregnancies. The shape of the fecundability graph is almost exactly the same as the number of pregnancies graph, while the population at risk graph is less similar to the number of pregnancies graph. A procedure was devised (see Appendix) to estimate the contribution of the varying population at risk and monthly pregnancy rates to monthly variation in the number of pregnancies. The procedure confirms that nearly all of the variation in the monthly number of pregnancies is contributed by the pregnancy rate (fecundability). The seasonal pattern of the population at risk plays only a small part, then, in modulating the seasonality of pregnancy, which is basically determined by the seasonality of fecundability.

[FIGURE 3 ABOUT HERE]

Many researchers have, in fact, assumed that seasonality of fecundability is the underlying cause of seasonality of birth or pregnancy; their hypotheses have usually focused on different determinants of fecundability (for example, coital frequency, hormones; or weather, holidays at the less proximate level). Other researchers have approached the problem by looking for patterns of seasonality in the termination of contraceptive use (implying seasonality of size of the at-risk population). Our results based on U.S. populations allow us to eliminate the explanation involving seasonality in the size of at-risk population and encourages the continued exploration of the question of why fecundability shows a seasonal pattern.

Although our study suggests that seasonality of fecundability underlies the seasonality of pregnancy, it cannot, unfortunately, tell us exactly *what* form the seasonal fecundability pattern takes in the general population (or our total sample). What *is* shown in our fecundability graph (Figure 2) is variation in fecundability across different monthly at-risk populations, populations which hardly differ in size but may differ in their fecundability. Women can enter or leave the at-risk state each month through a variety of mechanisms which may be correlated with seasonality of fecundability (for a variety of reasons). The fecundability of the changing at-risk population from Figure 2 may or may not represent a seasonal fecundability pattern for the general population, which theoretically could be estimated if a fixed group of women or a constant at-risk population were to have their fecundability measured monthly in some manner. But if the changing at-risk populations were known to have similar socio-demographic characteristics, we could be more certain that Figure 2 is an adequate portrayal of seasonality of fecundability for the general population.[4]

If we can assume for the sake of discussion that our fecundability graph is a good approximation of a true fecundability pattern for the U.S. population, and we think it does have external validity, then part of the pattern is interpretable in the context of past research. We notice two valleys, one in the spring and one in the hotter summer months. The spring valley is hard to explain, but the summer valley is consistent with the hypothesis of lower coital frequency due to summer heat (Bongaarts, 1986; Seiver, 1985; Lam and Miron, 1985). If summer heat causes the summer trough, then the subsequent autumn rise in fecundability may reflect a return to previous levels of sexual activity. Pregnancies have consistently been found to peak in October through December in studies based on vital statistics (Warren, et al. 1986), and although this has puzzled researchers for some time, the early autumn increase in coital frequency is one possible explanation.

DISCUSSION

In order to examine the components of seasonality of pregnancy, we compute seasonal patterns for the size of the population at risk and for fecundability. We find that while there is some seasonality in the size of the population at risk, it contributes little to the overall seasonality of pregnancy, which is determined in this population almost completely by seasonality of fecundability.

We should keep in mind that the population on which our study is based consists mostly of women who control their pregnancy risk through the use of effective contraception. In any such population there is opportunity for wide shifts in the number of women at risk by month. Yet, one can imagine populations in which such variation might account for more of the seasonality of pregnancy than in the present sample. Seasonal variations in fecundability are less under volitional control, with only frequency of coitus and lactation being the voluntary contributors. In this study, we arbitrarily declared a woman as not at risk for three months following a birth, and did not take breastfeeding into account. In any case, the two components may make different contributions in different populations.

FOOTNOTES

[1]Literature reviews of the patterns and possible mechanisms can be found in Lam and Miron (1985) and Kevan (1979).

[2]For each pregnancy during 1979 to 1981, the NSFG created an interval record with information on the timing of contraceptive use and periods of non-intercourse in the period prior to that pregnancy. If a woman stopped or began contracepting or having intercourse in month x, we defined her as at-risk in month x. When the month of beginning contraception is not reported by the woman but she reported using prior to the pregnancy, her start month was assigned by us as the month of her previous delivery (or before January 1979 if it is the first interval record for the 1979-81 period we are examining). When the stop date is not known but the start month comes before the month of interest, the woman is defined by us as not at risk in that month.

For spans of time in the period of interest for which there is no interval record to provide retrospective information on contraception and intercourse timing, we fall back on either of two possible sources of information: (1) the questionaire (respondent record) item which asks for the start and stop time of the woman's first, second, third, and last contraceptive use, or (2) the method-use calendar. Which source we use depends on which can provide the most relevant information for a particular woman. The method use calendar allows the determination of risk status for January and July only, for each of the three years covered in our study. When this calendar was chosen to be the source of at-risk information for a respondent, the following decision rules were applied: If the woman was not at risk (either because of contraceptive use, non-intercourse, or being sterile) in two consecutive semi-annual observations, we assumed the woman was not at risk for every month between the observations. If a woman was at risk in two consecutive semi-annual observations, we assumed she was at risk in the intervening months. If a woman switched from one risk status to the other between two semi-annual observations (an uncommon event), we randomly assigned a switch month within the period, with an equal probability of falling in each month.

Because of the greater tendency for pregnancies, and thus pregnancy records, to occur in the early part of the 1979-81 period for this sample of women married prior to January 1979, the risk status of women can be ascertained more accurately for the early part of the period.

[3]The definition of the at-risk population provided the denominator from which monthly probabilities of pregnancy were computed. This is the only way in which the computation makes sense, but it introduces the assumption that women not at risk by our definition do not get pregnant. Yet, we know that some women get pregnant while contracepting. In fact, some women who we define as not at risk do get pregnant, but the monthly probabilities of pregnancy for our not-at-risk population never exceed .005. The problem may, therefore, be considered negligable for present purposes.

[4]In a sense the problem is analagous to one we face when trying to infer over-time (longitudinal) changes or patterns within one population from the observation of synthetic cohorts in the cross-section. One needs to assume in the latter case that the

synthetic cohorts have the same characteristics except for one: duration or time in some state. Although our problem is not really one of synthetic cohorts, to interpret our graph as a real seasonality pattern we would have to assume that the at-risk populations have identical characteristics. The socio-demographic characteristics of the monthly at-risk populations can be examined and possibly adjusted or controlled with our data (possibly yielding a purer seasonality pattern), but that is beyond the scope of the present paper.

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FIGURE 1. SIZE OF AT-RISK POPULATION BY MONTH, 1979-81 COMBINED

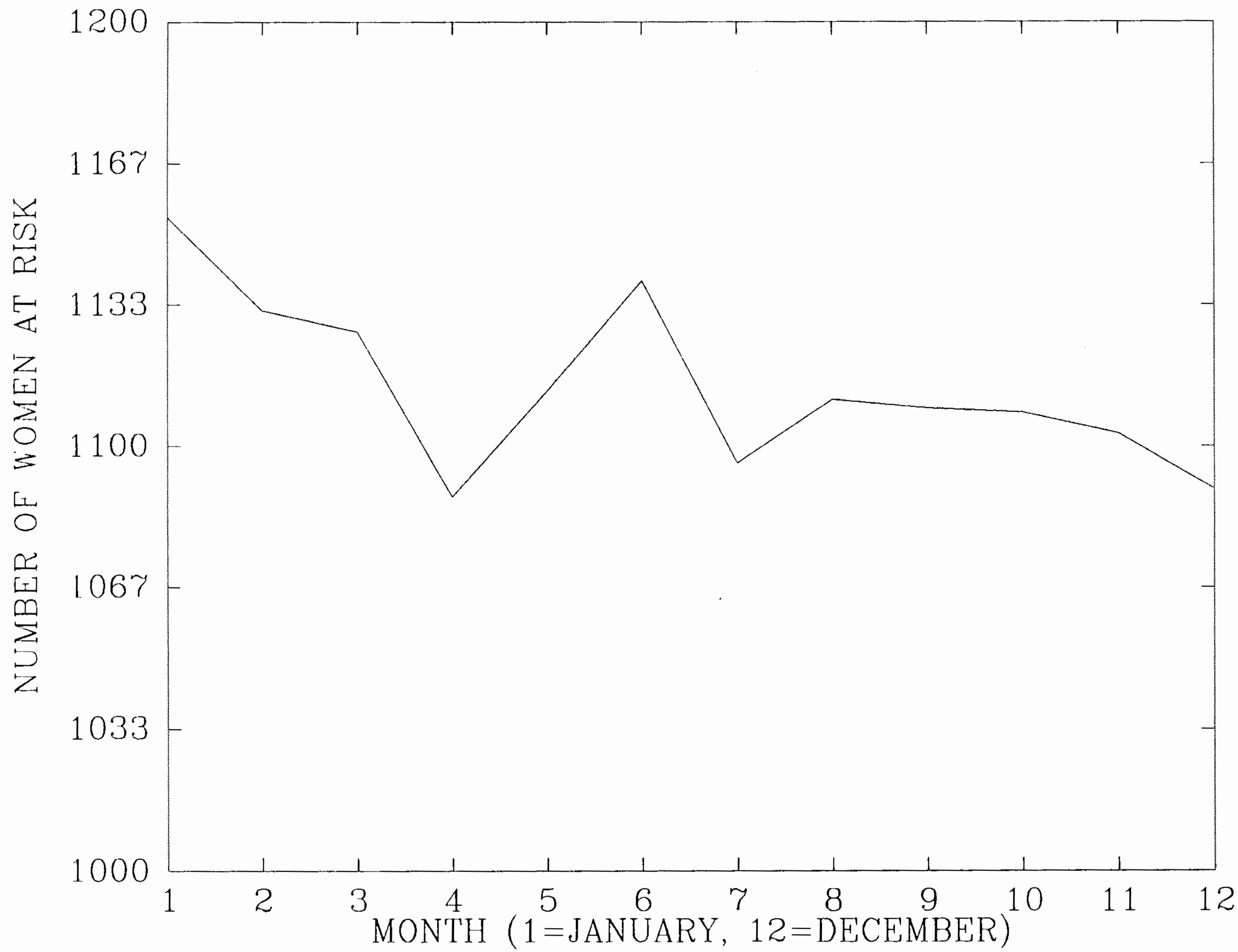


FIGURE 2. PROBABILITY OF PREGNANCY (FECUNDABILITY), 1979-81 COMBINED

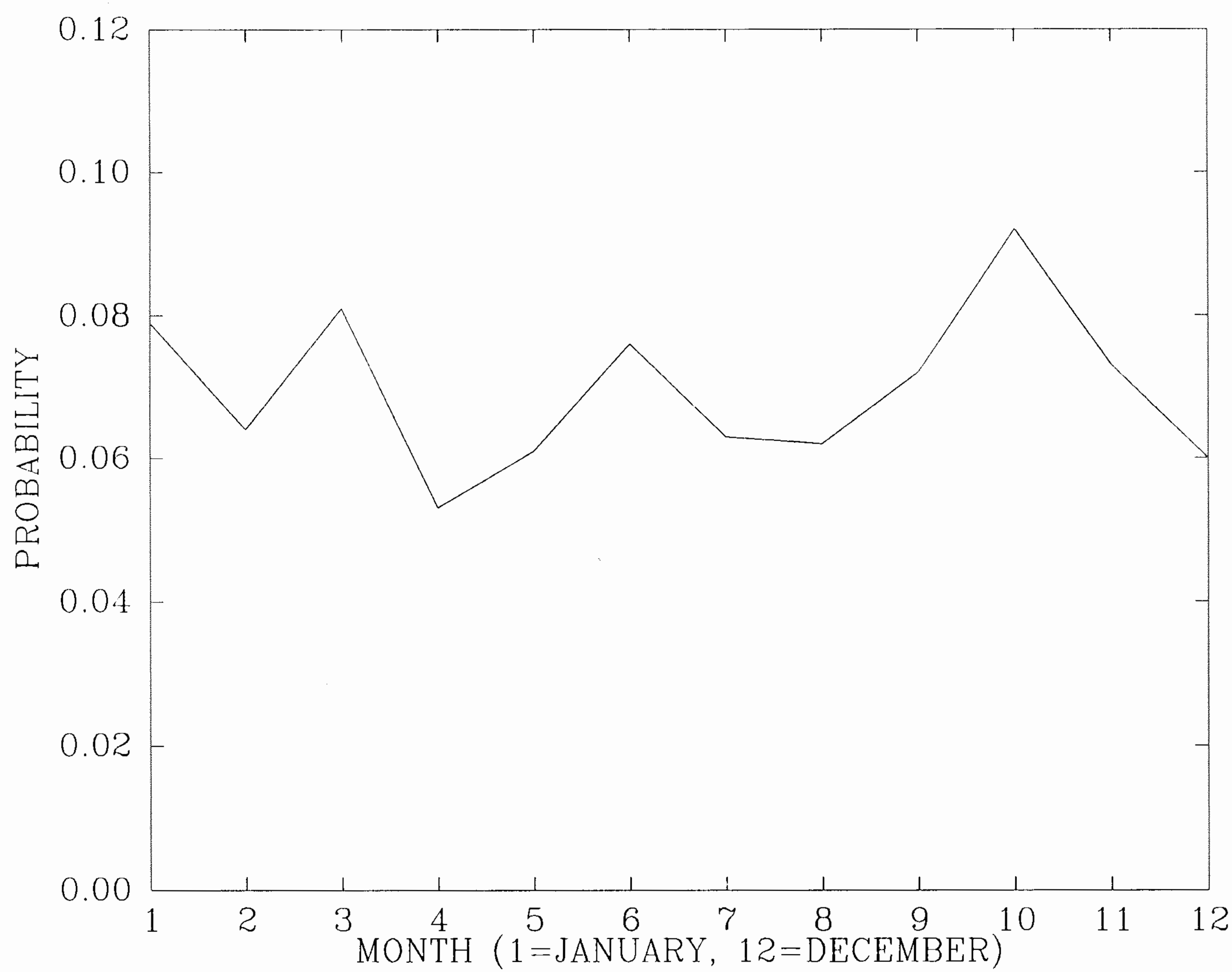
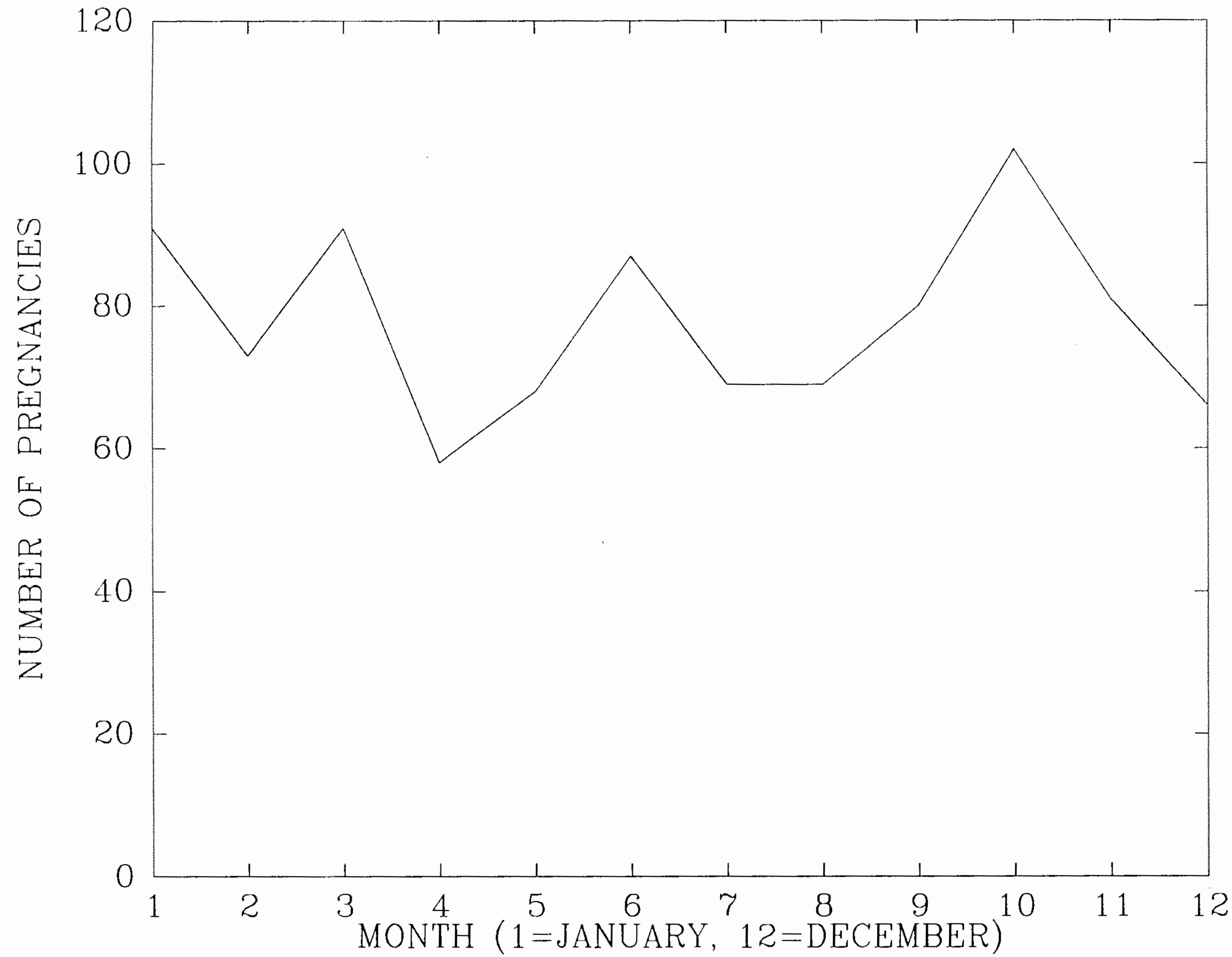


FIGURE 3. NUMBER OF PREGNANCIES, 1979–81 COMBINED



Appendix

Decomposition of variability of monthly pregnancies into components attributable to the population at risk and monthly rate of pregnancy:

Let

d_i denote the number of pregnancies in month i,

N_i the number at risk in month i,

and p_i the pregnancy rate in month i, i=1,2,......12.

Then, the total variance in the number of pregnancies can be expressed as

$$\text{Variance } (d) = \frac{1}{12} \Sigma_i (d_i - \bar{d})^2 \tag{1}$$

where $\bar{d} = \frac{1}{12} \Sigma d_i$.

The expression (1) can also be written as

$$\text{Variance } (d) = \frac{1}{24 \times 12} \sum_{i=1}^{12} \sum_{j=1}^{12} (d_i - d_j)^2 \tag{2}$$

But

$$\sum_{i=1}^{12} \sum_{j=1}^{12} (d_i - d_j)^2 = \Sigma\ (N_i p_i - N_j p_j) \tag{3}$$

Expression (3) can be rewritten as

$$\begin{aligned} \sum_{i=1}^{12} \sum_{j=1}^{12} (d_i - d_j)^2 &= \sum_i \sum_j \left(\frac{N_i + N_j}{2}\right)^2 (p_i - p_j)^2 \\ &+ \sum_i \sum_j \left(\frac{p_i + p_j}{2}\right)^2 (N_i - N_j)^2 \\ &+ \sum_i \sum_j \left(\frac{N_i + N_j}{2}\right) \left(\frac{p_i + p_j}{2}\right) (p_i - p_j)\ (N_i - N_j). \end{aligned} \tag{4}$$

Note that the equation (4) has three terms. When all the monthly rates of pregnancy are equal and the population at risks are unequal, the first and the third term will vanish. Thus, the second term can be attributable to variations in the population at risk alone. Similarly the first term is attributable to variations in fecundability. The third can be termed as an interaction factor. For the data presented here,

$\Sigma\ (d_i - d_j)^2 = 22067$ and the three components are respectively

19669, 265, and -2133.

These calculations show that nearly all variation in the distribution of pregnancies by month is due to variations in the monthly rate of pregnancy, i.e., fecundability.

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